Parameter identifiability and identification of a hierarchical epidemiological model

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PROGRAM

ABSTRACT BOOK

Conference Centre – UNICAMP

July 16-20, 2007, Campinas, SP, Brazil

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WELCOME

The organizers of the Second Conference on Computational and Mathematical Population Dynamics (CMPD2) and the University of Campinas (UNICAMP) have the pleasure and the honor to greet and welcome you all who are joining this intellectual venture.

Indeed, this is the first time that the CMPD or any of its predecessors conferences meet at the Tropics and we thank those sensible people that in the last CMPD chose it to be in Brazil, among all possible (also nice but never nicer) places below the Equator.

You will be living during the next few days in Campinas ("Meadowlands"), a lively and large city of about one million inhabitants, too close (~100km) to the megalopolis São Paulo (almost 20 million inhabitants), but still keeping some peculiarities of Brazilian small town. Unicamp is located inside a borough of Campinas, called Barão Geraldo named after a Baron (yes, Brazil was once and for a long time a tropical, Empire, up to 1889) in a region which just 35 years ago was only a large sugar cane plantation. If you travel a few kilometers westward in the São Paulo State you still can visit those "green deserts", now in such strident vogue due to the current ethanol crush.

Unicamp is not a typical Brazilian university since it was planned to be primarily a scientific institution. Despite the initial motivation, arts and social sciences are well represented today, which makes UNICAMP a full-blown university; in fact, it has become an important graduate center in the recent years, with almost half of its student body registered in a MS or PhD programs.

Although nowadays the globalization wave seems to be inescapable and pervasive, you should beware that under the superficial sameness of shopping centers and McDonalds, a lot of “Brazilianess” can still be found by the attentive eye. We hope that more often than not you meet the nicest part of it during your stay with us.

This conference will be a unique opportunity for many students and young researchers from Brazil as well as from South America to place themselves in a first hand contact with the state-of-the-art in Biomathematics and related areas of research. So, an intensive and extensive mingling of leaders and aspiring researchers would be the ideal scenario for this CMPD2.

Let us hope then that the population wave of bio-mathematicians now invading the Campinas region can, at least momentarily, increase considerably our support capacity on such scientific matters, and bring enough contagious enthusiasm to completely infect the local susceptible population, in such extent that the threshold parameter R_0 will be far greater than one for a long time in the future.

This Conference was made possible due to generous contributions of voluntary work of many individuals. We particularly thank personnel from UNICAMP and UNESP-Botucatu as represented by Prof. Drs. José Tadeu Jorge (Rector of UNICAMP) and Jayme Vaz Jr. (Director of IMECC-UNICAMP) and Prof. Drs. Maria de Lourdes M. V. Paulino (Director of IBB-UNESP) and Helenice O. F. Silva (Head of the Department of Biostatistics, IBB-UNESP). The members of organizing committee as well as all the participants extend gratitude and acknowledgement for the sponsor from Institutions (UNICAMP, USP, UNESP and IMECC-UNICAMP), Scientific Societies (SBMAC, ESMTB and SMB) and Government Funding Agencies (FAPESP, CAPES and CNPq) and support from PETROBRAS, SABESP, CONSORTIUM for the AIS and NOVARTIS.

BEM-VINDOS

Wilson Castro Ferreira Jr.
On behalf of Organizing and Local Committees

CMPD2 – General View

Period	16 (M)	17 (T)	18 (W)	19 (Th)	20 (F)
8:30-9:30	Registration	G. Chowell -Puente	Pierre Auger	Bedr´Eddi-ne Ainseba	Faustino Sanchez
9:40-10:40	Registration	Sessions	Sessions	Sessions	Sessions
10:40-11:00	Registration	Coffee	Coffee	Coffee	Coffee
11:00-12:20	Karl Sigmund	Sessions	Sessions	Sessions	Eduardo Massad
14:00-15:00	Sessions	E. Tan-nenbaum	Cláudio Struchiner	V.M. (Nitant) Kenkre	Meeting
15:00			Biomat2007 15:00 – 15:15	Consortium 15:00 – 15:15	Meeting
15:10-16:30	Sessions	Sessions	Sessions (at 15:20)	Sessions (at 15:20)	Meeting
16:30-17:00	Coffee	Coffee	Coffee	Coffee	
17:00-18:00	Bryan Grenfell	Sessions	Poster	Poster	
18:00	Opening Ceremony and Reception	Cultural (18:30 to 19:30)	Dinner (18:30 to 21:30)	Cultural (18:30 to 19:30)	

Plenary Sessions

(Auditorium: 1)

Monday, July 16, 11:00 AM

Chairperson: W.C. Ferreira Jr.

Collaborative Efforts and Altruistic Punishment

Karl Sigmund

Professor, Faculty for Mathematics, University of Vienna, Vienna, Austria

In human societies, groups of individuals often collaborate in joint efforts. Those contribute fare less well than those who defect and exploit the others. Why don't the defectors take over? The threat of punishment seems to be very effective, but this raises a `second order social dilemma'. It is often costly to punish others, so how can the threat of punishment remain credible? This issue will be studied by means of deterministic and stochastic models from evolutionary game theory. The possibility that players are free not to participate in the joint enterprise may be a key factor in the evolution of cooperation in sizable groups.

Monday, July 16, 5:00 PM

Chairperson: Gerardo Chowell-Puente

Waves, sparks and nonlinearities in the dynamics of infectious diseases

Bryan Grenfell

Alumni Professor of Biology, Department of Biology, The Pennsylvania State University, USA

Infectious diseases have exerted a huge toll on human and animal populations, both historically and up to the present. Starting with measles as an example, this lecture explores how the pattern of epidemics in space and time depends on a balance between the spread of infection and the nonlinear `herd immunity' of the host population. I then go on to explore how the evolution of immune escape in influenza and other infections affects these dynamics. The lecture concludes by discussing how this balance between invasion and nonlinear feedback also drives the within-host dynamics of infection.

Tuesday, July 17, 8:30 AM

Chairperson: Michel Langlais

The effects of spatial heterogeneity in the spread of infectious diseases

Gerardo Chowell-Puente

Professor, Center for Nonlinear Studies, Los Alamos National Laboratory, USA

I will present a county-based metapopulation model for the spread of foot-and-mouth disease (FMD) in the context of the 2001 foot-and-mouth disease epidemic in Uruguay, and a spatial analysis of the great influenza pandemic of 1918-19 in England and Wales. Retrospective analyses of these real epidemic scenarios using mathematical models can play an important role in the development of new theories on the effects of spatial heterogeneity and the design of control interventions. We compared a spatial model with a null model based on the homogeneous mixing assumption by use of a spatial dataset of the 2001 FMD epidemic in Uruguay. We used a parameter estimation scheme to calibrate the model from the spatial epidemic time series. The spatial model was able to recover a two-mode epidemic and, we used it to assess the role of control interventions.

Next, I will present an analysis of spatial heterogeneity using weekly number of influenza deaths during the fall and winter waves of the 1918-19 pandemic in England and Wales, at two levels of spatial resolution (333 administrative units and 62 counties). Transmissibility was estimated through the reproduction number, R, using a sequential Bayesian approach combined with a SEIR (susceptible-exposed-infectious-removed) model. Death rate and R estimates were then tested for correlations against historical census variables. Findings indicated significant heterogeneity of R across counties during the fall and winter waves. We observed a systematic decline in transmissibility and death rates from the fall to the winter wave, suggesting a possible effect of cross-immunity between the influenza viruses circulating in the two waves, and in agreement with anecdotal evidence. Transmissibility tended to be higher as a function of larger populations and higher population density. The cumulative number of influenza deaths varied as a power law of population size with an exponent significantly less than one (~0.85), driven by the more rural and sparsely populated areas.

Tuesday, July 17, 2:00 PM

Chairperson: J. F. Fontanari

Selective advantage for sexual reproduction

Emmanuel Tannenbaum

Professor, School of Biology, Georgia Institute of Technology, USA

This talk develops a simplified model for sexual reproduction within the quasispecies formalism. The model assumes a diploid genome consisting of two chromosomes, where the fitness is determined by the number of chromosomes that are identical to a given master sequence. We also assume that there is a cost to sexual reproduction, given by a characteristic time τ_{seek} during which haploid cells seek out a mate with which to recombine. If the mating strategy is such that only viable haploids can mate, then when $\tau_{seek}=0$, it is possible to show that sexual reproduction will always out compete asexual reproduction. However, as τ_{seek} increases, sexual reproduction only becomes advantageous at progressively higher mutation rates. Once the time cost for sex reaches a critical threshold, the selective advantage for sexual reproduction disappears entirely. The results of this talk suggest that sexual reproduction is not advantageous in small populations per se, but rather in populations with low replication rates. In this regime, the cost for sex is sufficiently low that the selective advantage obtained through recombination leads to the dominance of the strategy. In fact, at a given replication rate and for a fixed environment volume, sexual reproduction is selected for in high populations because of the reduced time spent finding a reproductive partner.

Wednesday, July 18, 8:30 AM

Chairperson: Cláudio Struchiner

Aggregation methods in population and community dynamics

Pierre Auger
Centre IRD de l'île de France, Bondy, France

The inclusion of two time scales in a dynamical system has the advantage of allowing the reduction of the system which may become, in this way, mathematically more tractable. Otherwise, taking into account several patches and density-dependent migrations may lead to so complex models that only few general results can be obtained analytically. 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. 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, I present several applications of these aggregation methods in population and community dynamics:

- Prey-predator model incorporating individual behaviour for predators disputing captured preys according to a hawk-dove game at a fast time scale.
- Prey-predator model in a patchy environment with predator (resp. prey) density dependent prey (resp. predator) dispersal at a fast time scale.
- Host-parasitoïd community in a patchy environment (2D network) with fast migration.

Wednesday, July 18, 2:00 PM

Chairperson: Pierre Auger

The Genetic Control of Vector-Borne Diseases: The Role of Tranposable Elements as a Gene Drive System

Cláudio José Struchiner
Professor, Fiocruz, Brazil

We explain the role of gene drive systems as a necessary component of vector-borne control strategies based on the genetic modification of the vector. Exploring the available data from the genomes of the malaria vector, Anopheles gambiae, and the dengue vector, Aedes aegypti, we reconstruct the demographic history of different classes of transposable elements and discuss the implications of their use as gene drive systems of refractory genes for those diseases.

Thursday, July 19, 8:30 AM

Chairperson: Carlos A. Braumann

Optimal Control of Structured Epidemics and Public Prevention

Bedr'Eddine Ainseba

Directeur UFR Sciences et Modélisation, Université Victor Segalen Bordeaux 2, France

In this talk I will first discuss about the last developments of population dynamics control problems. Then to fix ideas I will focus my talk on the optimal screening strategies in age structured epidemic models of SIR type. In this program the control is quantified by the rate performed by the health institutions, while the success of the program depends on the functional response of the population and on the time for detecting the disease within an individual. The cost function to be minimized is a combination of several quantities which include the social cost of the disease, the cost of the vaccination program and the cost of the treatment. The last part of my talk will be dedicated to the introduction of a dynamical economic model of Ramsey type, where the labor population is affected by an infectious disease. To control the spread of this disease, the government has the possibility to set up a screening procedure. We will study the optimal balance between the economical problem consisting in the maximization of the discounted sum of instantaneous utility and the classical social problem consisting in minimizing the number of infected individuals. Using the Pontryagin's maximum principle we will show how the level of economic development, the price of the screening campaign and the price of medications affects the dynamic of public intervention.

Thursday, July 19, 2:00 PM

Chairperson: J. F. Fontanari

Thoughts about Animal Motion from a Physicist's Perspective: Epidemics Spread and Related Problems

Vasudev M. (Nitant) Kenkre

Distinguished Professor of Physics and Director, Consortium of the Americas for Interdisciplinary Science, University of New Mexico, USA

Simple thoughts about the motion of living objects ranging from bacteria to rodents as it affects phenomena such as the spread of epidemics, e.g., the Hantavirus and the West Nile virus, will be presented from a physicist's perspective. Spatiotemporal patterns in bacteria in a Petri dish, refugia of epidemics in a landscape, and patterns arising from nonlinear competitive interactions will be among the subjects discussed. The work has been done in collaboration with a number of field biologists and theoretical physicists and is supported by the NSF, NIH and DARPA.

Friday, July 20, 8:30 AM

Chairperson: Eduardo Massad

Emergence of spatial patterns in pollinator-herbivore-plant models

Faustino Sanchez Garduño

Professor, Department of Mathematics, National Autonomous University of Mexico, Mexico

In this talk we explore the emergence of spatial ordered structures in the densities of three interacting populations. The interaction pollinator-plant is obligated for the plant and facultative for the pollinators population; meanwhile the interaction herbivore-plant can be seen as a predator-prey interaction. The existence of the herbivore diminish the rate of pollinator-plant encounters. The model considers Holling response of type II and IV and linear diffusion terms for the herbivore and pollinator populations.

Friday, July 20, 11:00 AM

Chairperson: Faustino Sanchez

The influence of climate on vector-borne infections

Eduardo Massad

Professor, School of Medicine, University of São Paulo, Brazil

In this work we analyse the impact of climate changes on the dynamics of vector-borne infections. After a brief and historical introduction to the theory of insect transmitted infections we present a model that describes the impact of global warming on the dynamics of malaria. We demonstrate that there is a clear temperature threshold above which the basic reproduction ratio for malaria crosses the transmission threshold upwards, becoming established in a susceptible population. The model consists in a dynamics that coupled the mosquitoes' life cycle with that of malaria transmission, in which the parameters are explicit functions of the local temperature. Next, we present a model for the impact of climatic factors in the spread of dengue in Singapore and in the city of Santos, Brazil. The model reproduces the actual epidemics with good accuracy and a simulation of the optimum control strategy for control is performed. The model derived control strategy was implemented in Singapore and the 2005 epidemic was controlled.

Massad, E. & Forattini, O.P. Modelling the temperature sensitivity of some physiological parameters of epidemiologic significance 1998. Ecosystem Health, 4(2): 119-129.

Burattini, M.N., M. Chen, A. Chow, F. A. B. Coutinho, K. T. Goh, L. F. Lopez, S. Ma and E. Massad. 2007. Modelling the control strategies against dengue in Singapore. Epidemiology and Infection, Forthcoming article, doi: 10.1017/S0950268807008667, Published online by Cambridge University Press 31 May 2007.

SESSIONS

(Auditoriums 1, 2 and 3)

ORAL PRESENTATIONS

Monday, July 16

Session: Dynamics and Control of Infectious Diseases – I Chairperson: Lourdes Esteva	Time: 14:00 Auditorium: 1
14:00-14:20	Results and Challenges on HIV Transmission Dynamics *Abba Gumel*
14:20-14:40	A cellular automata model to describe the dynamical aspects of parasitaemia in the blood cycle of Malaria *Rita M. Zorzenon dos Santos, Suani T. R. Pinho, Claudia P. Ferreira and Priscila C. A. da Silva*
14:40-15:00	A theoretical framework for biological control *Nik Cunniffe, Doug Bailey and Chris Gilligan*

Session: Ecosystem Dynamics – I Chairperson: Maria Florencia Carusela	Time: 14:00 Auditorium: 2
14:00-14:20	The response of *Drosophila melanogaster* to modeled odor plumes with different shapes *Lia Hemerik, Marjolein Lof and Maarten de Gee*
14:20-14:40	Analytical models of forest dynamics and tree species coexistence: from field data to theory *Miguel A. Zavala, Pablo Gómez Mourelo, Oscar Ângulo and Rafael Bravo de la Parra*
14:40-15:00	Consequences of increase the CO2 atmospheric for plants' ecology *Marlon Luiz Hneda and Luis Augusto Trevisan*

Session: Mathematics of Physiology – I	Time: 14:00
Chairperson: Jeong-Mi Yoon	Auditorium: 3
14:00-14:20	Mitochondrial energy metabolism modelling *Jean-Pierre Mazat and Christine Nazaret*
14:20-14:40	Mathematical modelling of cell population dynamics in the colonic crypt and in colorectal cancer *Matthew D. Johnston, Philip K. Maini, S. Jonathan Chapman, Carina M. Edwards and Walter F. Bodmer*
14:40-15:00	BV compactness and convergence of a numerical scheme to a nonlinear kinetic model of Tumor-Immune cell interactions *Bouchra Aylaj and Ahmed Noussair*

Session: Predator-Prey/Multi Systems – I	Time: 15:10
Chairperson: Magno Enrique Mendoza Meza	Auditorium: 1
15:10-15:30	Allee Effects in the Pattern Formation in Prey-Predator Systems *Denílson J. Seidel, Luiz Alberto D. Rodrigues and Diomar C. Mistro*
15:30-15:50	On the Fractional Predator-Prey Model *R. Figueiredo Camargo, E. Capelas de Oliveira and F. A. M. Gomes*
15:50-16:10	A Convex Approach for Controlled Lotka Volterra Multi-Species Models *S. Palomino Bean, D. F. Coutinho and K. A. Barbosa*
16:10-16:30	Spontaneous Emergence of Spatial Patterns in a Predator-Prey Model *M. V. Carneiro and I. C. Charret*

Session: Meta-populations – I	Time: 15:10
Chairperson: Cláudia Pio Ferreira	Auditorium: 2
15:10-15:30	Modeling indirect transmission of pathogens between host populations sharing environment *Ezanno P. and Lurette A.*
15:30-15:50	Scylla and Charybdis in the Virus World: revisiting Eigen's prebiotic information crisis *Daniel de A. M. M. Silvestre and José F. Fontanari*
15:50-16:10	The effect of spatial scale on meta-population patch models for infectious disease in the United Kingdom *Iain Barrass, Ian Hall and Steve Leach*

Tuesday, July 17

Session: Mathematics of Infectious Diseases – I Chairperson: Abba Gumel	Time: 9:40 Auditorium: 1
9:40-10:00	Qualitative Study of Transmission Dynamics of Antibiotic-resistant Malaria *Lourdes Esteva and Abba Gumel*
10:00-10:20	Models to directly transmitted diseases based on fuzzy rules *Laécio C. de Barros, Renata Z. G. de Oliveira , Maria Beatriz F. Leite and Rodney C. Bassanezi*
10:20-10:40	A Simple SI Model with Two Age Groups and Its Applications to HIV *Roxana López, Yang Kuang and Abdessamad Tridane*

Session: Ecosystem Dynamics – II Chairperson: Ezio Venturino	Time: 9:40 Auditorium: 2
9:40-10:00	Modelling transmission of Toxoplasma sp. in Amazonia *A. Bourassi, M. Langlais, B. Sancho and P. Silan*
10:00-10:20	The ecotoxicology of the zooplankton-cyanobacteria interaction *Elise Billoir, Aloysio da S. Ferro Filho, Marie-Laure Delignette-Muller and Sandrine Charles*
10:20-10:40	Sublethal toxic effects in a simple aquatic food chain *B.W. Kooi, D. Bontje, G.A.K van Voorn and S.A.L.M. Kooijman*

Session: Computational Biology in Clinical and Ecological Trials – I Chairperson: Ivanka Horova	Time: 9:40 Auditorium: 3
9:40-10:00	Modeling Uncertainty and Data Quality *José-Maria Caridad y Ocerin, Josef Fiala, Jan Ministr, Michal Hejc and Jirí Hrebícek*
10:00-10:20	ARROW - Assessment of ecological state of surface waters in the Czech Republic *Jaroslav Rácek, Danka Némethová, Jan Hodovský and Jirí Jarkovský*
10:20-10:40	Analytical Internet tool for cancer epidemiology in the Czech Republic - a computational base for predictive risk models *Ladislav Dusek, Jirí Hrebícek, Jan Zaloudík, Rostilav Vyzula, Jana Koptíková, Miroslav Kubásek, Jan Muzík, Tomás Pavlík, Jitka Abrahámová and Jirí Vorlícek*

Session: Dynamics and Control of Infectious Diseases – II Chairperson: Raymundo Soares de Azevedo Neto	Time: 11:00 Auditorium: 1
11:00-11:20	On the Modelling of the West Nile *Gustavo Cruz-Pacheco, Lourdes Esteva and Cristóbal Vargas*
11:20-11:40	Modelling the effects of temporary immune protection and vaccination against infectious diseases *Silvia Martorano Raimundo, Hyun Mo Yang and Alejandro B. Engel*
11:40-12:00	Optimal Control of Mosquito *Aedes aegypti* by the Sterile Insect Technique *Roberto Carlos Antunes Thomé*
12:00-12:20	Epidemiological Characteristics of Lyme-Simile in Children *Saulo D. Passos, Edison Luiz Durigon, Rosa Estela Gazeta, Maria do Rosário Latorre, Giancarla Gauditano and Natalino H.Yoshinari*

Session: Stochastic/Numerical Approaches – I Chairperson: Oscar Angulo	Time: 11:00 Auditorium: 2
11:00-11:20	Animal growth in random environments *Carlos A. Braumann and Patrícia Filipe*
11:20-11:40	Model of Two Dynamic Competing Species *David Zavaleta Villanueva*
11:40-12:00	Probabilistic cellular automata describing a predator-prey system and the dynamics of an epidemic *Tânia Tomé*
12:00-12:20	Analysis of the outputs of a simulator for the propagation of powdery mildew over vine with a SEIR model *Jean-Baptiste Burie, Agnès Calonnec and Michel Langlais*

Session: Spatial Diffusion of Vectors/Infections Chairperson: Norberto A. Maidana	Time: 15:10 Auditorium: 1
15:10-15:30	Optimal Control of Mosquito Population Applying Insecticide *Anderson Luis Albuquerque de Araujo and José Luiz Boldrini*
15:30-15:50	Dengue: Minimum Parameters and the Endemic State *Líliam C. C. Medeiros, César A. R. Castilho, Wayner V. Souza and Carlos F. Luna*
15:50-16:10	*Aedes aegypti* population model for analysis of dengue control strategies in Rio de Janeiro, Brazil *P. M. Luz, C. T. Codeco, D. Valle, J. Medlock, A. Galvani and C. J. Struchiner*
16:10-16:30	Modelling demographic processes of wild mosquito populations *Papa Ibrahima Ndiaye, Dominique Bicout and Philippe Sabatier*

Session: Meta-populations – II	Time: 15:10
Chairperson: Ezanno Pauline	Auditorium: 2

15:10-15:30	The Role of Spatial Refuges in Coupled Map Lattice Model for Host-Parasitoid Systems *Diomar C. Mistro, Luiz A. D. Rodrigues and Maria C. Varriale*
15:30-15:50	Regions of endemicity for disease in metapopulations *M. Jesse, S. Davis, P. Ezanno and J.A.P. Heesterbeek*
15:50-16:10	Application of parasite-host-fodder plant system dynamics to the description of larch bud moth population cycles *Lev V. Nedorezov*

Session: Predator-Prey/Multi Systems – II	Time: 17:00
Chairperson: Luiz Alberto Díaz Rodrigues	Auditorium: 1

17:00-17:20	Predator-prey systems subject to threshold policy with hysteresis *Magno Enrique Mendoza Meza, Amit Bhaya and Eugenius Kaszkurewicz*
17:20-17:40	Mathematical Analysis of a Diffusive Predator-Prey System with Non-Local Intra-Specific Competition *Mehran Sabeti and Luiz Antonio Ribeiro de Santana*
17:40-18:00	Three Mathematical Models for Endemic Cutaneous Leishmaniasis Considering Human, Animal Host and Vector Populations *E. De la Pava, C. Castillo-Chávez and J. Montoya-Lerma*

Session: Mathematics of Physiology – II	Time: 17:00
Chairperson: Christine Nazaret	Auditorium: 2

17:00-17:20	Mathematical Modeling and Simulation of the Immune System - Dynamics in Human Herpesvirus-6 Infection *Jeong-Mi Yoon and Mandri Obeyesekere*
17:20-17:40	Impact of initial antiretroviral regimens in viral load and CD4+ cell counts in patients living with HIV *Claudia Courtouké, Aluísio Cotrim Segurado and Joyce da Silva Bevilacqua*
17:40-18:00	Towards understanding Natural Killer cell development in the bone marrow: combining experiments and mathematical modeling *Marjet Elemans, Maria Johansson, Eleftheria Rosmaraki, Klas Krre and Ramit Mehr*

Wednesday, July 18

Session: Mathematics of Infectious Diseases – II Chairperson: Slimane Ben Miled	Time: 9:40 Auditorium: 1
9:40-10:00	The deterministically chaotic dynamics in a realistic multi-strain dengue model with temporary cross-immunity *Maíra Aguiar and Nico Stollenwerk*
10:00-10:20	Modeling approach to explore influenza pandemic profiles and their associated interventions *Elisabeta Vergu, Solen Kerneis, Rebecca F. Grais, Laurent Coudeville, Pierre-Yves Boëlle and Antoine Flahault*
10:20-10:40	Bifurcations in an SEIQR Model for Childhood Diseases *David J. Gerberry*

Session: Ecosystem Dynamics – III Chairperson: Luis Augusto Trevisan	Time: 9:40 Auditorium: 2
9:40-10:00	Spiders as a biological controllers of the Langa Astigiana vineyards *Samrat Chatterjee, Marco Isaia and Ezio Venturino*
10:00-10:20	Modeling the effect of chemical information and resources abundance on colonization of an orchard by *Drosophila melanogaster* *Marjolein Lof, Lia Hemerik and Maarten de Gee*
10:20-10:40	A Mathematical Model of Pirarucus (Arapaima gigas) Population Dynamics *Eliane dos Santos de Souza Coutinho, Luiz Bevilacqua and Helder Lima de Queiroz*

Session: Computational Biology in Clinical and Ecological Trials – II Chairperson: Jan Ministr	Time: 9:40 Auditorium: 3
9:40-10:00	Hazard function for cancer patients and cancer cells dynamics *Ivanka Horová, Zdenek Pospísil and Jirí Zelinka*
10:00-10:20	Biological protection of plants — a model and its parameters *Zdenek Pospísil and Václav Pink*

Session: Dynamics and Control of Infectious Diseases – III Chairperson: Cristobal Vargas Jarillo	Time: 11:00 Auditorium: 1
11:00-11:20	Seasonal Effects on the West Nile Virus Infection *Gustavo Cruz-Pacheco, Lourdes Esteva and Cristóbal Vargas*
11:20-11:40	Travelling Waves Describing the Spatial West Nile Virus Epidemic *Lourdes Esteva, Norberto Anibal Maidana and Hyun Mo Yang*
11:40-12:00	Yellow fever vaccination in the absence of infection risk: How to decide? *Claudia Codeço, Paula Luz, Flavio Coelho, Alison Galvani and Claudio Struchiner*
12:00-12:20	Extremely low numbers of *Schistosoma mansoni* eggs and the difficulties for standardization of parasitological diagnostic methods *Cristiane Ceruti Franceschina, Candida Fagundes Teixeira and Carlos Graeff-Teixeira*

Session: Stochastic/Numerical Approaches – II Chairperson: Jean-Baptiste Burie	Time: 11:00 Auditorium: 2
11:00-11:20	A Numerical Method for Separable Models with Finite Maximum Age *Oscar Angulo, J.C. López-Marcos and M.A. López-Marcos*
11:20-11:40	The ultimate age-profile in an asymptotically linear population model *Jordi Ripoll and Mimmo Iannelli*
11:40-12:00	A Tinnitus´ Models Using McKendrick´s Equations *M. O. Pinho, A. A. Azevedo, R. R. Figueiredo and A. C. M. Alvim*

Session: Dynamics and Genetics of Structured Populations – I Chairperson: Paulo Roberto de Araujo Campos	Time: 15:20 Auditorium: 1
15:20-15:40	A Theoretical Framework to Understand the Emergence of Drug Resistance: A Case Study of Tuberculosis *Domingos Alves, Aquino L. Espíndola and Henrique Fabrício Gagliardi*
15:40-16:00	Stochastic bottlenecks and Muller's ratchet *Leonardo P. Maia*
16:00-16:20	Modelling of a One-sex Age-Structured Population *Vladas Skakauskas and Sarunas Repsys*
16:20-16:40	Control of spatially structured pathogenic bacterial populations by probiotics: modeling and simulation of a mixed-culture biofilm system *Hermann J. Eberl, Hedia Fgaier and Hassan Khassehkhan*

Session: Mathematical Modeling of Fisheries Management – I Chairperson: Nadia Raissi	Time: 15:20 Auditorium: 2
15:20-15:40	The optimal spatial distribution of small and large scale fisheries *P. Cartigny, W. Gómez and H. Salgado*
15:40-16:00	Some bio-economic models of fisheries: Stabilizability of a controlled system and Optimal spatial distribution *Rachid Mchich*
16:00-16:20	The study of the viability domain for a fishing problem with reserve *P. Cartigny, M. Jerry and A. Rapaport*
16:20-16:40	A dynamical fishing model on many zones: Simulation on the sardine stock in Atlantic Moroccan cost *Najib Charouki*

Thursday, July 19

Session: Mathematics of Infectious Diseases – III Chairperson: Elisabeta Vergu	Time: 9:40 Auditorium: 1
9:40-10:00	Modelling disease spreading in symbiotic communities *Mainul Haque and Ezio Venturino*
10:00-10:20	Influenza A evolution paradigm shift: how can we explain the recurrence of influenza A epidemics in humans? *Sébastien Ballesteros, Marc Senneret, Elisabeta Vergu and Bernard Cazelles*
10:20-10:40	Rich dynamics in multi-strain epidemiological models: from evolution towards criticality to reinfection threshold and new deterministically chaotic attractors *Nico Stollenwerk*

Session: Ecosystem Dynamics – IV Chairperson: Lia Hemerik	Time: 9:40 Auditorium: 2
9:40-10:00	Robust feedback control design for a nonlinear wastewater treatement model *Mustapha Serhani*
10:00-10:20	Size-Structured Modelling of Aquatic Ecosystems – with Emphasis on Trophic Ladder Organisation *Martin Pedersen and Ken H. Andersen*
10:20-10:40	Variable Interactions between Freshwater Amphipods and Algae *M. F. Carusela, F. Momo and L. Romanelli*

Session: Computational Biology in Clinical and Ecological Trials – III	Time: 9:40
Chairperson: José Luiz Boldrini	Auditorium: 3
9:40-10:00	Mixed immunotherapy and chemotherapy of tumors: Feedback design and model updating schemes *S. Chareyron and M. Alamir*
10:00-10:20	Influence of the fractal dimension of a fungal colonial area in its population dynamics *Lilian Akemi Kato*
10:20-10:40	Application of the Fuzzy Sets Theory in the Prediction of the Pathological Stage of the Prostate Cancer *Graciele Paraguaia Silveira and Laércio Luis Vendite*

Session: Dynamics and Control of Infectious Diseases – IV	Time: 11:00
Chairperson: Gustavo Cruz-Pacheco	Auditorium: 1
11:00-11:20	Estimating rubella incidence from deterministic models *Marcos Amaku and Raymundo S. Azevedo*
11:20-11:40	Optimal Control Strategy of Malaria Vector Using Genetically Modified Mosquitoes *L. Bevilacqua, M. Rafikov and A. P. P. Wyse*
11:40-12:00	The Multiple Faces and Feats of Diffusion Models in Biology: From Bets to Bits and Beyond *Wilson Castro Ferreira Jr.*
12:00-12:20	Epidemiological signatures of alternative cross-immune responses to BCG *Natalia B. Mantilla-Beniers and Gabriela M. Gomes*

Session: Genetics – I	Time: 11:00
Chairperson: Rodrigo Martinez	Auditorium: 2
11:00-11:20	Fixation in haploid populations exhibiting density dependence *Christopher Quince and Todd L. Parsons*
11:20-11:40	The effects of the intersexual competition on the sex-ratio of hermaphrodite *Slimane Ben Miled and Amira Kebir*
11:40-12:00	Genetic linkage and the subversion of natural selection *Philip Gerrish, Alexandre Colato and Paul Sniegowski*

Session: Dynamics and Genetics of Structured Populations – II Chairperson: Domingos Alves	Time: 15:20 Auditorium: 1
15:20-15:40	Patterns of genetic variation in pathogen populations *Paulo R. A. Campos and Isabel Gordo*
15:40-16:00	Inverse density dependence in the evolution of communication *José Fernando Fontanari*
16:00-16:20	Parameter identifiability and identification of a hierarchical epidemiological model *Béatrice Laroche and Suzanne Touzeau*
16:20-16:40	Speed of adaptation in structured populations *Edilson de A. Gonçalves, Alexandre Rosas, Viviane M. de Oliveira and Paulo R. A. Campos*

Session: Mathematical Modeling of Fisheries Management – II Chairperson: Mounir Jerry	Time: 15:20 Auditorium: 2
15:20-15:40	Analysis of management measures effects on the fisheries *C. Jerry, P. Auger and N. Raïssi*
15:40-16:00	Endemic steady states and travelling wave solutions for a class of age-structured epidemic models *Ducrot Arnaud*
16:00-16:20	New contributions to fish growth studies: from classical parameter estimation to new stochastic growth models *Russo T., Magnifico G., Baldi P., Parisi A., Pulcini D., Corsi E., Antonacci L., Romei J., Mariani S. and Cataudella S.*

Friday, July 20

Session: Mathematics of Infectious Diseases – IV	Time: 9:40
Chairperson: Maira Aguiar Freire dos Santos	Auditorium: 1
9:40-10:00	The role of sexually isolated groups in two-sex demographic and epidemic logistic models with non-linear mortality *Daniel Maxin and Fabio A. Milner*
10:00-10:20	Epidemiological modelling for managed herds *Sébastien Gaucel, Béatrice Laroche, Suzanne Touzeau and Elisabeta Vergu*
10:20-10:40	Conditions for the existence of a convergence stable strategy in a discrete stage-structured population model *Qingguo Zhang and Li Xu*

Session: Genetics – II	Time: 9:40
Chairperson: Christopher Quince	Auditorium: 2
9:40-10:00	Y-linked bisexual branching models *M. González, R. Martínez and M. Mota*
10:00-10:20	Proximate individual based model for sex-ratio dynamics of an hermaphrodite population *A. Kebir, S. Ben Miled and M. L. Hbid*
10:20-10:40	The Evolution of the Assortative Mating in Sympatric Speciation *Fabiano Ribeiro and Nestor Caticha*

POSTER PRESENTATIONS
(Hall)

Wednesday, July 18, 17:00

	Session: Mathematics of Infectious Diseases
P01	New Lyapunov functions for epidemic models with variable population size *Cruz Vargas De Leon*
P02	Global stability of host-vector disease models via the Lyapunov method *Cruz Vargas De Leon*
P03	Dengue Epidemics: *Urbi et Orbi* *Lucy Tiemi Takahashi, Wilson Castro Ferreira Jr. and Luis Alberto D'Afonseca*
P04	Assessing the effects of BCG vaccine on the transmission of Tuberculosis *Marcio Rodrigues Sabino, Silvia Martorano Raimundo and Hyun Mo Yang*

	Session: Dynamics and Control of Infectious Diseases
P05	Mathematical Model to study the spread of information from educative campaigns and rumors *Clarice Gameiro da Fonseca Pachi, Silvia Martorano Raimundo, Eduardo Massad and Marcelo Nascimento Burattini*
P06	Modeling bovine brucellosis control by vaccination *Ricardo Augusto Dias, Fernando Ferreira, José Soares Ferreira Neto and Marcos Amaku*
P07	Bayesian Estimation of SEIR Models *Luiz Koodi Hotta*
P08	A mathematical model for *Aedes aegypti* life cycle and epidemics control *Raquel Martins Lana, Leandro Lhermsdorff, Romuel F. Machado, Américo T. Bernardes and Sérvio Pontes Ribeiro*
P09	Modeling the impact of sexual health education and behavior of young women on vertical HIV transmission: the demographic question *Silvia Martorano Raimundo, Hyun Mo Yang and Alejandro B. Engel*

	Session: Meta-populations
P10	Agent-based Model for Migration of Individuals in Metapopulation *Aquino L. Espíndola and Domingos Alves*
P11	Turing's instabilities and sinchronism in hierarchical models *Flávia Tereza Giordani and Jacques A. L. da Silva*

	Session: Ecosystem Dynamics
P12	Population dynamics of a prey-predator model in a cassava system *A. C. S. Vilcarromero, J. F. R. Fernandes and O. Bonato*
P13	Interactions between traps and other reservoirs in *Aedes aegypti* oviposition behavior *Arthur Weiss, Flavio Coelho and Claudia Codeço*
P14	A mathematical model to describe the lethargic crab disease *C. P. Ferreira, M. R. Pie, L. Esteva, P. F. A. Mancera, W. A. Boeger and A. Ostrensky*
P15	Estimation of time lag in a reaction of self-regulative mechanisms: application of Hutchinson model for fitting of diamondback moth population dynamics *Lev V. Nedorezov, Bernhard L. Lohr and Dinara L. Sadykova*
P16	Prey-predator Modeling of CO2 Atmospheric Concentration *Luis A.Trevisan, Fabiano M.M. Luz and Roberto A. Clemente*
P17	Modeling dog population control *Marcos Amaku, Ricardo A. Dias and Fernando Ferreira*
P18	Fuzzy Clustering and Allometry to Freshwater Stingrays of the Rio Negro Basin *Karla Tribuzy, Maria Lúcia G. Araújo and Rodney Bassanezi*
P19	Modelling the regional dynamics of annual plants *Stephen J. Galsworthy, Matthias C. Wichmann, Rosie S. Hails, James M. Bullock and Philip K. Maini*
P20	Intraguild predation and foraging dynamics in blowflies *Thais Irene Souza Riback and Wesley Augusto Conde Godoy*
P21	Investigation of population dynamics of seabob shrimp *Xiphopenaeus kroyeri* in Ubatuba Bay, Brazil by Artificial Neural Networks *Vinicius Tragante do Ó, Fernando Luis Medina Mantelatto, Adilson Fransozo, Rogério Caetano da Costa e Renato Tinós*

	Session: Spatial Diffusion of Vectors/Infections
P22	Epidemic Spread in Populations at Demographic Equilibrium *Karen R. Ríos-Soto, Carlos Castillo-Chavez, Michael G. Neubert, Edriss S. Titi and Abdul-Aziz Yakubu*
P23	Semiparametric generalized additive model for ordinal response variables applied to epidemiology spatial data *Liciana Vaz de Arruda Silveira, Ana Carolina Cintra Nunes and Ricardo Carlos Cordeiro*
P24	Approximate reduction of non-linear discrete models with two time scales *M. Marvá, R. Bravo de la Parra, E. Sánchez and L. Sanz*
P25	An EDP model with fuzzy dispersion parameter applied on foot-and-mouth disease in bovines *Maristela Missio, Laécio Carvalho de Barros and Marina Ribeiro Barros Dias*
P26	A Fast nEMD Algorithm and its applications on Population Dynamics *Raphael G. D. d'A. Vilamiu and Wilson C. Ferreira Jr.*

Thursday, July 19, 17:00

	Session: Dynamics and Genetics of Structured Populations
P01	Mean-field analysis of a replicator dynamics in a lattice *Anderson A. Ferreira and José F. Fontanari*

	Session: Mathematics of Physiology
P02	A Mathematical Model for the Dynamics of Rotavirus Infection *Andressa Pinheiro and Hyun Mo Yang*
P03	An environment for knowledge discovery on eukaryotic cell cycle and population dynamic regulations – a multiscale model *Beatriz Stransky, Rogerio Rizzio, Walter Nestor Trepode and Junior Barrera*
P04	Dynamics of the Interaction Between the Immune System and Measles Systemic Infection *Elias Tayar Galante and Hyun Mo Yang*
P05	Conceptual spatio-temporal models of marine viral infections *Ivo Siekmann, Horst Malchow and Ezio Venturino*
P06	The Immune Response against HIV-1 as an Optimal Process *F. L. Biafore and C. E. D'Attellis*
P07	Modelling the Interaction between *Trypanosoma cruzi* and the Immune System *Licia Silva Oliveira and Hyun Mo Yang*
P08	A dynamic model to evaluate antiretroviral therapy for HIV type 1 *Márcia Perez Resende Oliveros, Clarice Gameiro da Fonseca Pachi, Silvia Martorano Raimundo and Luis Fernandez Lopez*
P09	A Reliability Analysis of The Emergency Diesel Generators of a Four Loops PWR Nuclear Power Plant Under Aging and Perfect Repair by McKendrick Equations *Ion Maghali Santos de Oliveira, Marcos Oliveira de Pinho and Paulo Fernando Ferreira Frutuoso e Melo*
P10	Computer model for stem-cell action in the maintenance and regeneration of living tissues *Neila M. Gualberto and Carlos H. C. Moreira*
P11	Methodology of Rehabilitation of the muscle cuadriceps for individuals with knee artrosis using logical Fuzzy *Nelson Herrera Gómez and Liliam C. C. Medeiros*
P12	Modeling the Interaction between Immune System and bacterium Streptococcus and Staphylococcus *Yu Jun and Hyun Mo Yang*

	Session: Stochastic/Numerical Approaches
P13	Small World Effect Using Cellular Automata: A Case Study for Epidemics *Henrique Fabrício Gagliardi and Domingos Alves*
P14	A cellular automata model to describe the postfeeding larval dispersion in blowfly species *Ernesto A. B. F. Lima, Cláudia P. Ferreira, Andressa M. Bernardes and Wesley A.C. Godoy*
P15	A stochastic simulation of a yellow fever outbreak (Buenos Aires, 1871) *M. L. Fernández, M. Otero, N. Schweigmann and H. Solari*
P16	A Bayesian Approach for the Stochastic Fowler's Population Growth Model *Marinho G. Andrade, Selene Loibel and Monica F. B. Moreira*

	Session: Computational Biology in Clinical and Ecological Trials
P17	A simple model for terraccumulation of antibiotics and the development of antimicrobial resistance in environmental bacterial populations *H. J. Eberl*
P18	An individual-based model for trophic interactions and species assembly *Henrique Corrêa Giacomini*
P19	Multiscale modelling for the virotherapy of avascular tumours *L. R. Paiva, S. C. Ferreira Jr. and M. L. Martins*
P20	Prey preference, food web structure and long term stability *Lucas Del Bianco Faria and Michel Iskin da Silveira Costa*
P21	Dynamics Population of Microorganisms applied to the Food Conservation *Roberta Regina Delboni and Hyun Mo Yang*
P22	Modeling species competition with intermingled basins *Rodrigo Frehse Pereira, Sandro Ely de Souza Pinto, Ricardo Luiz Viana, Iberê Luiz Caldas and Celso Grebogi*

	Session: Predator-Prey/Multi Systems
P23	A short history of mathematical population dynamics *Bacaer Nicolas*
P24	The stability of the Lotka Volterra system with uncertainty parameters - Monte Carlo simulation and Fuzzy sets approach *José Raimundo de Souza Passos and Laécio Carvalho de Barros*
P25	A fuzzy approach for a predator-prey model connected parasitism *Magda da Silva Peixoto, Laécio Carvalho de Barros and Rodney Carlos Bassanezy*

ABSTRACTS

ORAL
and
POSTER
PRESENTATIONS

Results and Challenges on HIV Transmission Dynamics

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Since its emergence in the 1980s, the HIV/AIDS pandemic continue to pose an unprecedented threat to global health and human development. An estimated 34- 46 million people are currently living with the virus, and over 20 million people have died due to AIDS-related causes over the last two decades. In addition to the enormous socio-economic burden it imposes, AIDS is now the leading cause of death in sub-Saharan Africa, and has cut the life expectancy in a number of countries in this region.

The talk will address some of the modelling issues and challenges associated with evaluating established and new strategies for curtailling the spread of HIV in Africa, such as the use of (i) antiretroviral drugs; (ii) potential (imperfect) vaccine, (iii) condom use and male circumcision. The talk will assess the impact of one other issue that is crucially important to the spread of HIV in Africa, namely the co-infection of HIV with other curable diseases such as mycobacterium tuberculosis and malaria.

BV compactness and convergence of a numerical scheme to a nonlinear kinetic model of Tumor-Immune cell interactions

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Keywords:Population dynamics, Kinetic models, transport projection scheme, Helly compactness theorem

We consider a class of model with physiological structure of a immune cell population consisting of n interacting species. For each population, its necessary to identify the most important set of activities. Each activity differs from population to population and is tagged by a suitable dimensionless variable x, that denotes the intensity with which the activities are preformed. The value x=1 corresponds to the highest value of the peculiar activities of the cell, while x=0 correspond the complet funtionnal inhibition (no activities). The governing equations are

$$\begin{cases} \dfrac{\partial f_i}{\partial t} + \dfrac{\partial}{\partial x}\big(v_i f_i\big) + \mu_i(P(t),x) f_i = \displaystyle\sum_{j=1}^{n} Q_{ij}(f_i,f_j), \\ v_i(0) f_i(t,0) = \displaystyle\int_0^1 \beta_i(P(t),x) f_i(t,x)dx, \\ f_i(t,0) = f_{i0}(x), \end{cases} \tag{1}$$

where the operator of encounters $Q_{ij} = Q_{ij}^{+} - Q_{ij}^{-}$, with the gain part is

$$Q_{ij}^{+}(f_i,f_j) = \int_0^1 \int_0^1 \eta_{ij}(y,z)\psi_{ij} f_i(t,y) f_j(t,z)dydz$$

and the loss part

$$Q_{ij}^{-}(f_i,f_j) = f_i(t,x)\int_0^1 \eta_{ij}(x,y) f_j(t,y)dy.$$

The functions v_i, $\mu_i(P,x)$ and $\beta_i(P,x)$ denote the natural vital (growth, brith and death) rates of the i^th species. The encounter rate between pairs of individuals, of the i^{th} population in the state x and of the j^{th} population in the state y is identified by the term $\eta_{ij}(x,y)$. The probability that an individual of the i^{th} population in the state y ends up in the state x conditionally to an encounter with an individual of the j^{th} population in the state z, has a density denoted by $\psi_{ij}(y,z,x)$. The approximate solution $f^{k,\Delta}$, generated by the scheme, at time t_n, is denoted $f^{k,n}(x) = f^{k,\Delta}(t_n,x)$, and $f^{k,n}(.)$ is built in such following way formulation:

$$\begin{cases} f_i^{k,n+1} = f_i^{k,n} - \dfrac{\Delta t}{\Delta x}\left(v_{i+1/2}^k f_i^{k,n} - v_{i-1/2}^k f_{i-1}^{k,n}\right) \\ \quad + \Delta t \displaystyle\sum_{l=1}^{N_f} Q_i^{kl,n} - \mu_i^{k,n} f_i^{k,n} \\ \quad + \dfrac{a_{i+1/2}}{2}\left(f_{i+1}^{k,n} - f_i^{k,n}\right) - \dfrac{a_{i-1/2}}{2}\left(f_i^{k,n} - f_{i-1}^{k,n}\right) \\ \text{with} \\ v_{1/2} f_0^{k,n} = \displaystyle\sum_{i=1}^{I_L} \beta_i, f_i^{k,n}\Delta x \end{cases}$$

The coeficient $a_{i+1/2}$ are chosen to bound locally the influence of numerical viscosity.

The three estimates :(1) L^∞ bound, (2) a uniform total variation bound and (3) L^1-continuity in time of the approximate solution are established with a self-contained treatment of the stability and convergence properties.

Parameter estimation

We define the last squares cost functional

$$\begin{aligned} J(v,\beta,\mu) &= \sum_{\kappa} J^{\kappa}(v^{\kappa},\beta^{\kappa},m^{\kappa})) \\ &= \sum_{\kappa}\sum \left| \int u^{\kappa}(t_n)da - z_i^n \right|^2 da \end{aligned} \tag{2}$$

which is minimized over some set $V \times B \times M$ to be chosen later . In general there are two main difficulties involved in trying to implement this approach: the function u^κ must be computed numerically, and the infinite dimensional parameter set must be approximated by a finite dimensional set, for computational purposes, we seek to minimize

$$\begin{aligned} J^{\Delta}(v^{\Delta},\beta^{\Delta},\mu^{\Delta}) &= \sum_{\kappa} J^{\kappa,\Delta}(v^{\kappa,\Delta},\beta^{\kappa,\Delta},m^{\kappa,\Delta})) \\ &= \sum_{\kappa}\sum \left| \int u^{\kappa,\Delta}(t_n)da - z_i^n \right|^2 da \end{aligned} \tag{3}$$

over a set $V^\Delta \times B^\Delta \times M^\Delta$. A major concern in this class of problems is the convergence of the finite dimensional problem to the infinite dimensional one.

Modelling the effects of temporary immune protection and vaccination against infectious diseases

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Keywords: Epidemic Model; Vaccination; Booster; Vaccination coverage; Immunological memory.

Mathematical models have a long history of contributing to understanding the impact of vaccination. So much so that one of the earliest models in epidemiology dealt exactly with the impact of vaccination. Vaccination is a method for inducing immunity in the population in order to control the spread of the disease. However, infection can happen even in individuals that acquired immunity by ways of the disease itself or by vaccination, this is re-infection. Re-infection may occur without any clinical symptoms. In this way, re-infected individuals can become a repository of the virus and be the vehicle of its propagation. Even with concrete proof that re-infection is a real phenomenon, most health authorities have ignored it, since no clear re-vaccination strategy existed. Moreover, when statistical data is gathered, no distinction is made between first time infected individuals and re-infected ones. Lately, however, some countries have taken re-infection seriously and have started re-vaccination programs. In this work we build a mathematical model in which there is a distinction between first time infected individuals and re-infected ones. Also, we assume not only vaccination, but re-vaccination as well. With this and other assumptions, the situation is modeled by a system of coupled non-linear differential equation. We use qualitative analysis of this system to find equilibrium points. This will allow us to understand why even with vaccination and re-vaccination the virus cannot be totally eradicated.

References

[1] S. M. Raimundo; H.M. Yang; A. B. Engel Modelling the effects of temporary immune protection and vaccination against infectious diseases. Appl. Math. Comput. doi:10.1016/j.amc.2006.12.051, 2007

[2] M. G. Gomes; A. O. Franco; M. C. Gomes; G.F. Medley. The reinfection threshold promotes variability in tuberculosis epidemiology and vaccine efficacy. Proc. Biol. Sci. Mar 22;271(1539):617-23, 2004.

Speed of adaptation in structured populations

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Keywords: Adaptation, Clonal interference, Scale free.

Adaptation in microbe populations, in the simplest scenario, consists of rare beneficial mutations which through rapid selective sweeps reach fixation in the population. These fixation events are alternated by periods of low activity, where no beneficial mutation is fixed. Although the occurrence of deleterious mutations are much more likely, this simplistic view can be broken down upon the assumption of very large population sizes or high mutation rates. Upon these circumstances, the coexistence of distinct beneficial mutations becomes common. In asexual populations, where genomes carrying distinct mutations can not recombine to form a better adapted entity, this leads to a strong competition between distinct beneficial mutations in order to reach fixation with the ultimate loss of the remaining ones, a phenomenon which is known as clonal interference. The clonal interference results in longer fixation times and consequently a slower adaptation rate in asexual populations.

The phenomenon of clonal interference has been extensively studied for a long term. However, most of the advances were restricted to the assumption of non-structured populations. A first attempt to tackle the problem of structured populations was the analysis of the advantageous mutations fixation dynamics in a spatially structured asexual haploid population [1, 2]. It was shown that the beneficial mutation substitution rate in a spatially structured population is smaller than the one observed in non-structured populations. This is more pronounced as the adaptive mutation rate increases, exactly where clonal interference becomes more relevant.

However, to our knowledge, the role of network topology and structuring on the adaptive process of subdivided populations has not been analyzed yet, perhaps because of the belief that population structure does not influence the population dynamics. This is precisely what we have observed as long as neither extinction nor clonal interference are considered. Therefore, this contribution focuses on the competition between different clones for fixation, how it changes this scenario, and the topology affects the competition.

Here, we investigate the dynamics of fixation of beneficial mutations in a subdivided population, which has recently been introduced by Campos and Gordo [4] in the context of epidemiology. The subpopulations (demes) can exchange migrants among their neighbors. The migration network is assumed to have either a random graph or a scale-free topology. As expected from Maruyama's claim [3], the probability of fixation of a given beneficial mutation is not influenced by neither the rate at which individuals move from one deme to the other nor by the topology of the network. However, the same is **not** true when extinction occurs, since extinction creates an asymmetry in the probability of emigration and immigration. What is more, in the presence of extinction the probability of fixation depends on the topology. In addition, we have also observed that a topological dependence of the adaptive evolution of the population exists when clonal interference becomes effective.

References

[1] I. Gordo and P. R. A. Campos, Genetica **127**, 217 (2006).

[2] L. Perfeito, I. Gordo and P. R. A. Campos, Eur. Phys. J. B **51**, 301 (2006).

[3] T. Maruyama, Genetical Research **15**, 221 (1970).

[4] P. R. A. Campos and I. Gordo, J. Stat. Mech. **L12003** (2006).

Proximate individual based model for sex-ratio dynamics of an hermaphrodite population

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Keywords: Individual based model, Grouper, Density dependence, Sexual inversion, Temporal variability, sex-ratio.

In the Mediterranean sea, the *Epinephelus marginatus* populations show great variability in effective between the northern and southern part. Fluctuated environmental condition are closely linked with reproduction, in fact the recent literature argue for the density dependence of the sex reversing. The abundance of species seems to depend on the gender structure of the population.

For this protogynous species, the sexual inversion is not really a spontaneous mechanism ontogenic, *i.e.* which it would be held at a data size, but rather a plastic trait depending on social factors [3, 2].

Generally, to face this gender "conflict" [4], hermaphrodite can adjust the ratio of resources invested in mating in the male role versus the female role. This adjustment depends in selection pressures and environmental conditions [1].

An individual based model was developed to examine the effect of such behaviour on the population dynamics. Our motivations her is to study the juvenile sexual status of the grouper from the proximate point of view. In an attempt to define an approach that represent the physiological complexities of this species, we give the life cycle of the grouper (fig:1) and we provide a link between demographic processes and individual processes.

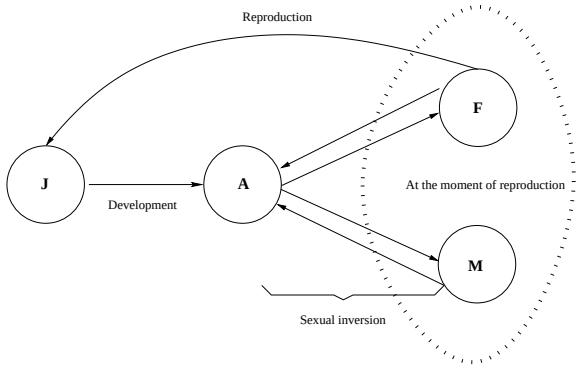

Figure 1: Life cycle of the grouper, J : Juvenile, A : Asexual, F : Female and M : Male

We developed a stochastic model, where population is treated as collections of individuals, each of which is represented by a set of variables: size, stage and sexual status. This temporally variable characteristics change as a function of the individual current state and the interaction with other individuals.

The growth (Size/age) is represented by the model Von Bertalanffy:

$$S(t) = S_\infty(1 - e^{-k(t-t_0)}).$$

After growth, we distinguish between two individuals having the same size s by a normal distribution $N(m, \sigma)$, where mean $m = S(S^{-1}(s) + 1)$.

The sexual status depends on the amount of competitive pressure experienced during the asexual growth, if the density of population is weak the individuals will tend to be females. Then we assume that an individual choose its reproduction role by a Bernoulli distribution, where the probability "to be a female" depend on the weighted total population size.

After simulations we notice that the observations answer to the questions raised by C. Chauvet [3]. For a low population density, the sex-ratio is in favour of the females; the mortality has a destabilizing effect on the population and for a variable mortality along time there are strong oscillations of the sex-ratio.

References

[1] E. L. Charnov, The Theory of Sex Allocation, Princeton, New Jersy: Princeton University Press.

[2] C. Chauvet, Etude de la croissance du mrou Epinephelus guaza (Linn, 1758) des ctes tunisiennes. Aquat. Living Resourc., 1: 277-288 1988.

[3] C. Chauvet, Le frai d'Epinephelus marginatus (Lowe, 1834) : seize annes d'observations in situ (1976 1992). Second International Symposium on the Mediterranean Groupers. P. Francour (ed), 10-13 May 2007, Nice, Nice University publ. 2007 (in press).

[4] Michiels, N. K. and Newman, L. J., Sex and violence in hermaphrodites. Nature, 391, 647 1998.

Aggregation of variables in population dynamics

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Keywords: aggregation of variables, reduced model, bifurcation, stability.

We present an overview of aggregation of variables methods. Aggregation of variables takes advantage of two time scales involved in the dynamics of a dynamical system to obtain a reduced model governing some global variables at a slow time scale. We present an application to a predatorprey Lotka-Volterra model in a two patch environment. Predator (resp. prey) dispersal is prey (resp. predator) density-dependent. Predators remain on a patch when there are many preys on that patch. When the predator density is large, the dispersal of prey is more likely to occur. We assume that prey and predator dispersal is faster than local predatorprey interaction on each patch. We take advantage of the two time scales in order to reduce the complete model to a system of two equations governing the total prey and predator densities. Using bifurcation analysis methods, we study the effects of the density-dependence of migration on the stability of the prey-predator system.

References

[1] Auger P. & Poggiale J.C.. Aggregation and Emergence in Systems of Ordinary Differential Equations. Math. Computer Modelling. Special issue "Aggregation and Emergence in Population Dynamics". P. Antonelli & P. Auger guest-Editors. 27(4), pp. 1-21, 1998.

[2] Auger P. et Bravo de la Parra R. Methods of aggregation of variables in population dynamics. Comptes-Rendus de lAcadémie des Sciences,Sciences de la Vie, 323, 665-674, 2000.

[3] Auger P., Bravo de la Parra R., Morand S. & Sanchez E. A predator-prey model with predators using hawk and dove tactics, Mathematical Biosciences, 177 & 178, pp. 185-200, 2002.

[4] Auger P., Kooi B., Bravo de la Parra R. and Poggiale J.-C. Bifurcation Analysis of a Predator-prey Model with Predators using Hawk and Dove Tactics. Journal of Theoretical Biology. 238 : pp. 597-607, 2006.

[5] Iwasa Y., Andreasen V., Levin S.A., 1987. Aggregation in model ecosystems I. Perfect aggregation. Ecol Model. 37: 287-302.

[6] Iwasa Y., Levin S.A., Andreasen V., 1989. Aggregation in model ecosystems II. Approximate aggregation. IMA. J. Math. Appl. Med. Bio. 6: 1-23.

Animal growth in random environments

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Keywords: growth models, stochastic differential equations, estimation, prediction, time to maturity.

The models used to describe the growth of animals in terms of their weight (or size) $X(t)$ at time t assume the form of a differential equation ([1],[2],[3]). One can see that the models commonly used, after a suitable change of variable $Y(t)=g(X(t))$ with g strictly increasing, can be written in the form

(1) $\mathrm{d}Y(t)/\mathrm{d}t = b(A-Y(t)), \quad Y(0)=y_0.$

The Bertalanffy-Richards model corresponds to the choice $g(x)=x^c$ with $c>0$ (typical choices are $c=1$, the Mitscherlich model, and $c=1/3$) and the Gompertz model corresponds to $g(x)=\ln x$.

Here, $y_0=g(x_0)$ and $A=g(a)$, where x_0 is the weight at birth and a is the asymptotic weight (weight at maturity); b is a rate of approach to maturity.

If the animal is growing in a randomly fluctuating environment, we can model growth ([4]) through a stochastic differential equation of the form

(2) $\mathrm{d}Y(t) = b(A-Y(t))\,\mathrm{d}t + \sigma\,\mathrm{d}W(t),$

where σ measures the strength of environmental fluctuations and $W(t)$ is a Wiener process. This is equivalent to consider that the maturity value A in (1) is perturbed by white noise fluctuations.

The transient and stationary behaviours of (2) are well-known (see, for instance, [5]) and the consequences to individual growth and to the distribution of weights (or sizes) in a population of individuals are considered. We also study the properties of the time required for an individual to reach a given size a^* (if a^* is close to a, we may call it the time to maturity, which is relevant in the exploitation of farm animals and in the design of harvesting policies).

Realistically, however, one may expect different individuals to have different maturity values A. We therefore study the distribution of weights in a population when the distribution of A values among its individuals is Gaussian.

We also consider the problem of parameter estimation (by maximum likelihood and, for small samples, the use of bootstrap methods to obtain the distribution of the estimators). The problem of prediction of the size of individuals in future times is also considered. These statistical issues studied here are extremely important for the practical application of the models. We consider the case where we have data at several time instants coming either from a single individual or from several individuals.

The results and methods are illustrated using bovine growth data provided by Carlos Roquete (University of Évora).

References

[1] L. von Bertalanffy, Quantitative laws in Metabolism and Growth. The Quarterly Review of Biology 32, 217-231, 1957.

[2] H. N. Oliveira, R. B. Lôbo e C. S. Pereira, Comparação de Modelos Não-Lineares para Descrever o Crescimento de Fêmeas da raça Guzerá. Pesquisa Agropecuária Brasileira 35, 1843-1851, 2000.

[3] F. J. Richards, A Flexible Growth Function for Empirical Use. Journal of Experimental Botany 10, 290-300, 1959.

[4] O. Garcia, A Stochastic Differential Equation Model for the Height of Forest Stands. Biometrics 39, 1059-1072, 1983.

[5] C. A. Braumann, Introdução às Equações Diferenciais Estocásticas e Aplicações, Edições SPE, 2005.

Extremely low numbers of *Schistosoma mansoni* eggs and the difficulties for standardization of parasitological diagnostic methods

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Keywords: rare events, helminth eggs, parasitological methods, schistosomiasis

Schitosoma mansoni is a worm parasitic of the intestinal veins in man and several other vertebrate animals. The infection may be diagnosed by finding eggs of this helminth in feces. Aproximately 200 million people are infected in the world and the parasitosis is expanding to new transmission focus. In 1997 the southernmost focus in Brazil was identified in the metropolitan area of Porto Alegre, at a locality named Esteio [1]. Epidemiological studies in Esteio and genomic analysis of the parasite has indicated the very recent introduction of the infection in that area. Few individuals were found infected with extremely low numbers of eggs released in feces. The application of classical diagnostic methods has shown the limitations of their sensitivity, what usually is not a problem in high transmission endemic areas.

Similar challenging situations are recognized in areas where control measures have reduced the prevalence and the intensity of transmission, in future evaluation of vaccines and in travel medicine clinics. Tourists that spend a few days in an endemic area may become very lightly infected and classical diagnostic methods fail to detect the parasitosis [2].

Evaluation of very low numbers of eggs in feces poses several problems for statistical analysis and their interpretation.

Seeding experiments are essential, since we need to know as much exactly as possible the initial number of eggs in the fecal sample to be evaluated by any method. Eggs are counted, collected through a fine plastic canula and transferred to an non-infected fecal sample. The sample is then submitted to the diagnostic method for evaluation of its sensitivity.

Difficulties begin with handling of a very few eggs: they may be lost during the steps of transferring from a microscope slide to the fecal sample. We have done an experiment to estimate the loss of eggs in the above situation.

Several numbers of eggs (20, 10, 7, 5, 3, 1) were counted and transferred to a second microscopic slide and the procedure repeated and eggs counted again in a third slide. The results are shown in the Table.

Table – Five sequences of initial numbers of *S.mansoni* eggs (slide 1) and the numbers found after the first (slide 2) and second transference through pipetting

Slide 1	Slide 2	Slide 3
1,1,1,1,1	1,1,1,1,1	1,1,1,0,1
3,3,3,3,3	3,2,3,3,3	3,2,2,3,1
5,5,5,5,5	5,4,3,5,4	5,4,3,5,3
7,7,7,7,7	7,7,7,5,7	6,7,4,5,6
10,10,10,10,10	10,10,10,9,8	9,6,10,9,3
20,20,20,20,20	20,20,19,13,16	20,19,14,9,15

The usual reasoning in the laboratory is that chances to loss eggs are higher with lower numbers being manipulated, approaching the “all or nothing” situation with 1 egg. The results of this experiment did not support that reasoning and we do not have an explanation for this contradiction.

References

[1] Graeff-Teixeira C, Valar C, Moraes CK, Salvany AM, Brum CO et al. (2004) The initial epidemiological studies in the low endemicity schistosomiasis area in Esteio, Rio Grande do Sul, the southernmost Brazilian State, 1997 to 2000. Mem Inst Oswaldo Cruz 99: 73-78.

[2] Doenhoff MJ, Chiodini, PL, Hamilton JV (2004) Specific and sensitive diagnosis of schistosome infection: can it be done with antibodies? Trends Parasitol 20: 35-39.

The optimal spatial distribution of small and large scale fisheries

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Abstract

In this paper the problem of spatially sharing a common fish resource between two quite different harvesting activities is modelled. The problem is studied from the viewpoint a of regulator in charge of deciding how to share the resource. We assume that the regulator has to fix the percent of the area opened to each of the agents and in this way design two patches. One of the patches should be used only by small scale fisheries and plays the role of a reserve that contains the reproduction area. In the second patch a more efficient and intensive activity should be permitted. This problem is motivated by a study case in Chile. The model is introduce stepwise and takes into account the different impact of the activities and a natural biomass transfer into the patches. We end up with an optimal control problem that try to find social relevant solutions. The resulting control problem is treated using a calculus of variation approach and some conditions ensuring the existence of interior solutions are proposed. Finally some academical tests are done in order to see the feasibility of the proposed conditions.

Mixed immunotherapy and chemotherapy of tumors: Feedback design and model updating schemes

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Keywords: Mixed Immunotherapy-Chemotherapy Cancer Therapy - Nonlinear Predictive Control - Model Updating

This paper is based on some recent works [2, 1] where a system of ordinary differential equations (ODE's) has been proposed to model the growth of cell population levels in presence of tumor cells and under a combined immune, vaccine and chemotherapy of cancer. While modelling the entire immune system is a highly complex task, reasonably simple models can be obtained when concentrating on the reaction of the immune system to tumor growth. In particular, the model proposed in [2, 1] enables the basic qualitative phenomenons such as tumor dormancy, oscillation in tumor size as well as bifurcation-like behavior to be reproduced under particular realistic conditions. Moreover, the tumor and the immune system responses to particular medical interventions (chemotherapy drug and immune response modifiers injection) have been modelled. Corresponding simulations show clearly the following facts [2] : first combined immune and chemotherapy may be crucial to the success of cancer treatment, and also as the model parameters may heavily depend on the patient, the same open-loop injection profile can be successful for one patient and inadequate for another even when starting from the same initial state.

It is a well known fact that controlled systems gain in robustness when a feedback control is applied to achieve the desired behavior. More precisely, feedback decreases the sensitivity of the overall result to parametric uncertainties and modelling errors. Moreover, when large deviations in the values of parameters are expected to hold, it may be necessary to use the available measurements in order to improve the treatment robustness.

The aim of this work is to propose a reactive (feedback) mixed treatment strategy based on the model of [2] governing the cancer growth on a cell population level with combination of immune and chemotherapy. More precisely, two possible Nonlinear Model Predictive Control formulations are defined using two different optimal control problems :

1. In the first, the problem is to minimize the tumor size at the end of the prediction horizon while keeping the number of circulating lymphocytes beyond a minimal level during the whole therapy.

2. In the second, the problem is to maximize the number of circulating lymphocytes while imposing a contraction rate on the number of tumor cells at the end of the prediction horizon.

The feedback design is based on the above nonlinear constrained model predictive controls together with an adaptation scheme that enables to compensate the effects of unavoidable modelling uncertainties. The effectiveness of the proposed strategies are shown under realistic human data showing the advantage of treatment in feedback form as well as the relevance of the adaptation strategy in handling uncertainties and modelling errors. The strong interest of the treatment strategy defined by the original optimal control problem formulation (2.) will be shown through dedicated simulations as it may lead to tumor regression under better health indicator profile.

References

[1] de Pillis, L. G. and Radunskaya, A. E., Immune response to tumor invasion. Bathe, K. (Ed.), Computational Fluid ans Solid Mechanics, vol. 2, MIT Press, Cambridge, MA., 2003.

[2] L. G. dePillis and W. Gu and A. E. Radunskaya, Mixed immunotherapy and chemotherapy of tumor: modeling, application and biological interpretations, Journal of Theoretical Biology, 2005, v. 238, 841-862.

A dynamical fishing model on many zones: Simulation on the sardine stock in Atlantic Moroccan cost

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Keywords: dynamic model, optimal fishing activity, Moroccan sardine stock, durable fishing activity.

Abstract: The main propose of the fisheries management is to permit a durable and optimal fishing activity. In this context, this work is presenting a dynamic model governing the evolution of a stock of fish distributed on many zones and targeted by a composite fleet. We set the hypothesis that there is no fish migration between the different zones while a part of the fleet can move between those fishing regions. The model is a set of ODE equations with two components: A stock component witch describes the evolution of the exploited population on its zones of distribution, and a fleet component governing the dynamic of the fishing effort. We start by solving analytically the system using the aggregation techniques and propose some necessary and optimal conditions on the effort to ensure a durable fishing activity and optimize the harvest. A simulation on the sardine stock in Atlantic Moroccan cost is then proposed: The dynamic parameters of harvesting are estimated by using a models fitting techniques based on a sets of available data. The optimal conditions to maximize the activity of harvest are then proposed.

References

[1] Auger, P.M. & Bravo de la Parra, R., Methods of Aggregation of Variables in Population Dynamics, Comptes Rendus de l'Académie des Sciences, série III, Paris. 323, 665-674, 2000.

[2] Auger, P.M. & Poggiale, J.C., Emergence of Population Growth Models: Fast Migration and Slow Growth, Journal of Theoretical Biology, 182: 99-108, 1996.

[3] Auger, P.M. & Poggiale, J.C., Aggregation and Emergence in Hierarchically Organised Systems: Population Dynamics, Acta Biotheoretica 44: 301-316, 1996.

[4] Clark, C.W., The Optimal Management of Renewable Resources, Mathematical Bioeconomics, 2nd ed., A Wiley-Interscience, 1990.

[5] R. Mchich, P. Auger & N. Raïssi, The stabilizability of a controlled system describing the dynamics of a fishery, Comptes Rendus Biologies, Volume 328, Issue 4, pp. 337-350, 2005.

[6] R. Mchich, N. Charouki, P. Auger, N. Raïssi & 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.

Mitochondrial energy metabolism modelling

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Keywords: modelling, EDO, mitochondrial metabolism.

Mitochondria are intra-cellular organelles that are the power plant of the cell. A series of chemical reactions catalysed by enzymes occur in the matrix of mitochondria that allow, among other things, the the production of ATP, a form of useable energy by the cell. This energy delivered by mitochondria also involves a series of redox reactions called the Respiratory Chain (RC). These reactions are catalysed by highly structured protein complexes and electron transporters embedded in the inner mitochondrial membrane. Electron transfer is linked to an extrusion of protons from the mitochondria, creating an electro-chemical gradient that is used for ATP production, according to the chemiosmotic theory.

Many models have been derived to simulate mitochondrial energy metabolism in specific tissues (see [1] to [7]). Our aim is to develop a mathematical model of the mitochondrial energy metabolism able to explain the different behaviors (rate of respiration, control coefficients, expression thresholds, redox status of electron transporters) observed in the different tissues. This general model which will be modular and adaptable to different types of mitochondria by incorporating the tissue specific parameters already determined or to be determined, involves: - a deterministic model of the tricarboxylic acid cycle (TCA) using ordinary differential equations. - as well as TCA module itself, the model will include other modules that can be interconnected : a model of RC, of the beta-oxidation of fatty acids, of the metabolite transport and exchange, particularly the different redox shuttles. The modelling of these modules will be conducted in the same way as the TCA module, i.e. developing a general model using empirical equations with specific (kinetic) parameters determined in Bordeaux or coming from the literature and also taking into account the thermodynamical properties of the redox reactions. Under some assumptions, we will give mathematical properties of the model (existence of a unique non negative solution, of a stationnary state, ...). Then, we will do quantitative simulations.

References

[1] Beard, D.A., A biophysical model of the mitochondrial respiratory system and oxidative phosphorylation. PloS Computational Biology 1, 252-264, 2005.

[2] Bohnensack, R., The role of the adenine nucleotide translocator in oxidative phosphorylation. A theorical investigation on the basis of a comprehensive rate law of the translocator. J. Bioenerg. Biomem., 14, 45-61, 1998.

[3] Cortassa, S., Aon, M.A., Marban, E., Winslow, R.L., O'Rourke, B., An integrated model of cardiac mitochondrial energy metabolism and calcium dynamics. Biophys J., 84, 2734-55, 2003.

[4] Dynnik, V.V., Heinrich, R. and Sel'kov, E.E., A mathematical model of carbohydrate energy metabolism interaction between glycolysis, the Krebs cycle and the H-transporting shuttles at varying ATPase load (in Russian), Byokimika, 45, 771-782, 1978.

[5] Garfinkel D., Simulation of the Krebs Cycle and Closely Related Metabolism in Perused Rat liver. Computers and Biomedical Research, 1-17 and 18-42, 1971.

[6] Korzeniewski, B., Froncisz, W., An extended dynamic model of oxidative phosphorylation, Biochim. Biophys. Acta 1060: 210-223, 1991.

[7] Yugi K., Tomita M., A general computational model of mitochondrial metabolism in a whole organelle scale. Bioinformatics 22;20(11)1795-6, 2004.

Fixation in haploid populations exhibiting density dependence

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Keywords: Fixation, density dependence, haploid, population genetics, population dynamics.

We extend the one-locus two allele Moran model [2] of fixation in a haploid population to the case where total the size of the population is not fixed. The model is defined as a two dimensional *birth-and-death* process for allele number. Changes in allele number occur through density independent death events and birth events whose per capita rate decreases linearly with the total population density. Uniquely for models of this type [1, 3], the latter is determined by these same birth and death events. The two types, type defined by the allele possesed by an individual, can differ in their birth and death rates. This model provides a framework for investigating both the effects of fluctuation in total population number through demographic stochasticity, and deterministic density dependent changes in mean density, on allele fixation. We analyse this model using a combination of asymptotic analytic approximations supported by numerics.

The analysis of this model splits neatly into two cases, the non-neutral case where the two alleles have different expected equilibrium population sizes [4] and the quasi-neutral case where their equilibrium population sizes are the same [5]. In the non-neutral case we find that for advantageous mutants demographic stochasticity of the resident population does not affect the fixation probability, but that deterministic changes in total density do. In contrast for deleterious mutants the fixation probability increases with increasing resident population fluctuation size, but is relatively insensitive to initial density. These phenomena can not be described by simply using a harmonic mean effective population size.

The restriction in the quasi-neutral case, that both allelic types have the same equilibrium population densities, results in birth rates that are proportional to death rates. This can be viewed as a life history trade-off. The deterministic dynamics possesses a stable manifold of mixtures of the two types. We show that the fixation probability is asymptotically equal to the fixation probability at the point where the deterministic flow intersects this manifold. The deterministic dynamics predicts an increase in the proportion of the type with higher birth rate in growing populations (and a decrease in shrinking populations). Growing (shrinking) populations therefore intersect the manifold at a higher (lower) than initial proportion of this type. On the center manifold, the fixation probability is a quadratic function of initial proportion, with a disadvantage to the type with higher birth rate. This disadvantage arises from the larger fluctuations in population density for this type.

The results we present for the two cases are asymptotically exact. They have direct relevance for allele fixation, models of species abundance, and epidemiological models. More generally, these results, extend our understanding of how changes at the level of individuals interact with the dynamics of the population as a whole.

References

[1] M. Kimura and T. Ohta, Probability of gene fixation in an expanding finite population. Proc. Natl. Acad. Sci., 71, 3377-3379, 1974.

[2] P. A. P. Moran, Random processes in genetics. Proc. Camb. Phil. Soc., 54, 60-71, 1958.

[3] S. P. Otto and M. C. Whitlock, The probability of fixation in populations of changing size. Genetics, 146, 723-733, 1997.

[4] T. L. Parsons and C. Quince, Fixation in haploid populations exhibiting density dependence I: The non-neutral case. Theor. Pop. Biol., *to appear*, 2007.

[5] T. L. Parsons and C. Quince, Fixation in haploid populations exhibiting density dependence II: The quasi-neutral case. Theor. Pop. Biol., *to appear*, 2007.

Yellow fever vaccination in the absence of infection risk: How to decide?

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Keywords: epidemic model, decision theory, yellow fever, Bayesian melding

Yellow fever (YF), an arthropod-borne viral disease, is endemic in regions of tropical America and Africa. Since 1942, after strong vaccination efort and vector eradication, the urban cycle of this disease was eradicated from Brazil. The sylvatic cycle, however, remained endemic in the north and west regions, with low human activity due to the high vaccine coverage. In the last three decades, however, with the reinvasion of many cities by the urban vector - *Aedes aegypti* - reurbanization became a threat, leading to a debate on the pros and cons of preemptive vaccination of populations in areas currently free of disease. One problem is that, in the absence of immediate risk, vaccination programs face ethical issues since even small risks associated with the vaccine are greater than the immediate disease risk (which is zero). On the other hand, yellow fever is a life-threatening disease and many deaths would be expected during an outbreak in a unvaccinated population. How to decide? Here, we developed a mathematical model that formalizes the vaccination decision process from the perspective of a rational susceptible individual deciding between vaccinating now or waiting for an emergency situation. The cost of choosing strategy W (wait) depends on many uncertain variables: the probability of an outbreak, the probability of getting infected if an outbreak occurs, the probability of getting vaccinated before the infection event, and the probabilities of serious disease given infection and serious adverse events from the vaccine. The cost of choosing V (to vaccinate now) depends only on the (immediate) risk of adverse event by the vaccine. We are interested in dening epidemiological situations where vaccinating immediately is advantageous from the individual perspective, that is, $cV < cW$. That clearly depends on the risk of experiencing an outbreak, and upon occurrence, the probability of receiving the vaccine or escaping disease. Any prediction regarding a future outbreak of urban yellow fever is inevitably uncertain. To reduce uncertainty, we seeked to consider all information available: the transmission cycle of yellow fever (represented by a disease transmission model), a set of reasonable ranges for the parameters in the model (from the entomological literature), and a reasonable range for the reproductive ratio. To reconstruct the joint distribution of these parameters, we used a restricted version of the Bayesian melding method (Poole and Raftery, 2000). Our model suggests that, if disease is considered possible but unlikely, delaying vaccination is a good strategy if a reasonably eficient campaign is expected. The advantage of waiting increases as the rate of transmission is reduced, suggesting that vector control programs and emergency vaccination preparedness work together to favor this strategy. The opposing strategy, vaccinating preemptively, is favored if the probability of yellow fever urbazination is high or if expected R_0 is high. Our results hightlight the importance of surveillance and preparedness. In addition, specically for dengue endemic regions, this result brings to attention the interplay of diferent diseases that can be jointly prevented by a single control measure.

References

[1] D. Poole and A. E. Raftery, Inference for deterministic simulation models: the Bayesian melding approach. JASA 95, 1244, 2000.

Impact of initial antiretroviral regimens in viral load and CD4+ cell counts in patients living with HIV

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Keywords: HIV; AIDS; antiretroviral therapy; linear model; laboratory outcome; patient follow-up.

More than two decades after its identification the global AIDS epidemic has been significantly affected by prophylatic and therapeutic interventions [1]. Particularly in Brazil, where care policies include universal access to antiretroviral therapy (ART), provided free of charge by the Public Health Sector, a striking decline in AIDS-related morbidity and mortality was noticed.

In the natural history of the HIV infection, a ten-year long asymptomatic period precedes disease development. However, viral-host interaction is a dynamic rather than a static event. Mathematical models have been crucial to rule out the concept of viral latency, since persistent and intense viral replication was demonstrated throughout the asymptomatic phase. One important contribution revealed that most plasma viral producing cells had become infected few days before [2],[3].

Mathematical modelling is also a useful tool in the study of viral kynetics and host immune responses in individuals under chemotherapy as demonstrated in hepatitis B and C infections [4]. The present study aims at evaluating such an interaction in HIV infection, investigating the impact of initial ART regimens (mono vs. bitherapy vs. HAART) in the course of disease.

Using an available database with at least six sequential CD4+ cell counts and HIV viral load assessments of 1391 patients under clinical follow-up at a reference care center in São Paulo, we classified patients acoording to a linear approximation model of viral load and CD4+ cell counts into favorable and unfavorable outcomes.

The results point out for a benefitial effect of ART rescue regimens for 2% patients with persistent viremia. No particular initial regimen was associated with undetectability during follow-up. Unfavorable outcomes were associated with most patients who resumed viremia transiently (69%) or at the end (100%) of follow-up.

References

[1] 2006 Report on the global AIDS epidemic, UNAIDS, Geneva, 2006.

[2] P.P. Ho , A.U. Neumann, A.S. Perelson, W.Chen, J.M. Leonard, M. Markowitz, Rapid turnover of plasma virions and CD4 lymphocytes in HIV-1 infection. Nature 373, 123-126, 1995.

[3] L.M. Wein , S.A. Zenios, M.A.Nowak, Dynamic multidrug therapies for HIV: a control theoretic approach. J. Theor. Biol. 185, 1529,1997.

[4] E.S.A. de Araújo, A.U. Neumann, C. Courtouké et al., Early HCV kinetics predict sustained virological response to treatment with peginterferon-A2A and ribavirin in brazilian HIV/HCV co-infected patients. 12th International Symposium on Hepatitis C Virus and Related Viruses, Montral, Abstract Book, 94-94, 2005.

A cellular automata model to describe the dynamical aspects of parasitaemia in the blood cycle of Malaria

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Keywords: cellular automata, malaria, blood cycle

Malaria is an important cause of morbidity and mortality worldwide. One striking aspect regarding malaria is the fact that individuals living in endemic areas do not develop immunity against the parasite, falling ill whenever they are exposed to the parasite. The understanding of why immunity is not developed in the usual way against Plasmodium is crucial to the improvement of treatment and prevention. In this work, we study some aspects of the dynamics of the blood cycle of malaria using both modelling and data analysis of observed case-histories described by parasitemia time series. By comparing our simulations with experimental results we have shown that the different behaviour observed among patients may be associated to differences in the efficiency of the immune system to control the infection.

References

[1] L. Molineaux and K. Dietz, Parassitologia **41**, 221-231 (1999).

[2] L. Molineaux,H. H. Diebner, M. Eichner, W. E. Collins, G. M. Jeffery and K. Dietz, Parasitology **122**, 379-391 (2001).

[3] G. A. Funk, V. A. A. Jansen, S. Bonhoeffer, and T. Killingback, J. Theo. Bio. **233**, 221-236 (2005).

[4] R. M. Zorzenon dos Santos and S. G. Coutinho, Phys. Rev. Lett. **87**, 161102 (2001).

[5] M. Blatt, S. Wiseman and E. Domany, Phys. Rev. Lett. **76**, 3251-3254 (1996).

ON THE MODELLING OF THE WEST NILE VIRUS

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Keywords: West Nile Virus, dynamics, nonlinear, stability, mosquitoes

ABSTRACT: We study the dynamics of the West Nile Virus infection between mosquito and bird population. We show an analytic analysis and several numerical examples.

References

[1] G. Cruz-Pacheco, L. Esteva, J. A. Montaño-Hirose, Cristóbal Vargas. A Mathematical Model for the dynamics of the West Nile Virus. System, Structure and Control 2004, ELSEVIER,2004,PP .475-480.

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Scylla and Charybdis in the Virus World: revisiting Eigen's prebiotic information crisis.

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Keywords: Metapopulation dynamics, package models, viral evolution, simulation.

The existence of essential genes to virus replication and structure, shared by a broad variety of viruses but missing from cellular genomes (virus hallmark genes) suggested the Koonin's Virus World hypothesis. [2]. Those authors state that cellular life was predated by complex community of viruses and selfish genetic elements continuosly exchanging and competing for genetic information. Despite the ingenuity of this hypothesis and the solid background underlying its assumptions little light is cast on a more fundamental issue, namely the Eigen's prebiotic information crisis [6, 4]. So, the problem remains the same and a widely accepted solution for this crisis awaits to be found. By the way, in the present work we critically assess the ancient Virus World using a more quantitave approach based in the models presented in [1, 3, 8, 7]. Our objective is to demonstrate that ancient viruses populations and the prebiotic vesicles behave in a very similar way and, therefore, they should show the same threshold phenomena. In order to fully reproduce the whole set of parameters proposed in [2], we rebuild the prebiotic models of [1, 7] to allow a simple form recombination and migration and construct a viability diagram for parameter space. Our results point toward an early assembly of genes into more organized units (protocromosomes) as a fundamental step to attain higher levels of genetic diversity, in fully accordance with the results found in [5]. We conclude by briefly discussing the implications of thresholds found for the preservation of genetic information under the Virus World scenario and for present day viruses like orthomyxoviruses and bunyaviruses.

References

[1] D. A. M. M. Silvestre & J. F. Fontanari. Preservation of information in a prebiotic package model. arXiv:q-bio/0612047v2 [q-bio.PE], 2007.

[2] E. V. Koonin, T. G. Senkevich & V. V. Dolja. The ancient Virus World and evolution of cells. Biology Direct, 1(1):1-29, 2006. (doi:10.1186/1745-6150-1-29)

[3] J. F. Fontanari, M. Santos and E. Szathmáry. Coexistence and error propagation in pre-biotic vesicle models: A group selection approach. Journal of Theoretical Biology, 239(2):247-256, 2006

[4] E. Zintzaras, M. Santos & E. Szathmáry. Living Under the Challenge of Information Decay: The Stochastic Corrector Model vs. Hypercycles. Journal of Theoretical Biology, 217(2):167-181, 2002.

[5] M. Santos. Origin of Chromosomes in Response to Mutation Pressure. The American Naturalist, 152(5):751-756, 1998.

[6] M. Eigen & P. Schüster. Stages of emerging lifefive principles of early organization. Journal of Molecular Evolution 19:4761, 1982.

[7] U. Niesert, C. Bresch, & D. Harnasch. Origin of life between Scylla and Charybdis. Journal of Molecular Evolution 17:348353, 1981.

[8] C. Bresch, U. Niesert & D. Harnasch. Journal of Theoretical Biology, 85(3):399-405, 1980

The role of sexually isolated groups in two-sex demographic and epidemic logistic models with non-linear mortality

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Keywords: logistic growth, population dynamics, two-sex models, sexually transmitted diseases, isolation, dynamical systems.

We introduce and analyze several gender structured population models governed by logistic growth with non-linear death rate. We extend these models to include groups of people isolated from sexual activity and individuals exposed to a mild and long-lasting sexually transmitted disease in the sense that there is no disease-induced mortality nor recovery from the disease (Herpes simplex is one example). The transmission of the disease is modeled through formation/separation of heterosexual couples assuming that one infected individual automatically infects his/her partner. We establish a demographic threshold on the parameters that separates the existence of a positive steady state from the extinction equilibrium, and we found this condition to be similar to the one derived by Castillo-Chávez and Huang for the two-sex logistic model with non-linear fertility [1]. We also analyze how the non-reproductive groups may change the demographic tendencies in the general population and whether they can decrease or eliminate the growth of the infected group while keeping the healthy population at acceptable levels. We do this by computing the epidemic reproductive number using a method based on the next-generation matrix developed in [2], and we use it to establish conditions for the stability of the disease-free equilibrium. We also found that, under some conditions, the presence of isolation from reproduction can cause the total population to settle at higher levels than in the absence of it, if the separation rate of the infected pairs is large. A comparison with our similar work on homogeneous two-sex models [3] and epidemic one-sex S-I-type models [4] is also provided.

References

[1] Carlos Castillo-Chavez and Wenzhang Huang. *The Logistic Equation Revisited: The Two-Sex Case*, Math. Biosc. 1995 Jul-Aug;128(1-2):299-316.

[2] P. van den Driessche and James Watmough. *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Math. Biosc. 180(2002) pp. 29-48.

[3] F. A. Milner. *How may segregation from sexual activity affect population growth?*, Math. Biosc. and Engin. 2 (2005), pp. 579-588.

[4] D. Maxin and F. A. Milner *Effects of Non-Reproductive Groups on persistent sexually transmitted diseases*, Math. Biosc. and Engin. 4 (2007), pp. 505-522.

Bifurcations in an SEIQR Model for Childhood Diseases

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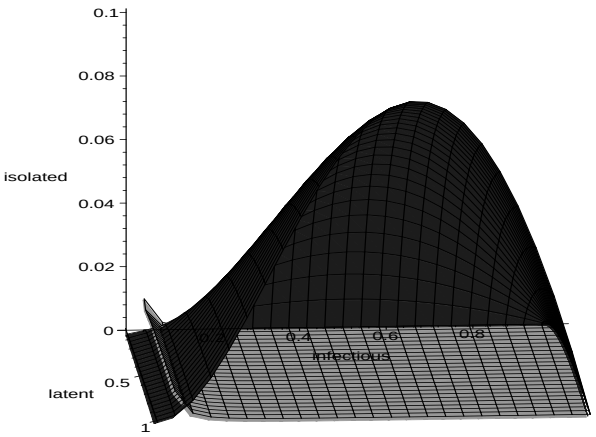
Explaining the recurrent outbreaks of measles and other childhood diseases has long been a topic of discussion in the modeling community. Initial explanations included that deterministic epidemiologic factors gave rise to dampened oscillations which converged to an endemic equilibrium. It was then argued that either seasonal or stochastic forcing could excite these oscillations into becoming undamped. In [1], the authors show that autonomous deterministic factors alone can cause sustained oscillations if a class of isolated individuals is included.

While the model proposed in [1] succeeds in the primary goal of demonstrating sustained oscillations, it does not lend itself well to comparison with real data because it does not include a class of latently infected individuals. In this work, we extend the model of [1] to include a latent class. We are able to prove the existence of supercritical Hopf bifurcations and hence sustained oscillations. Through this extension, we are able to gain more insight into the behavior of solutions in three-dimensional parameter space. We are also able to prove the existence of homoclinic bifurcation in a biologically feasible unfolding of a simplified version of our model. Along with these analytic results, we can observe a better comparison with real data from childhood diseases.



References

[1] Z. Feng and H. Theime, Recurrent outbreaks of childhood diseases revisited: The impact of isolation. Math. Biosci. 128, 93-130, 1995.

[2] L. Wu and Z. Feng, Homoclinic bifurcation in an SIQR model for childhood diseases, J. Differential Equations. 168, 150-167, 2000.

Model of Two Dynamic Competing Species

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Keywords: stochastic noise, population dynamics, Lotka-Volterra model.

Abstract

We study the interplay between noise and periodic modulation in a classical Lotka-Volterra model of two competing species. The consideration of noise changes the behaviour of the systems and leads to new situations which is no longer a deterministic case. Among others, noise is responsible by periodic solutions of the species and stochastic resonance phenomenon. Recently, the influence of noise on population dinamycs has been the subject of intense theoretical investigation involving stochastic methods. The principal aim of this work is to study the generalized Lotka-Volterra model with a random interaction parameter between two competing species. In the presence of a noise terms, the equations are given by,

$$u' = u(a_1 - b_1 u - c_1 v) + f_u(u,v)\,\xi_u(t)$$

$$v' = v(a_2 - b_2 u - c_2 v) + f_v(u,v)\,\xi_v(t)$$

where u and v are the populations densities and a_i, b_i, c_i, i = 1,2 are the physical parameters which are taken positive constants. The terms $f_i(u,v)\,\xi_i(t)$, i = u, v model the contribution of the random forces. We want to show that noise cannot systematically be neglected in models of population dynamics. Its presence is responsible for the geration of temporal oscilations .

References

[1] A.D Bazykin, Nonlinear dynamics of interacting populations, World scientific series on nonlinear science. series A, vol 11, world scientific singapore, 1988.
[2] C. V. Pao, Coexistence and Stability of a Competition-Diffusion System in Population Dynamics, Journal of Mathematical Analysis and applications 83 (1981), 54-76.
[3]B. Oksendal, Stochastic diferential equations: an introduction with aplications, springer-verlag, sexta edição, new york 2005.
[4] P.E. Kloeden and E. Platen, numerical Solution of Stochastic Differential Equations, Springer-Verlag, Berlin, 1995.

The Role of Spatial Refuges in Coupled Map Lattice Model for Host-Parasitoid Systems

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Keywords: Host-Parasitoid systems, spatial refuges, coupled map lattices.

A very important question in Ecology is the coexistence of species. In homogeneous habitats, the nontrivial equilibrium of discrete Nicholson-Bailey model is never stable. However, when individuals are allowed to disperse to neighboring patches, the host and parasitoid populations can persist together (Hassell et al., 1991).

Spatial refuges are sites where a fraction of hosts may hide and be protected against the parasitoids. They can be classified as complete or partial refuges. The former are characterized by the absence of the parasitoid or by a null parasitoid efficiency (parasitoids can access the interior of the refuge but they do not attack there); the latter is for the scarcity of the parasitoid or for a very low parasitoid efficiency (Krivan, 1998; Murdoch, 1995). Both operate as preservation sites for vulnerable species, that otherwise could go to extinction. These refuges indirectly benefit the parasitoid since a food source is guaranteed by a host spill over which will be dispersed outward their protection area.

The primary purpose of this work is to address the stabilizing effects of refuges on host-parasitoid dynamics. This debate has promoted many results (Hassell, 2000; Holt and Hassell, 1993; Krivan, 1998) and we try to provide some insight concerning questions like the effect of refuge size and shape, localization and number of them. In order to explicitly consider refuges in space we use a Coupled Map Lattice (CML) formulation for Nicholson-Bailey host-parasitoid dynamics with patch dependent parasitoid efficiency.

Our CML model is formulated with an explicit spatial distribution of partial refuge areas. Parasitoids can occupy any place on its habitat but, in well localized refuges, their efficiency is lower than in the non-refuge areas. In other words, partial refuges are included by choosing some patches where it is assumed the parasitoid efficiency is much lower than outside, such that the number of hosts which escape from parasitism is enhanced inside the refuges.

From the simulations we have performed, we conclude that the spatial refuges may alter the host-parasitoid spatial and temporal dynamics in both local (within refuge) and global (whole habitat) scales. As many authors have already concluded, our results show that refuges may stabilize as well as destabilize (Hassell, 2000; Holt and Hassell, 1993; Krivan, 1998) host-parasitoid dynamics. The stabilization depends on many factors such as refuge size and shape, number and nearness, movement rates of individuals and parasitoid attack rate in a very complex way.

References

[1] M. P. Hassell, The Spatial and Temporal Dynamics of Host-Parasitoid Interactions, Oxford University Press, New York, 2000.

[2] M. P. Hassell, H. N. Comins and R. M. May, Spatial structure and chaos in insect population dynamics. Nature, 353, 255-258, 1991.

[3] R. D. Holt and M. P. Hassell, Environmental heterogeneity and the stability of host-parasitoid interactions. Journal of Animal Ecology, 62, 89-100, 1993.

[4] V. Krivan, Effects of optimal antipredator behavior of prey on predator-prey dynamics: The role of refuges. Theoretical Population Biology, 53, 131-142, 1998.

[5] W. W. Murdoch, R. F. Luck, S. L. Swarbrick, S. Walde, D. S. Yu and J. D. Reeve, Regulation of an insect population under biological control. Ecology, 76(1), 206-217, 1995.

A Theoretical Framework to Understand the Emergence of Drug Resistance: A Case Study of Tuberculosis

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Keywords: drug resistance, population dynamics, population genetics, tuberculosis.

Tuberculosis accounts for more deaths among adults than all other infectious diseases combined. The introduction of anti-tuberculosis drugs in the 1950s and the development of the various drug regimens meant that by the 1980s there was a 98% chance of cure. Unfortunately the very success of the drug treatment of tuberculosis has been the catalyst for the emergence of a new wave of drug resistance. If drug resistant tuberculosis is introduced then the "epidemic" will be drug resistant. Mathematical models have been developed to describe the transmission dynamics and the impact of interventions for many infectious diseases including drug-sensitive and drug-resistant tuberculosis [1]. Previous work on population dynamics of drug resistance has shown that the fitness of drug-resistant strains is a key determinat of the future burden of drug-resistant pathogens in general [2].

In this work, a simple alternative theoretical framework has been used to study the spread and evolution of M. tuberculosis resistance due to antimicrobial agents within human communities. Both, the dynamics of bacterial growth within individuals and the interaction among individuals are taken into account. Under the umbrella of theoretical epidemiology, a simple dynamical system is obtained by considering a population genetic approach to examine the role of the relative fitness and mutation of the antibiotic-sensitive or antibiotic-resistant strains to one or more antibiotics. In this way, the spatiotemporal dynamics and persistence of M. tuberculosis are determined at two scales: the within-host scale (a local population scale; parasites within hosts) and the epidemic scale (spatial and/or social aggregation of hosts).

The within-host model treat the changes in the incidence of sensitive and resistante bacteria in an individual host during and following antibiotic therapy. In fact, we consider the case of a single gene as the determinant of resistant with the bacterial genotypes S (sensitive) and R (resistant) to a particular antibiotic. In the ausense of antibiotic use, sensitive genotype is at a fitness advantage. The computational implementation of such a dynamics was done by using genetic algorithms in which we include a fitness cost when resistance has been acquired. Therefore, the bacterial drug resistance is modeled inside of each individual as an inner world, where every single individual acts a bacterial repository and their evolution occurs in the presence of drugs administration on such individual. Hence, the bacterial rate in each person acts as a classifier to his current external clinical state which describes the transmission rules of the inter-individual interaction dynamics.

The epidemic model treat the evolution and spread of resistance in communities of hosts. We developed a computational probabilistic cellular automata model in order to describe the transmition dynamics and emergence of resistance among the bacteria resident in a population of human hosts. Furthermore, in this model the heterogeneity of interactions between human individuals is featured by a composition of two types of social contacts described in terms of local (through a neighborhood) and global influences (as in a mean-field approach) [3]. In particular, such contacts provide very interesting scenario wherein the dynamics of tuberculosis can be studied in terms of the speed and impact of transmission.

References

[1] Sally M. Blower, Julie L. Gerberding, Understanding, predicting and controlling the emergence of drug-resistant tuberculosis: a theoretical framework. J. Mol. Med. 76, 624-636, 1998.

[2] D. J. Austin, K. G. Kristinsson and R. M. Arderson, The relationship between the volume of antimicrobial consumption in human communities and the frequency of resistance. PNAS 96, 1152-1156, 1999.

[3] D. Alves, V. Hass, A. Caliri, The Predictive Power of R0 in an Epidemic Probabilistic Model, Journal of Biological Physics, Vol. 29, 2003.

Endemic steady states and travelling wave solutions for a class of age-structured epidemic models

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Keywords: Integro-differential equation, endemic steady state, travelling waves, epidemic models.

This work is devoted to the study of endemic equilibria and travelling wave solutions for an age-structured epidemic model for a multi-group population. The model we investigate takes into account inter-group infection as well as age-specific inter-cohort interactions. We use a modified argument ever used by Kubo and Langlais in order to transform the system without comparison principle into a suitable system of equations with monotonic properties. We derive some sufficient conditions on the epidemic parameters that ensures the existence of travelling front solutions connecting the disease free equilibrium and an endemic steady state. A more precise study is given for a scalar epidemic equation.

A Mathematical Model of Pirarucus (*Arapaima gigas*) Population Dynamics

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Keywords: Pirarucu, *arapaima gigas*, population dynamics, mathematical model.

The pirarucu (*Arapaima gigas*) is the most important natural fishery resource of the Amazon region [1]. With the introduction of more modern technologies of exploitation the specie became threatened. It is necessary therefore to improve management strategies to prevent extinction.

The present paper proposes a model that intends to simulate the pirarucu population dynamics. The model divides the life span of pirarucu into three different stages: eggs, larvae and adult. Despite the very different time interval corresponding to these phases they present certain particular characteristics that we would like to stress. As a matter of fact the phase designed with egg means fertilized eggs and therefore requires the participation of male and female. The phase designed with larvae is characterized by parental care [1]. Males of this specie keep watching the larvae till they can survive with adult protection.

Preliminary results indicate that there the dynamical system describing the population evolution presents two attractors, one corresponds to extinction and the other to a stable number of males an females. These two attractors are located on opposite sides of a separatrix that contains an unstable saddle point.

It is clear from the model that there is a critical number of fishes separating extinction from stable preservation. That is if a number of fishes is introduced in a lake above the critical limit the tendency is the population to grow up to a certain point. On the opposite case the population tends to extinction. Certainly the validation of the model still needs several field data. But preliminary observations are encouraging and do not present any severe incongruence with the observed distribution of these three stages.

References

[1] Queiroz, H.L. Natural history and conservation of pirarucu, Arapaima gigas, at the Amazonian Várzea: Red giants in muddy waters. Thesis submitted for the degree of Doctor of Philosiphy, University of St. Andrews, 2000.

The ecotoxicology of the zooplankton-cyanobacteria interaction

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Keywords: DEBtox; estimation; bayesian inference; Leslie matrix population models;

Life-table experiments experiments were performed to study the effects of the toxic cyanobacterium *Microcystis aeruginosa* on *Moina micrura*, a cladoceran of the tropical Jacarepagu Lagoon (Rio de Janeiro, Brazil) [1]. Different experimental designs were used to estimate toxic effects of both eld samples and laboratory cultures of Microcystis aeruginosa on life history parameters.

Based on the Dynamic Energy Budget theory, DEBtox models were proposed (Kooijman and Bedaux, 1996) [2] to analyze standard toxicity tests. Especially, reproduction effect models were built for *Daphnia magna*, a common zooplankton organism. We extended these effect models to the experimental data obtained with *Moina micrura*. DEBtox models assume five possible modes of action for toxic compounds, among which, according to data and fit quality, it's possible to choose the most appropriate. Each mode of action is described by mechanistic models of reproduction and growth, which have parameters with a clear biological meaning: the maximum body size, the growth rate, or the No Effect Concentration (NEC), a key concept in the DEBtox theory. A DEBtox software is available on-line leading to parameter estimates based on the least square criterion.

Expert knowledge on the possible range for these parameters is often available in ecotoxicology laboratories or in the literature. For the sake of more transparency and precision of parameter estimates, we took this expertness into account and developed an innovative Bayesian approach, which combines a priori knowledge about parameter distributions and data without constraint [3]. Contrary to the DEBtox software, the Bayesian approach does not need to fix some of the parameters. The substantial gain we introduced with the Bayesian approach is the possible simultaneous fit of both reproduction and growth models on data. We show how parameter estimates and fit quality are really improved, in the case study of *Moina micrura*.

By combining DEBtox theory and matrix population models [4], every toxic effects were extrapolated from the individual (reduced fecundity, growth and survival) to the population level. All lethal and sublethal effects were integrated into the population growth rate [5], a classical endpoint to quantify population health.

References

[1] A.S. Ferro-Filho and S.M.O. Azevedo, Effects of unicellular and colonial forms of Microcystis aeruginosa from laboratory cultures and natural populations on two tropical cladocerans. Aquatic Ecology, 37: 23-35, 2003.

[2] S.A.L.M. Kooijman and J.J.M. Bedaux, The analysis of aquatic toxicity data, VU University Press, Amsterdam, 1996.

[3] W.R. Gilks, S. Richardson and D.J. Spiegelhalter, Markov Chain Monte Carlo Methods in Practice, Chapman & Hall, 1996

[4] H. Caswell, Matrix Population Models - Construction, analysis, and interpretation, 2nd edition, Sinauer Associates, Sunderlands, Massachussets, 2001.

[5] E. Billoir, A.R.R. Pry and S. Charles, Integrating the lethal and sublethal effects of toxic compounds into the population dynamics of *Daphnia magna*: a combination of the DEBtox and matrix population models, Ecological Modelling, in press.

Three Mathematical Models for Endemic Cutaneous Leishmaniasis Considering Human, Animal Host and Vector Populations

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In malaria and dengue, is widely documented and accepted the contribution of humans as reservoirs of their respective infectious agents. Conversely, in cutaneous leishmaniasis humans are regarded, with few exceptions, as dead-hosts for Leishmania parasites. However, there are areas, like the Colombian Pacific Coast, where a high prevalence of the disease exists, close to 10% in humans, in the apparent absence of natural reservoirs of the parasite [1]. Actually, the evaluation of a high number of mammals in the search of potential reservoirs, have rendered only an edentate naturally infected. The present study departs from this apparent paradox and hypothesizes that the humans can act as sink or source of the parasites. The main objective was to clarify whether a variation to the orthodox cycle of transmission is taking place in these areas. Three mathematical models were formulated with the aim to determine the participation of the human as reservoir of Leishmania Viannia in the Colombian Pacific Coast, an endemic region for cutaneous leishmaniasis. The complexity of the problem takes place by the multiplicity of factors involved in the transmission of the disease. These models show that the human as reservoir of the parasite exactly has the same implications of the reservoirs animals, but with a great difference that human can be transporting the parasite to much greater distances. In situations when several hosts can act as reservoir of infection, the process can be explored within a cycle of transmission. This is the case for Leishmaniasis in which converge different vectors and, at least, three hosts (humans, dogs, rodents, etc.) in absence of animals reservoir, the humans take the place from reservoirs [2]. The goal of this study was to explore the role of humans as an essential reservoir in the transmission chain of the Leishmaniasis in urban settings with a limited access to wild host populations. Our presentation is organized as follows: firstly, a model which assumes that parasites (ingested by vector from both, human and animal reservoirs) suffer a successful development to infective forms. This model defined a biting rate for the humans and another for animal reservoir. The three populations (human, animals and vectors) are subdivided into classes: susceptible and infected. The transition from one class to the other occurs basically by the biting rate. This model shown that the participation of humans in the transmission cycle is equally important as that recorded for animal reservoir, when the populations of the later are negligible. The second model has the same assumptions as model 1, but in addition assumes that there is a probability of preference of the insect vector for humans, depending on the size of their populations. This model establishes conditions under which the disease in the human can stay in endemic state, that is, the infection once it has invaded a locality it persists for a long time. If each case of cutaneous leishmaniasis generates, in average, more than a case during the infection period, producing an epidemic that reaches a stable endemic equilibrium. This model demonstrated that although this condition is not satisfied, it is likely that an outbreak epidemic appears in some regions where normally do not occur. The third model approaches the problem of the transmission of the disease and the participation of the human as reservoir of the parasite from another social situation of the country. This model hypothesizes that the troops or other groups involved in the armed Colombian conflict are responsible to originate epidemic outbreaks in towns where there are vectors but parasite are absent. The model considers that after passing several weeks or months, soldiers quartered around in a town, where there is a high sandfly biting rate, a high probability of transmission occurs. Hence, the infected populations reach quickly the endemic equilibrium, typical of epidemic outbreaks.

References

[1] Travi B., Montoya J., Solarte Y., Lozano L., and Jaramillo C., Leishmaniasis in Colombia. I. Studies on the phlebotomine fauna associated with endemic foci in the Pacific coast region. Am.J.Trop.Med. & Hyg. 39:261-266. 1988.

[2] Montoya-Lerma J., Palacios R., Osorio L, Jaramillo C. and Cadena H. 1998. Further evidence of humans as source of Leishmania Viannia for sandflies. Memorias do Instituto Oswaldo Cruz 93: 735-736.

Modeling indirect transmission of pathogens between host populations sharing environment

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Keywords: metapopulation, buffer zone, epidemiology, R_0, compartmental model.

In a metapopulation, between-patch contact occurs via movements of individuals or via neighboring relationships. In the short term, we can assume that no movement of individual occurs between patches. If a disease is present in a patch, it will first spread in the neighborhood of infected patches.

In this short-term context, we consider a pathogen which spreads only indirectly between individuals, either within or between patches. We assume this pathogen is shed and survives in the environment and that the main transmission route is a contact with the contaminated environment. An example of such a pathogen could be parasites with a free-living stage, or bacteria (e.g. *Salmonella*).

Competing hypotheses exist about the influence of patches overlapping on pathogen spread in a metapopulation (e.g. Nunn and Dokey, 2006). Decreasing patches overlapping reduces the home range and enhances its use, increasing pathogens accumulation in the environment. However, increasing patches overlapping (e.g. individuals of different patches share environment for their foraging activity) enhances between-patch transmission. At contrast, in territorial species patches overlapping is reduced. However, edges are marked by 'patrols', whose marks may be contaminated and visited by neighbors. Exposure to infection is then heterogeneous.

We consider a metapopulation whose patches are separated by a buffer zone used occasionally by individuals of neighboring patches. Individuals cannot migrate or visit other patches. Two cases are studied: (1) the buffer zone is used occasionally by all individuals, e.g. as a foraging area; (2) only some individuals 'patrol' edges. We evaluate the influence of the size of edges, i.e. patches overlapping, and of the time spent in this zone by individuals on pathogen spread.

An SIR dynamics is considered within-patch, with $1/\lambda$ the length of the infectious period. The number of infectious units in the environment is explicitly modeled by E_x in patch x and E_{xy} in the buffer zone between patches x and y. Infectious individuals shed γ infectious units per time unit in the environment. Infectious units are removed from the environment at a rate μ. Each individual spends a proportion Δ of its time in the buffer zone.

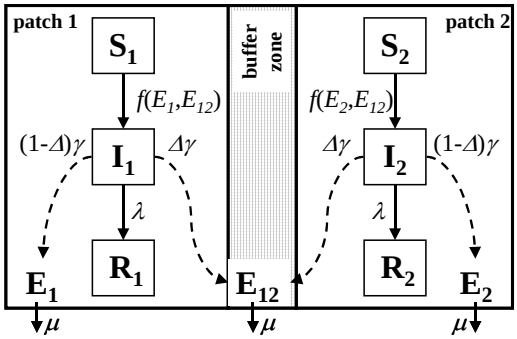

Fig. 1: SIR within-patch model with indirect transmission in a 2-patch metapopulation.

The basic reproduction number is calculated for a 2-patch system. The n-patch case is studied numerically.

Our model may help to understand the role of landscape fragmentation on disease spread in a metapopulation, especially if fragmentation is related to a differential space use by individuals.

References

[1] C.L. Nunn, A.T. Dokey, Ranging patterns and parasitism in primates. Biol. Lett. 2(3), 351-4, 2006.

Spiders as a biological controllers of the Langa Astigiana vineyards

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Keywords: spiders; predator-prey population models; biological control; agroecosystems.

Spiders are typical terrestrial predators that show a high degree of diversity in agroecosystems according to prey capture strategies. Wise [6] proposed the spider as a model terrestrial predator and specified that some spider families differ so much in how they forage and utilize their surroundings that it may prove risky to generalize about their role in terrestrial communities. For example, both web builders and wandering spiders frequently face a shortage of prey in nature. Confronted with prey shortages, web builders generally choose the sit-and-wait strategy, remaining still and waiting for an increase, while wanderers search for more productive microhabitats. Many wandering spiders, in fact, select microhabitat on the basis of prey abundance [1, 3, 5]. Moreover, several studies suggest that spiders select actively preys in a manner to optimize the proportion of essential amino acids in the diet [4, 2]. So, the composition of spider communities is strongly influenced by the physiognomy or physical structure of the environment. Wise [7] suggested that the way vegetation structure influences spider communities is rather complex and must take into account several variables like microclimate, prey availability and exchange with adjacent areas. For this particular character of the spider as a predator, they play an important role as a biocontrol agents. The contribution of spiders to pest control in agroecosystems can be considered mostly as the action of the whole group on the pest community. In [7] it is argued strongly for the importance of the spider assemblages in limiting insect pests in agroecosystems. Their role as biological controllers constitutes a challenging topic in applied ecology. Here we propose some mathematical models to understand the spiders' role as an biological controller in the agroecosystems with relevant economic impact for the first wine producing region in the world. We incorporate also the effect of human intervention through insecticide spraying and its relationship with the biological pest controllers.

References

[1] Edgar, W.D., 1971, The life-cycle, abundance and seasonal movement of the wolf spider, Lycosa (Pardosa) lugubris, in central Scotland, Journal of Animal Ecology 40(2), 303-322.

[2] Greenstone, M.H., 1979, Spider feeding behaviour optimises dietary essential amino acid composition, Nature, 181, 501- 503.

[3] Kronk, A.E., Riechert, S.E., 1979, Parameters affecting the habitat choice of a desert wolf spider, Lycosa santrita Chamberlin and Ivie, J. Arachnol. 7, 155-166.

[4] Miyashita K., 1968, Quantitative feeding biology of Lycosa T-insignita Boes et Str (Araneae: Lycosidae) under different feeding conditions, Applied Entomology and Zoology, 3, 81-88.

[5] Morse, D.H., R.S. Fritz, 1982, Experimental and observational studies of patch choice at different scales by the crab spider Misumena vatia, Ecology, 63, 172-182.

[6] Wise, D.H., 1984, The role of competition in spider communities: insights from field experiments with a model organism, in Ecological Communities: Conceptual issues and evidences Strong, D.R., D. Simberloff, L.G. Abele & A.B. Thistle (eds.)., Princeton University Press, Princeton, 42-53.

[7] Wise, D.H., 1993, Spiders in Ecological Webs, Cambridge University Press, Cambridge, UK.

Modelling disease spreading in symbiotic communities

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Keywords: ecoepidemiology; population models; epidemic models; symbiotic communities; stability; bifurcations.

Ecoepidemiological models describe disease transmission in interacting populations. We focus here on a nonlinear (standard) incidence function and its consequences on symbiotic communities, extending the model considered in [1].

References

[1] Venturino, E., How diseases affect symbiotic communities, Math. Biosc. 206, 11-30, 2007.

The Evolution of the Assortative Mating in Sympatric Speciation

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Keywords: evolution of assortative mating; reproductive isolation; sympatric speciation.

We have studied a computational model in order to show the evolution and emergency of the assortative mating in a sympatric process with disruptive selection. Both evolution and emergency of the assortative mating happen in two distinct situations. In the first case, the marker trait of the individuals is totally responsible for their ecology. In the second case, the individuals have two trait expressed by two different loci: a trait totally responsible for the ecology and another one independent of the ecology. Our results for this model shows the coupling between the two loci and the evolution from a random mating state to a assortative mating state.

References

[1] A. Kondrashov and F. Kondrashov. Interactions among quantitative traits in the course of sympatric speciation. Nature 400, 351 (1999).

[2] U. Dieckmann and M. Doebeli. On the origin os species by sympatric speciation. Nature 400, 354 (1999).

[3] M. Kirkpatrick and V. Ravigné. Speciation by Natural and sexual selection: models and experiments. American Naturalist 159, S22 (2002).

[4] S. Geritz and E. Kisdi. Adaptative Dynamics in diploid, sexual populations and the evolution of reproductive isolation. The Royal Society, 1671 (2000).

[5] D. Udovic. Frequency-Dependent Selection, Disruptive Selection, and the Evolution of Reproductive Isolation. The American Naturalist, Vol. 116, No. 5 (Nov. 1980).

[6] D. Ortiz-Barrientos, B. A. Counterman and M. A. F. Noor. The Genetics of Speciation by reinforcement. PLoS Biol 2(12):e416.

SEASONAL EFFECTS ON THE WEST NILE VIRUS INFECTION

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Keywords: West Nile Virus, dynamics, nonlinear, stability, mosquitoes, seasonality.

ABSTRACT: We study the effects of seasonal changes on the dynamics of the West Nile Virus infection. We find a mechanism of parametric resonance responsible of the creation of new infection outbreaks starting from an endemic situation.

References

[1] G. Cruz-Pacheco, L. Esteva, J. A. Montaño-Hirose, Cristóbal Vargas. A Mathematical Model for the dynamics of the West Nile Virus. System, Structure and Control 2004, ELSEVIER,2004,PP .475-480.

[2] G. Cruz-Pacheco, L. Esteva, J. A. Montaño-Hirose, Cristóbal Vargas. Modelling the dynamics of West Nile Virus. Bull. Math. Biol. 2005, 67, 1157-1172.

[3] P. Reiter. From Shakespeare to Defoe: Malaria in England in the Little Ice Age. Emerg. Infec. Dis. 2000, 6 (1) 1-11.

Control of spatially structured pathogenic bacterial populations by probiotics: modeling and simulation of a mixed-culture biofilm system

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Keywords: probiotics, pathogens, biofilm, quasi-linear diffusion, pH, simulation

Probiotic bacteria are defined to be live food ingredients that confer health benefits to the host system if administered in sufficiently large quantities. For many decades the prevalent use of probiotic bacteria has been as a functional food or nutraceutical, often administered as an additive or natural ingredient of dairy products. Only recently attention was brought to their potential as an alternative to antibiotics in the control of pathogens. Most bacteria, including pathogens and probiotics, live in biofilms. These are complex spatially structured communities of microorganisms embedded in a self-produced polymeric matrix (EPS), which offers improved living conditions (resistance to toxins; exchange of nutrients; beneficial enzymatic interactions). While in the recent decade many papers have been published that model the action of antibiotics against pathogenic biofilms, we present here a first model of probiotic action against such a biofilm community. The literature claims several possible mechanisms of probiotic action against pathogens, depending on the species involved. The specific system that we have in mind is the hardy food-bourne pathogen *Listeria monocytogenes* that is controlled by *Lactococcus lactis* by a modification of the pH such that it becomes unfavorable for the pathogen.

The probiotic biofilm control model is formulated as a system of five quasi-linear diffusion-reaction equations, following a biofilm modeling framework that was introduced in [1] for a mono-species biofilm system. Compared to the single species case, a multi-species model like the one developed here also needs to take competition for space into account. The model is expressed in terms of the independent variables biomass density of probiotic bacteria N_1 and pathogenic bacteria N_2, inert biomass N_3 (we do not distinguish between inert pathogens and probiotics) and the concentrations of the controlling substrates carbonated acids C and protonated ions P. The pH value is a monotonously decreasing function of the latter, $\mathrm{pH} = -\log_{10} P$.

The density dependent nonlinear diffusion coefficients $D_i(N)$ of the particulate substances N_i show two non-standard effects: (i) a degeneracy as in the porous medium equation for vanishing biomass density $N = N_1 + N_2 + N_3 = 0$ and (ii) a fast-diffusion singularity if the total biomass approaches the maximum possible cell density, $N \to 1$. Effect (i) causes the biofilm to spread only notably for relatively large biomass densities $N >> 0$ and induces a sharp interface between biofilm and liquid phase [initial data with compact support imply solutions with compact support], while (ii) ensures that the maximum cell density is obeyed, i.e, $N \leq 1$. Both effects together are required to describe biofilm formation. The diffusion coefficients for the dissolved substrates C and P depend on the biomass densities as well, albeit in a rather uncritical way. They are bounded between two a priori known positive constants. Thus diffusion of C and P behaves essentially like Fickian diffusion.

The interplay between biomass fractions and dissolved substrates is described by nonlinear reaction terms. The growth limiting substrates C, P are produced by the bacteria. On the other hand, the growth of both bacterial populations is inhibited by C and P. They enter a decay stage if one of the substrate concentrations becomes large. Then active biomass is converted into inert biomass.

We shall remark on the existence of solutions of the model and present a finite-difference scheme that is based on a non-local (in time) discretisation of the nonlinear diffusion coefficients. The model will be studied in an extensive numerical experiment consisting of a multitude of two-dimensional computer simulations. We will focus on the effect that inocculation of probiotics has on the fate of the pathogenic biofilm under uncertainties in the reaction parameters.

References

[1] Eberl HJ, Parker DF, van Loosdrecht MCM. A new Deterministic Spatio-Temporal Continuum Model For Biofilm Development, *J. Theor. Medicine*, 3(3), 2001

The effect of spatial scale on meta-population patch models for infectious disease in the United Kingdom

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Keywords: Meta-populations, spatial scale, infectious disease

Increasing concern about the threat posed by new emerging and re-emerging infectious diseases, such as possible pandemic strains of human influenza and the bio-terrorist release of smallpox, has recently demonstrated the importance of increasing the UK's capacity to predict and control such events. It is widely acknowledged that mathematical modelling is one such tool to address this.[2]

To reflect spatial heterogeneity of the UK recent models have have utilised either individual agent-based structures [1] or spatial meta-population patch models [4]. In this paper we examine the latter approach and consider the effect that the choice of size of the meta-populations has on the global dynamics in the case of two different diseases. Firstly, smallpox is a highly transmissible virus but has features which allow it to be easily controlled: a large proportion of the secondary cases are a result of overtly symptomatic individuals [3]. Recognition of these cases can result in prompt isolation and pre-emptive vaccination campaigns lessening the impact. Secondly, pandemic influenza is slightly less transmissible, but a large number of infectious cases may be asymptomatic and disease progression is over a few days meaning that robust intervention strategies are focussed around treatment. Smallpox cases are also overtly ill for a week rather than a few days, so despite higher disease transmissibility than influenza the expected daily transmission is lower than that expected by influenza cases.

We take a similar approach here to [4] and consider the UK as consisting of distinct administrative regions: Government Office Regions, counties, districts and wards. At each level, the patches are of roughly equal population sizes, ranging from 5-10 million at the GOR level, down to 1000 at the ward level. The individual patches are homogeneously mixed with the connection between them being due to daily commuter movement.

As the number of patches increases from around 500 districts to around 10000 wards the computation work required increases significantly with spatial scale. It is important, therefore, to ensure that we capture the global dynamics of the disease, with any interventions, at as large a scale as possible so that the necessary computation for reliable assessment of any policy may be completed in a reasonable time.

As we vary the patch size we compare the resulting global dynamics, paying particular attention to the spatial spread of disease activity. We see that the smaller patch populations at the smaller scales lead to earlier saturation of the growth of new cases. For smallpox, with its slow spatial spread (delayed propagation between patches), the national epidemic curve exhibits significant qualitative changes. For more rapidly propagating influenza this effect is less pronounced from district to ward level, but still presents an issue for real-time prediction of national incidence. Finally, we consider the implications of the choice of model structure on any assessment of the epidemic scenario.

References

[1] N.M. Ferguson, D.A. Cummings, C. Fraser, J.C. Cajka, P.C. Cooley, and D.S. Burke. Strategies for mitigating an influenza pandemic. *Nature*, 442:448–52, 2006.

[2] N.M. Ferguson, M.J. Keeling, W.J. Edmunds, R. Gani, B.T. Grenfell, R.M. Anderson, and S. Leach. Planning for smallpox outbreaks. *Nature*, 425:681–685, 2003.

[3] C. Fraser, S. Riley, R.M. Anderson, and N.M. Ferguson. Factors that make an infectious disease outbreak controllable. *Proceedings of the National Academy of Sciences of the United States of America*, 101(16):6146–6151, 2003.

[4] I.H. Hall, J.R. Egan, I. Barrass, R. Gani, and S. Leach. Comparison of smallpox outbreak control strategies using a spatial metapopulation model. *Epidemiology and Infection*, IN PRESS, 2007.

Spontaneous Emergence of Spatial Patterns in a Predator-Prey Model

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Keywords: individual based model, spatial pattern formation, prey-predator system, paradox of enrichment.

Mathematical modelling of population dynamics is widely studied due to many interesting features found on the organization of the individuals in nature. Since first attempts of modelling interacting populations by Lotka and Volterra, many works has been done in order to obtain a model that can represent those interactions in a most realistic way. The model initially proposed by these authors does not cover some critical variables observed on the real systems, such as movimentation skills and individual positions which are critical factors for the prey and predators survival, since predators are able to hunt preys within a nearby region only.

This characteristic can be related to diffusion phenomena. Diffusion models are commom in many natural phenomena, in particular in population dynamics studies. Diffusive populational models shows that the populations can be distributed in a heterogeneous way and exhibit distinct patterns like travelling waves and chaos [1]. The predator prey model can have your stability properties changed with the diffusive term insertion. It is been stated by Wilson and De Roos that spatial predator-prey systems are considerable more stable than the aspatial ones.

Adding some characteristics in the mathematical model generally increases hardly the difficulty level of the analysis, like diffusion of the population in a spatial model. To overcome this difficulty, some alternative methods are proposed which extensive mathematical analysis is not needed in order to obtain the desirable results. Some strategies of analysing and simulating these models includes Individual Based Models (IBM) using cellular automata; Individual Based Models without cellular automata; applying mean fields approximation. Finite element methods and pertubative methods are other alternative approaches to understand these systems. Our choice in this work is to use Individual Based Models (IBM) with cellular automata. It consists of turning the mathematical terms of the model into rules of interactions between individuals of the populations. On this method, time is discretized into time-step or iterations, space is discretized into cells or sites which form a lattice, the domain of the simulation, and the populations are discretized into individuals placed around the lattice. Individuals relate to each other according to cellular automata rules.

The main advantage of IBM is to generate models with many additional features observed on real systems without increasing the computational work exponentially, such as delay models, time history models and working with models whose each individual has a particular information, like the genetic code and age [2, 3].

Our work focus mainly on the spatial pattern emerged by an open three-trophic food chain and their relationship with the time series. Keitt et al. [4] discussed emergent patterns in diffusion-limited predator-prey interaction introducing spatial heterogeneity in the model. We observed emergent spatial patterns without this mechanism. Our model presents an self organization derived mainly to the dynamics to the system. Supported by IBM, we propose the predator-prey system consisting on a fixed plant population, a prey population, designate herbivore, which gather energy from plant population and it is able to diffuse through the system and the predator population which needs to hunt herbivores in order to reproduce and that is able to diffuse through the system as well as the herbivores.

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[1] Keitt, T., Ecological Modelling **102**, 243-258 (1997).
[2] Penna, T. J. P. and de Oliveira, S. M., Journal de Physique I, France **5**, (1697-1703), 1995.
[3] Charret, I. C., Costa Jr., A. T. and Louzada, J. N. C., to appear.
[4] Keitt, T. H. and Johnson, A., Journal of Theoretical Biology **172**, 127-139, (1995).

Hazard function for cancer patients and cancer cells dynamics

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Keywords: Hazard function, kernel estimation, Gompertzian growth

Survival analysis belongs to classical parts of mathematical statistics and occupies an important place in medical research (e.g. [1]). The goal of the paper is to treat cancer patients survival data both by parametric and nonparametric methods.

We propose a parametric form of hazard function for cancer patients based on a simple and acceptable assumption — the hazard depends on proliferation speed of cancer cells — and we describe a method for parameter estimation ([4]).

The nonparametric methods seem to be more adequate for survival analysis because there are rarely sound reasons for choosing a particular parametric model. A very effective nonparametric method is a kernel estimate of a hazard function. The properties of these estimates have been investigated by many authors with different techniques; see e.g. [2], [3], [5], [6], [7], [8].

Nevertheless, there is a serious difficulty with it – the determination of a smoothing parameter (bandwidth). Some of the proposed methods for optimal bandwidth selection depend on the hazard function being estimated. Therefore, one needs an "initial" estimation of the unknown hazard function and its second derivative and the developed parametric method can serve for this purpose.

On the contrary, a nonparametric estimate can be a basis for estimation of hazard function parameters. This idea suggests a kind of iterative method – parametric estimation gives an optimal bandwidth for a kernel method, then estimated function serves to more precise approximation of parameters.

The advantage of this approach consits in the fact that an optimal bandwidth can be estimated even in case of a small number of observations.

A biological or clinical relevance of parameters appearing in hazard functions should be a subject of future discussion with oncologists. These parameters may serve as a heuristic tool for classification of various cancer types or for comparison of different treatment methods. The benefit and limitations of the both approaches are discussed and illustrated on survival data provided by Masaryk Memorial Cancer Institute in Brno, Czech Republic.

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References

[1] Collett D. (2003). *Modelling Survival Data in Medical Research*. Chapman & Hall/CRC: Boca Raton-London-New York-Washington,D.C.

[2] Horová I., Zelinka J and Budíková M. (2006). Estimates of Hazard Functions for Carcinoma Data Sets. *Environmetrics*, **17**, 239–255.

[3] Horová I. and Zelinka J. (2006) Kernel Estimates of Hazard Functions for Biomedical Data Sets. In *Applied Biostatistics: Case studies and Interdisciplinary Methods*, Springer.

[4] Kozusko F and Bajzer Ž. (2003). Combining Gompertzian growth and cell population dynamics. *Mathematical Biosciences*, **185**, 153–167.

[5] Müller H.G. and Wang J.L. (1990). Nonparametric Analysis of Changes in Hazard Rates for Censored Survival Data: An alternative Change-Point Models. *Biometrika*, **77**(2), 305–314.

[6] Ramlau-Hansen H. (1983). Counting Processes Intensities by Means of Kernel Functions. *The Annals of Statistics*, **11**(2), 453–466.

[7] Tanner M.A. and Wong W.H. (1983). The Estimation of the Hazard Function from Randomly Censored Data by the Kernel Method. *The Annals of Statistics*, **11**(3), 989–993.

[8] Uzunogullari U. and Wang J.L. (1992) A comparision of Hazard Rate Estimators for Left Truncated and Right Censored Data. *Biometrika*, **79**(2), 297–310.

Modeling Uncertainty and Data Quality

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Keywords: uncertainty, data quality, modeling, monitoring, metrics

Data quality is an important characteristic feature of data monitoring and collection. When making evaluations and conclusions from collected data and based on them the population dynamics models and finally the decisions, one has to be very careful not to make fatal mistake. The important task of computing technology is to deal with the primary data quality and consider its uncertainty and thus to reduce the risk of decision mistake.

Imprecision and uncertainties of deterministic and stochastic models for population dynamics depend also on monitoring and collecting biological, environmental and medical data and information – these are necessary to start with the model research and to confirm and optimize the model.

There are some theoretical frameworks for understanding data quality to decrease their imprecision and uncertainty. The quality of information obtained from processing primary data and information is commonly measured by a number of factors including: *relevance, timeliness, accuracy, accessibility and clarity of the information, comparability of the statistics* and *coherence*.

Main methods of dealing with uncertainties are based on interval arithmetic, fuzzy and probabilistic approach [1]. Each of them has its strengths and weaknesses and needs different algorithm and data representation.

All theoretical demands mentioned above are satisfied and combined by the new proposed modular approach, where these demands are fulfilled as data attributes of separate modules. Uncertainty is here classified as one of four types: *parameter uncertainty, model uncertainty, decision rule uncertainty* and *variability*. There are three degrees of uncertainty distinguished: *technical uncertainties, methodological uncertainties* and *epistemological uncertainties*.

Every module has its own data model with some constants, parameters and numerous attributes, reflecting all requirements of uncertainty analysis mentioned above (parameters will be the subject of automated optimization, which relies on the later measured data, which are again burdened by the uncertainties and are represented the same way as the primary data). None from the modules is mandatory, as there are often the situations when there is nothing known about the condition of the data. In such cases the default module is used and some extra data are collected e.g. from public sources with the respect to human error pursuit.

All these attributes from all modules are mapped into one main feature of the data – the metrics – and resulting population dynamics model gives not only the required results (with basic statistical moments), but also their more accurately marked metrics, based even on the uncertainty in the knowledge behind the model (given by primary data). The mapping function is really complex as the domain of definition is so wide and diverse. Also the arithmetic is defined to make the combinations possible.

The main difference of the new proposed model prototype lies in representation of the input data and easy implementation and sharing of the modeling results. The strength of the model is that it is able to catch the data uncertainty arising mainly from human factor and also to catch the data uncertainty when any other ways of uncertainty measurement are not present.

References

[1] J. Hřebíček, M. Hejč, I. Holoubek. Current Trends in Environmental Modelling with Uncertainties. In Proceedings of the iEMSs Third Biennial Meeting "Summit on Environmental Modelling&Software". Burlington: International Environmental Modelling and Software Society, USA. 342-347. 2006.

ARROW - Assessment of ecological state of surface waters in the Czech Republic

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Keywords: biomonitoring; multivariate analysis; environmental modeling.

The ARROW project is the project of implementation of EU Water Framework Directive in the monitoring of surface waters of the Czech Republic and covers all aspects of this problem from data sampling to informatics solution (focusing on the ecostat). The project is based on long term development of this field in the Czech Republic and it is carried out under the supervision of the Ministry of the Environment of the Czech Republic.

The important part of the ARROW project is the development and implementation of a Czech approach to the evaluation of the ecological state using an analysis of monitoring data. The main idea of the system is an approach based on a network of reference sites and robust multivariate modeling of expected environmental and biological conditions. The approach presented is compatible with the EU Water Framework Directive and is implemented in the national-wide information system of the ecological state of surface waters of the Czech Republic.

The evaluation of ecological state is based on two former projects; the direct predecessor is TRITON project aimed on analysis of biomonitoring data from small watercourses which has been developed since 1999. The other source of inspiration and especially reference dataset is also PERLA system, which should be considered as a scientific background for biomonitoring activities in the Czech Republic.

Concerning analysis of surface water quality based on biological communities, there are several methodology approaches: single metrics, multimetric and a multivariate approach. The ARROW implements all these metrics on different levels of computation. The main approach based on multivariate comparison with the reference quality is very simple and it is implemented in almost all complex systems of surface water quality assessment; however, its implementation presents a difficult task. The process could be divided into three steps, each of them with its own methodology problems: preparation of the reference model, classification of unknown cases (sites) into reference categories and comparison of an unknown site and the reference status, i.e. assessment of unknown site quality. In the third step the difference in single metrics between observed and predicted state are used together with multivariate differences between biological communities for partial evaluation of the ecological state. Above the direct comparison with expected natural state there is multimetric combination of partial measures which forms the final measure of ecological state.

The principal basis of the whole analysis is the quality of the reference dataset, which should homogeneously cover all environmental conditions in the analyzed area and contain minimal influence of human activities.

Unfortunately, in the conditions of Central Europe, it is almost impossible to find real ”natural” sites; thus, localities in the reference dataset originate from long-term knowledge of the considered sites and consensus of hydrobiology experts on what is the site’s “nearest-to-natural-conditions” status.

The methodology is flexible and robust and could be adopted for different types of data and biological communities. All levels of the process are covered by objective statistical methodology and monitored by experts; their computations are based on robust multivariate and multimetric methods, some of them newly developed.

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References

[1] Jarkovský, J et al., Assessment of Ecological State of Surface Waters in ARROW Project: Robust Multivariate Predictive Models. Proceedings of Summit on Environmental Modelling and Software. IEMS, 2006.

Analysis of the outputs of a simulator for the propagation of powdery mildew over vine with a SEIR model

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Keywords: Powdery mildew, vine, phytopathology, SEIR model, parameter identification.

Powdery mildew, caused by the fungus Uncinula necator, is the most economically important and widespread disease of grapevines. A better knowledge of its propagation mechanisms could help to improve the treatment strategies.

A complex mechanistic model at the vine stock scale has been devised by a pluridisciplinary team of biologists, mathematicians and computer scientists, see [1]. It couples a 3D architectural model for the host growth with a model for the pathogen development and its dispersal.

In this talk, we will consider simpler mathematical models based on differential equations. Using the outputs of the mechanistic model, our goal is twofold: first we want to determine whether a simple model is able to catch the main dynamics of the disease, and then we expect to obtain numerical estimates of the parameters that are still ill known of the biologists. The method we use is a least squares approach.

At the vineyard scale, the previous models are extended with Fickian diffusion and we investigate the existence of travelling wave solutions.

References

[1] Agnès Calonnec, Guillaume Latu, Jean-Marc Naulin, Jean Roman, Gaël Tessier: Parallel Simulation of the Propagation of Powdery Mildew in a Vineyard. Euro-Par 2005: 1254-1264

Mathematical Modeling and Simulation of the Immune System Dynamics in Human Herpesvirus-6 Infection

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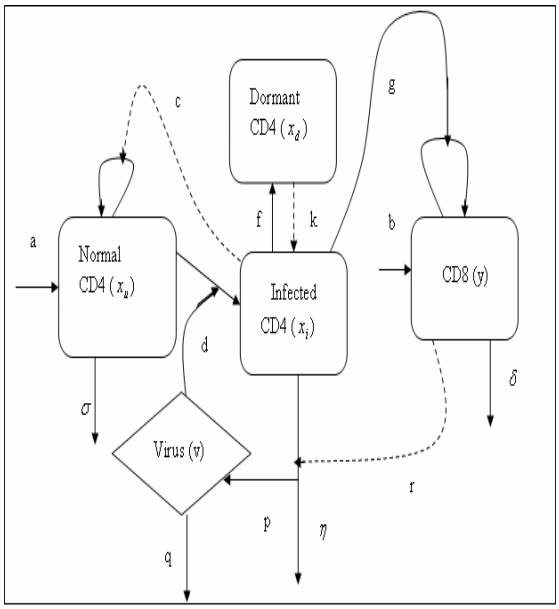
Keywords: Human Herpesvirus-6, CD4 T cells, Dormant Cells, CD8 T Cells, Primary and Secondary Infection.

Human Herpesvirus-6 (HHV-6) induced mononucleosis-like illness is a benign, self-limited, lymphoproliferative disease without major immune deficiency. HHV-6 is one of the most widespread members of the family of human herpesviruses infecting up to 90% of the population during infancy, as a primary infection. After the infection the virus can persist for the lifetime of the host. However, if a secondary infection by pathogens occurs, the infected dormant T-cells are reactivated and the viral DNA is replicated.

We present a model that simulates the virus-immune dynamics restricted to HHV-6. The immune system is represented by two T-cell populations, CD4 T-cell and CD8 T-cell.

The following diagram is the schematic representation of our model.



In this diagram, x_u is the uninfected CD4 T-cell population, x_d the dormant-infected CD4 T-cell population, x_i the infected CD4 T-cell population, y the CD8 T-cell population, and v the vírus load in terms of DNA copies ($\log_{10}/\mu l$). In addition to the natural dynamics of production and clearence of T cells and the virus, we include the indirect stimulation of the proliferation of uninfected CD4 T cells and the CD8 T cells by the infected CD4 T cells ($c(\frac{x_i}{K_1+x_i})x_u$ and $g(\frac{x_i}{K_2+x_i})y$), and the removal of infected CD4 T cells by CD8 T cells ($\eta\{1+r(\frac{y}{K_3+y})\}x_i$). We assume that a fraction of the infected cells will accumulate as dormant cells ($f(K_4-x_d)x_i$) and pathogens can activate these cells (kx_d). Free vírus buildup is assumed to be due to the lysis of the infected T-cells ($p\eta\{1+r(\frac{y}{K_3+y})\}x_i$).

We will present the corresponding mathematical model that consists of a system of nonlinear ordinary differential equations, the solutions of this system under different purturbations, and comparison studies of a few simulation results with known experimental results.

Analytical Internet Tool for Cancer Epidemiology in the Czech Republic – a Computational Base for Predictive Risk Models

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Keywords: cancer incidence, prevalence, survival, human health, population risk

Cancer epidemiology can be regarded as one of the most important and most frequently analysed topic in the field of human risk assessment. The bio indication from epidemiological data of course requires sufficient data sources. It means representative long-term profiles of incidence and mortality and a very good awareness of most important risk factors in population dynamics. Analyzing epidemiological trends we have to be able to distinguish between statistically significant trends and random fluctuations. For risk assessors of population dynamics it would be even more important to recognize environmentally related cases that can be attributed to external factors like pollution of air, drinking water and/or food. And again, the influence of these factors must be filtered on the substantial background of the other "natural" risk factors of population dynamics like age structure of the population, genetic factors or frequently omitted life style. Apart from these complicated circumstances we can concentrate our attention to some cohorts of cancer patients that might probably indicate external impact – if they increase in incidence and non-randomly in some regional or temporal scales.

All these analyses require support in estimates of incidence rate and easily available large data sets that are itself very expensive and typically not directly accessible - epidemiological cancer registries. In addition to it, we must aggregate cancer data with demographic data in order to attain for example age-specific profiles of incidence. We used in these analyses models that are based on estimates of *cancer incidence* [1], *prevalence* [2] and *survival*. We combined these components in a novel type of multi-stage model that consecutively works with newly diagnosed patients (the estimated incidence of cured patients) and with number of cancer patients diagnosed and cured in previous years with some probability to reach relapse or progression in a given year that is predicted. The approach combines epidemiological trends in incidence and prevalence with demographic databases (used for estimates of relative X-yrs survival) and with cancer-specific mortality data that were applied to estimate the rate of dissemination relapses of the disease. The functionality and power of the model is well documented on Czech National Cancer Registry with more than 1,4 million of cases reported since 1977. Long-term history of the registry allows even 30-years relative survival estimates with mortality data separating death events due to cancer from the other reasons.

Model outputs can be used for health care assessment as well as for population and environmental risk estimates. We supported the analyses with a new Internet tool on cancer epidemiology. This is web portal SVOD (http://www.svod.cz) that offers automatically generated and verified epidemiological analyses on cancer incidence and mortality in the Czech Republic, as well as the model estimates based on [1], [2]. The portal SVOD is developed in the Czech and English version. Its information services offer: *Current news*: regularly updated information about population risk assessment and tumour epidemiology; *Interactive analyses* that allow the user to investigate epidemiological trends of selected cancer diagnoses; *Predefined views of important topics (Authorised information service).*

References

[1] T. Dyba, T. Hakulinen, Comparison of different approaches to incidence prediction based on simple interpolation techniques, Statistics in medicine 19, 1741-1752, 2000.

[2] A. Verdecchia, G. De Angelis, R. Capocaccia Estimation and projection of cancer prevalence from cancer registry data. Statistics in medicine 21, 3511-3526, 2002.

The ultimate age-profile in an asymptotically linear population model

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Keywords: Continuous age-structured population models; equilibria and stability analysis; asymptotic age-profile.

Asynchronous exponential growth has been extensively studied in population dynamics. We find out the asymptotic behaviour of solutions in a non-linear age-dependent model which takes into account sexual reproduction interaccions rather than (the more frequently considered) competition interactions. The main feature of the system is that the non-linear process converges to a linear one as the solution becomes large. The steady states analysis and the corresponding stability analysis are completely made and are summarized in a bifurcation diagram according to the parameter R_0.

We introduce a normalized system, that is, an equation involving the age-profile (the age-density normalized to total integral 1), and perform another stability analysis. We have shown that both the stationary age-density and the stationary age-profile are unstable whenever exist ($R_0 > 1$).

Next we address the following question: does the solution stabilize in some sense? or more specifically, does the age-profile converge (in the L^1 norm) to an asymptotic age-profile?

We have found suitable conditions, according to the model parameters and the initial datum of the problem, for the existence and no-existence of the ultimate age-profile. On the one hand, we have determined a case such that the population goes to extinction but there is no convergence of the age-profile. On the other hand, we have shown that under suitable conditions the population grows unbounded and the age-profile converges to an asymptotic age-profile which turns out to be the stationary age-profile of the (linear) Lotka-McKendrick equation.

The proofs are based on an equivalent formulation of the original system as a non-linear renewal equation. For the convergence we need an additional hypothesis on the non-linearity of the problem.

References

[1] Iannelli, M.: *Mathematical Theory of Age-Structured Population Dynamics*, Applied Mathematics Monograph C.N.R. vol. 7, Giardini editori e stampatori, Pisa 1995

[2] Calsina, À., Ripoll, J.: A general structured model for a sequential hermaphrodite population, Math. Biosci. (2007), doi:10.1016/j.mbs.2006.09.014

[3] Gyllenberg, M., Webb, G.F.: Asynchronous exponential growth of semigroups of nonlinear operators, J. Math. Anal. Appl. 167 (1992), no. 2, 443–467

Inverse density dependence in the evolution of communication

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Keywords: Evolution of communication; Population dynamics; Inverse density dependence.

The case for the study of the evolution of language within a population dynamics framework was probably best made by Ferdinand de Saussure in a statement given at the famous course of lectures at the University of Geneva (1906-1911), "language is not complete in any speaker; it exists only within a collectivity... only by virtue of a sort of contract signed by members of a community" [1]. Translated into the biological jargon, this assertion means that language is not the property of an individual, but the extended phenotype of a population. For the purpose of mathematical modeling, language is viewed as a mapping between a fixed set of meanings (or objects) and a repertoire of signals [2].

Structured meaning-signal mappings, i.e., mappings that preserve neighborhood relationships by associating similar signals with similar meanings, are advantageous in an environment where signals are corrupted by noise and sub-optimal meaning inferences are rewarded as well. The evolution of these mappings, however, cannot be explained within a traditional language evolutionary game scenario in which individuals meet randomly, because the evolutionary dynamics is trapped in local maxima that do not reflect the structure of the meaning and signals spaces [3]. In addition, once a communication code is fixed in the population it cannot be changed even if a small fraction of the population adopts the more efficient structured code. This is essentially the Allee effect [4] of population dynamics that asserts that intraspecific cooperation might lead to inverse density dependence, resulting in the extinction of some (social) animal species when their population size becomes small (see [5] for a review). Of course, this effect is germane to the outcome of biological invasions involving such species as well as to the spread of linguistic innovations, regardless of their worth.

Here we use a simple analytical approach based on the game theoretical formulation of Eshel & Cavalli-Sforza [6] to show that when individuals adopting the same communication code meet more frequently than individuals using different codes – a result of the spatial organization of the population – then valuable linguistic innovations can spread and take over the population. This finding supports the "mother tongue" hypothesis that human language evolved as a communication system used among kin, especially between mothers and offspring [7].

References

[1] F. de Saussure, Course in General Linguistics. Translated by Wade Baskin. McGraw-Hill Book Company, New York, 1966.

[2] M.A. Nowak and D.C. Krakauer, The evolution of language. Proc. Natl. Acad. Sci. USA 96, 8028-8033, 1999.

[3] J.F Fontanari and L. I. Perlovsky, Evolving compositionality in evolutionary language games. IEEE Trans. Evol. Comput. doi:10.1109/TEVC.2007.892763, 2007.

[4] W. C. Allee, Animal Aggregations. A Study in General Sociology. University of Chicago Press, Chicago, 1931.

[5] F. Courchamp, T. Clutton-Brock and B. Grenfell, Inverse density dependence and the Allee effect. Trends Ecol. Evol. 14, 405-410, 1999.

[6] I. Eshel and L. L. Cavalli-Sforza, Assortement of encounters and evolution of cooperativeness. Proc. Natl. Acad. Sci. USA 79, 1331-1335, 1982.

[7] W. T. Fitch, Kin selection and mother tongues: A neglected component in language evolution, in: K. Oller & U. Griebel (Eds.), Evolution of Communication Systems: A Comparative Approach, MIT Press, Cambridge, MA, pp. 275-296, 2004.

Optimal Control of Mosquito Population Applying Insecticide

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Keywords: Optimal control, optimality conditions, Dubovitskii-Milyutin methodology.

We rigorously analyze a simplified distributed optimal control problem corresponding to a situation where one wishes to find an optimal trajectory in a sense to be clarified in the following. The admissible trajectories start at a fixed point (normalized to be the origin) and each of them is determined by a sufficiently regular function $\gamma : [0,T] \to \mathbb{R}^2$, with $0 < T < +\infty$ a given fixed final time. They are the possible trajectories of a device that is assumed to be continuously spreading a certain amount of insecticide as it goes around in an attempt to control a population of mosquitoes present in a C^2 bounded region $\Omega \subset \mathbb{R}^2$.

The main simplifying assumptions for the model are the following: (1) the mosquito population grows and diffuses respectively at constant rates $a > 0$ and $\alpha > 0$; (2) the insecticide immediately kills a fixed fraction of the population with an effective rate that decreases with the distance of the spreading source; this is mathematically realized by assuming that the effective killing rate at a certain point x obtained by spreading from a position $\gamma(t)$ is given by $b\,k(x-\gamma(t))$, where $0 \le k(\cdot) \le 1$ is a C^1-function with compact support and the constant $b > 0$ is the maximum killing rate; (3) there are no obstacles for the possible trajectories; We remark that these assumptions could be relaxed in several ways.

We want an optimal trajectory that at the same time reduces the mosquito population and the operational costs of the spreading device. In mathematical terms, we want to find an trajectory $\gamma^*(\cdot) : [0,T] \to \mathbb{R}^2$, the required optimal control, such that

$$F(\gamma^*) = \min\{F(\gamma) : \gamma \in \mathcal{A}\},$$

where $\mathcal{A} = \{\gamma \in H^1(0,T) : \gamma(0) = 0\}$ is the set of admissible controls, and

$$F(\gamma) = J(\gamma,u) \quad = \mu_0 \int_0^T |\gamma|^2 dt + \mu_1 \int_0^T |\gamma'|^2 dt \\ +\mu_2 \int_Q |u|^2 dx dt.$$

Here, $Q = \Omega \times (0,T)$; $\mu_0 \ge 0$, $\mu_1 > 0$ and $\mu_2 > 0$ are constants, and the mosquito population $u(x,t)$ satisfies

$$\begin{cases} u_t - \alpha\Delta u = a\,u - b\,k(x-\gamma(t))u & \text{in } Q, \\ (\partial/\partial n)(u) = 0 & \text{on } S = \partial\Omega \times (0,T), \\ u(0) = u_0 & \text{in } \Omega. \end{cases}$$

For this problem, we are able to prove the existence of an optimal solution and also characterize it by its corresponding first order optimality condition. This characterization is done by using the Dubovitskii e Milyutin methodology (see for instance [1], [2], [3],) which is based on the separation of certain cones associated to the functional to be minimized and the restrictions of the problem. Being $\gamma^*(\cdot)$ the optimal control, and $u^*(\cdot,\cdot)$ and $p^*(\cdot,\cdot)$ respectively the corresponding optimal state and adjoint state, the optimality conditions are the following:

$$\begin{cases} u_t^* - \alpha\Delta u^* = a\,u^* - b\,k(x-\gamma^*)u^* & \text{in } Q, \\ (\partial/\partial n)(u^*) = 0 & \text{on } S, \\ u^*(0) = u_0 & \text{in } \Omega, \\ -p_t^* - \alpha\Delta p^* = a\,p^* - b\,k(x-\gamma^*)p^* + u^* & \text{in } Q, \\ (\partial/\partial n)(p^*) = 0 & \text{on } S, \\ p^*(T) = 0 & \text{in } \Omega, \\ -\mu_1\gamma^{*''} + \mu_0\gamma^* - \mu_2 \int_\Omega p^* u^* k'(x-\gamma^*)dx = 0 & \text{in } (0,T), \\ \gamma^*(0) = 0, \quad \gamma^{*'}(T) = 0. \end{cases}$$

References

[1] I.V. Girsanov, Lectures on Mathematical Theory of Extremum Problems, Lectures notes in Economics and Mathematical Systems, 67, Springer-Verlag, Berlin, 1972.

[2] R. De Aguiar, Contribuições em Controle Ótimo Distribuído Via Formalismo de Dubovitskii-Milyutin. Aspectos Teóricos, Numéricos e Aplicados, Ph.D. Thesis, Unicamp-IMECC, 2002.

[3] F.P.M. Lopes, Alguns Problemas de Controle Multiobjetivos Governados por Equações Diferenciais Parciais, Ph.D. Thesis, Unicamp-IMECC, 2005.

The death-multiple immigration process as a tool for generating diverse fluctuation phenomena in discrete populations

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Keywords: Stochastic population models, fluctuations, monitoring, complexity

Construction of models is the most basic activity in science, lying at the heart of understanding, prediction, and practical applications. Uncovering the dominant mechanisms governing the deterministic evolution of nonlinear dynamical systems is an essential component of this activity. These systems are known to exhibit a wide variety of behaviour ranging from chaos to regular trains of discrete events. Various methods are employed to reduce the complexity of such problems. One important approach is to exploit the nonlinear interaction of quantities varying on different time and length-scales. This often makes it possible to eliminate rapidly fluctuating variables and represent the evolution of important properties of the system by *random* processes. Identifying stochastic models governing the behaviour of these properties then becomes the important driver for data acquisition and analysis. In reality measurements are always limited by practical considerations. Only finite data sets are available and these may relate only indirectly to the processes of interest. Thus distinguishing different models may present challenges.

In this paper we investigate an idealised version of this complex problem by discussing a specific stochastic population model that affords a great deal of flexibility in the type of behaviours it can produce, and so can be used as a tool with which to test wide varieties of data. The model is a variant on the canonical death-immigration model that generates a Poisson process that describes the fluctuations for the discrete number of individuals in the population. In this model, deaths occur in proportion to the instantaneous population size and single immigrants arrive spontaneously and are independent of the population size. The generalization of this model [1,2] allows the immigrations to occur in multiples, so that singletons, pairs, triplets – m-tuplets can arrive within the population at rates that are specific to their order. By pre-selecting the rates, specific non-Poissonian statistics can be generated for the fluctuations in the stationary state, and it is also shown how the inverse problem can be performed, whereby the rates for the multiple-immigrations can be determined from a prescribed stationary state.

The intrinsic fluctuations generated by the population will be discussed and the differences in prediction between the discrete phenomena and the continuum analogue will be highlighted.

The separate and practical problem of monitoring and characterizing the fluctuations is addressed by analyzing the statistics of individuals that leave the population. The fluctuations in the size of the population are transferred to fluctuations in the times between emigrants that form an intermittent time-series of discrete events. It is shown how easily realizable measures such as the continuous probability density functions for the time to the first event, inter-event times and the time to extinction, can be constructed and how these can be used to distinguish between population models whose mean-field evolution is identical. These diagnostic tools emphasize the importance of fluctuations in the dynamics of populations and illustrate the deficiencies of mean-field approaches in populations that have small size.

References

[1] E. Jakeman, K.I. Hopcraft, J.O. Matthews, ‘Distinguishing population processes by external monitoring’, Proc. Roy. Soc. Lond. (A), **459**, 623-639, 2003.

[2] J.O. Matthews, K.I. Hopcraft, E. Jakeman, ‘Generation and monitoring of discrete stable random processes using multiple immigration population models’, J. Phys. A: Math. Gen. **36**, 11585-11603, 2003.

Sublethal toxic effects in a simple aquatic food chain

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Keywords: Bifurcation analysis, bioaccumulation, ecosystem dynamics, ecological factors, global bifurcations, toxicological effects..

We study the sublethal effect of toxicants on the functioning (biomass production, nutrient recycling) and structure (species composition and complexity) of a simple aquatic ecosystem in a well-mixed environment (chemostat reactor). The modelled ecosystem consists of a nutrient consumed by a prey (e.g. bacteria, alga) which, in turn, is consumed by a predator (e.g. ciliates, daphnia) population. The dynamic behaviour of this ecosystem is described by a set of ordinary differential equations ODE's: one for the nutrient and one for each population [1]. The system is stressed by a toxicant dissolved in the in-flowing water. The transport of the toxicant is modelled using a mass balance formulation leading to an ODE. Bioaccumulation in the prey and predator populations is via uptake from the water phase, in case of the predator also via consumption of contaminated prey. Mathematically this process is described by a one-compartment model for the kinetics of the toxicant: uptake (from water and food) and elimination. The toxicant affects the development of individuals which make up populations [2]. In the model the physiological parameters depend on the internal concentration of the toxicant in individuals. Examples of physiological parameters are cost for growth or maintenance and assimilation efficiency. We use bifurcation theory [3]. In this way the parameter space is divided into regions with qualitatively different asymptotic dynamic behaviour of the system. As logical choice for bifurcation parameters are the strength of the forcing on the system determined by the input rate of nutrient and toxicant. Our analysis reveals that the relationship between the population biomass and the concentration of toxicant in the reactor is of paramount importance. The dynamic behaviour of the stressed ecosystem can be much more complicated than that of the unstressed system. For instance the nutrient-prey-contaminant system can show bi-stability and oscillatory dynamics. Due to the toxic effects a total collapse of the nutrient-prey-predator-contaminant system can occur after invasion of a predator, in which case both prey and predator population go extinct.

References

[1] B.W. Kooi, Numerical bifurcation analysis of ecosystems in a spatially homogeneous environment, Acta Biotheoretica 51(3)3 189-222 2003.

[2] S.A.L.M. Kooijman, Dynamic Energy and Mass Budgets in Biological Systems, Cambridge University Press, Cambridge, 2000

[3] Y.A. Kuznetsov, Elements of Applied Bifurcation Theory, 3rd Edition, Vol. 112 of Applied Mathematical Sciences, Springer-Verlag, New York, 2004.

Application of the *Fuzzy* Sets Theory in the prediction of the pathological stage of the Prostate Cancer

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Keywords: Mathematical Model, *Fuzzy* Sets, Biomathematics, Cancer.

In this work, we studied the construction of a mathematical model wich it was developed to predict the pathological stage of prostate cancer. The intention is to help specialist on the decision process about stage of the disease. The model consists on a system founded in *fuzzy* laws that it combine the pre-surgicals dates - clinic state, PSA levels and Gleason score - availing of a linguistic laws set made with base on informations of the existents nomograms. Herewith we were hoping to ger person's chance, with clinics characteristics determinates, is in each stage of tumor extension: localized, advanced locally and metastatic. Simulations were made with patient's dates of the Clinics Hospital/UNICAMP and the results were compared with Kattan's probabilities that are used on the medicals decisions.

References

[1] L.C. Barros, R.C. Bassanezi, Tópicos de Lógica Fuzzy e Biomatemática, UNICAMP/IMECC, Campinas - SP, 2006.

[2] M.J.P. Castanho, Construção e Avaliação de um Modelo Matemático para predizer a evolução do Câncer de Próstata e descrever seu crescimento utilizando a Teoria dos Conjuntos *Fuzzy*. Tese de Doutorado, Universidade Estadual de Campinas, Campinas - SP, 2005.

[3] U. Ferreira, A.C. Nardi, Câncer de Próstata. In. N. R. J. Netto, Urologia Prática, Atheneu, 4 ed., São Paulo, 1999.

[4] G.P. Silveira, Aplicação da Teoria de Conjuntos *Fuzzy* na predição do estadiamento patológico do Câncer de Próstata. Tese de Mestrado, Universidade Estadual de Campinas, Campinas - SP, 2007.

[5] A.J. Stephenson, M. W. Kattan, Nomograms for prostate cancer, *Journal Urological Oncology* 98, pp. 39-46, 2006.

Stochastic bottlenecks and Muller's ratchet

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In bottleneck serial transfers [2], microorganisms are cultivated for some time before small samples of the population be colected to seed new cultures. The bottlenecks simulate the loss of well-fit individuals in genetic drift and, after many passages, some aspects of the evolutionary dynamics emerge from the comparison of the replication rates of the mutants generated during the process and the rates of representants of the founders, preserved in antimutagenic conditions. Such experiments with RNA viruses supported the hypothesis of ocurrence of Muller's ratchet [3], a mutational degeneration phenomenon in finite populations.

It is remarkable that such complex systems can be mathematically modeled. In [1], a model was proposed to describe the behavior of Muller's ratchet in serial transfers of a unique organism in each bottleneck, besides other simplifying hypothesis. In this setting, the authors found the probability distribution of the minimum number of mutations in the population and theoretically justified the empirical fact that the logarithm of the mean fitness decays linearly with the number of transfers. However, it seems the "size" of the bottlenecks has measureable effects, besides the passage of just one individual being an extreme case.

This work shows that generating functions allow a systematic approach to the problem, generalizing the model for a variety of bottleneck sizes. In all the cases studied, the intensity of Muller's ratchet effect does not depend on the probability distribution of bottleneck size, suggesting the empirically observed decay could be related to absence of epistasis. Besides that, experiment gets simpler if isolation of a microorganism is not necessary and one expects bigger bottlenecks could reduce flutuations, so that the proposed formalism inspire hope that reliable estimatives of mutation and selection rates can eventualy be calculated.

This work has began when the author was in both Instituto de Física de São Carlos (IFSC) and Instituto de Ciências Matemáticas e de Computação (ICMC), in the Universidade de São Paulo (USP), and was supported by the Brazilian agency FAPESP.

References

[1] Colato, A. and Fontanari, J.F., Soluble model for the accumulation of mutations in asexual populations, *Phys. Rev. Lett.*, vol. 87, 238102 (2001)

[2] Elena, S.F. and Lenski, R.E., Evolution experiments with microorganisms: the dynamics and genetic bases of adaptation, *Nature Rev. Genet.*, vol. 4, pp. 457–469 (2003)

[3] Muller, H.J., The relation of recombination to mutational advance, *Mutat. Res.*, vol. 1, pp. 2–9 (1964)

Application of parasite-host-fodder plant system dynamics to the description of larch bud moth population cycles

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Keywords: time series analysis, dynamic model.

Analysis of time series of larch bud moth (*Zeiraphera diniana* Gn.) population fluctuations in Swiss Alps is based on the use of mathematical model of parasite-host-fodder plant system dynamics, which is presented in publication by P. Turchin et al. [1]:

$$N_{t+1} = \lambda_0 N_t \frac{Q_t}{\delta + Q_t} \exp\left[- gN_t - \frac{aP_t}{1 + ahN_t + awP_t} \right]$$

$$Q_{t+1} = (1-\alpha)\left(1 - \frac{N_t}{\gamma + N_t} \right) + \alpha Q_t$$

$$P_{t+1} = \phi N_t \left\{ 1 - \exp\left[- \frac{aP_t}{1 + ahN_t + awP_t} \right] \right\}$$

N_t is the density of larch bud moth, Q_t is the index of food quality, $0 \le Q_t \le 1$, and P_t is the density of parasites at moment t, $t = 0,1,2,\ldots$.

For identification the values of model parameters we used the time series from [2] (see also http://www.sw.ic.ac.uk/cpb/cpb/gpdd.html) and following statistical criterion:

$$LF(\vec{\alpha}, N_0, Q_0, P_0) \to \min_{\vec{\alpha}, N_0, Q_0, P_0}$$

where $\vec{\alpha}$ is vector of model parameters and N_0, Q_0 and P_0 are initial values of model variables. The loss-function LF has the following form:

$$LF(\vec{\alpha}, N_0, Q_0, P_0) = \sum_{t=0}^{N} (\tilde{x}_t - N_t^*(\vec{\alpha}, , N_0, Q_0, P_0))^2$$

where $N_t^*(\vec{\alpha}, N_0, Q_0, P_0)$ is solution of model for given values of model parameters $\vec{\alpha}$ and initial values of variables N_0, Q_0 and P_0.

Analysis all sub-models (Moran-Ricker model when $Q_t \equiv const$ and $P_t \equiv 0$; resource-consumer model when $P_t \equiv 0$; parasite-host model when $Q_t \equiv const$) showed that there is the absence of correspondence between theoretical and empirical time series as on qualitative as on quantitative levels. For whole model we expected good correspondence between theoretical and empirical time series on qualitative level: in model for best values of parameters there is cyclic regime of population fluctuations (in 8 years), the relation of the maximal density to minimal is of 6 orders, the parasitism maximum level is reached in two years after the population size maximum during the larch bud moth outbreak etc. But even in this case application of various statistical criterions for the analysis of residuals between theoretical and empirical datasets (in particular, Kolmogorov-Smirnov test, Shapiro-Wilk-Chen test, Durbin-Watson criterion etc.) shows that we have to reject the hypothesis that considering model can be applied for fitting of larch bud moth population fluctuations. It means also that we cannot conclude on the base of considering model that parasites play the main role in population regulation.

Additionally, the second equation of basic model doesn't describe the process of interaction between insect population and fodder plant. This equation needs in modification and in particular case it can be changed by the following equation

$$Q_{t+1} = f_1(N_t) f_2(Q_t) Q_t$$

In simplest case functions f_1 and f_2 can be presented in forms:

$$f_1(N_t) = (1 + rN_t)^{-1}$$

$$f_2(Q_t) = \min(u_1 + u_2 Q_t, 1)$$

where r, u_1 and u_2 are positive parameters.

References

[1] P. Turchin, S.N. Wood, S.P. Ellner, B.E. Kendall, W.W. Murdoch, A. Fischlin, J. Casas, E. McCauley, C.J. Briggs, Dynamical effects of plant quality and parasitism on population cycles of larch bud moth. Ecology, V. 84, N 5, 1207-1214, 2003

[2] W. Baltensweiler, A. Fischlin, The larch bud moth in the Alps. – In: Dynamics of forest insect populations: patterns, causes, implications. A. A. Berryman, editor. Plenum Press, New York, 331-351, 1988

The response of *Drosophila melanogaster* to modeled odor plumes with different shapes.

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Keywords: odor plumes, turbulence, chemotaxis, *Drosophila melanogaster*

Insects assess the availability of food and mates, as well as the presence of competitors and natural enemies using chemical information. The chemical substances involved are usually called "infochemicals" [1]. *D. melanogaster* uses food odors and aggregation pheromone to aggregate at suitable resources to avoid the negative effects of low population densities (finding a mate, larval exploitation of the resource). The efficiency to find an odor plume and the ability to track the plume to its source thus affect the reproductive success of the fruit fly population. In this study we model the response of *Drosophila melanogaster* to odor plumes containing food odors and their aggregation pheromone.

Odor plumes form as wind disperses odor molecules from their source. Their structure is complex. At large scale it exhibits a sinuous pattern as it moves with the wind. At a smaller scale the plume is intermittent [2]: patches with odors are interspersed with regions of clean air. In this study we compare a few plume models: one that takes features of a real odor plume into account with a meandering plume and with a simplified (time-averaged) model (see also [3]). We assess how these different models affect the spatial distribution of the fruit fly population and the rate of settlement on the resource.

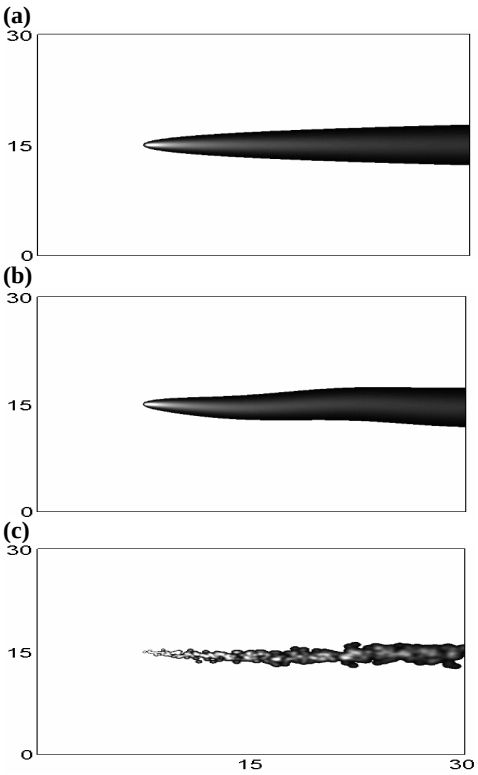

figure 1: the odor source lies at (7.5, 15) in (a) a time-averaged Gaussian plume is visualized, in (b) a meandering plume, and in (c) a filamentous plume.

References

[1] Dicke, M., Sabelis, M.W., 1988. Infochemical terminology: based on cost benefit analysis rather than origin of compounds? Funct. Ecol. 2: 131-139

[2] Farrell, J.A., Murlis, J., Long, X., Li, W., Cardé, R.T., 2002. Filament based atmospheric dispersion model to achieve short time-scale structure of odor plumes. Environ. Fluid Mech. 2: 143-169.

[3] Lof M., Hemerik L. and M. de Gee, 2007 Chemical communication: does odor plume shape matter? Proceedings of the Netherlands Entomological Society meeting, 61-70

Dengue: Minimum Parameters and the Endemic State

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Keywords: Mathematical Epidemiology, Stochastic Models, Endemic State.

Dengue fever is a viral disease transmitted by the *Aedes aegypti* mosquitoes. Dengue has been endemic in Brazil for more then 20 years and is one of the major public health problems [1], [2], [3]. In this work we build an stochastic automata model to simulate the dengue transmission dynamics in a population. It is a spatial model that couples vector and human dynamics. Individuals and mosquitoes are individually monitored. Mosquitoes live in a specific human residence and blood feed according to certain spatial distribution. Human population is not age-structured. Human mobility is taken into consideration and determines the central epoch and the duration of the epidemics outbreak . The main parameters of the model are: the human/vector population ratio, the human renewal rates, the mosquitoes biting rate, the mosquitoes biting spatial distribution, the contamination probabilities and the mosquitoes life-tables. The parameters used for the simulations were taking from field data (when available) and from the literature [4]. Two biting spatial distributions were considered. They are based in the observed fact that Aedes aegypti bites preferentially in the same place.

The main goal of this work it to determine the minimum human population size and human renewal rates that are capable of maintaining the endemic state. To this goal, human population size and renewal human rates were varied so to detect under which conditions the endemic state is possible. 100 simulations were performed for each set of parameters. The endemic state is said viable if in 90% on the cases the epidemics lasts more then 18 months. The results shows that under regular human renewal rates (around 3% per year) even very small communities can sustain the endemic state.

References

[1] Siqueira J.B. Jr., Martelli C.M., Coelho G.E., Simplicio A.C. e Hatch D.L., Dengue and dengue hemorrhagic fever, Brazil 1981-2002, *Emerging Infectious Diseases*, vol. 11, pp. 48–53 (2005)

[2] Marzochi K. B. F., Dengue endêmico: o desafio das estratégias de vigilância, *Revista da Sociedade Brasileira de Medicina Tropical*, vol. 37(5), pp. 413–415 (2004)

[3] Gubler D. J., Epidemic dengue/dengue hemorrhagic fever as a public health, social and economic problem in the 21st century, *TRENDS in Microbiology*, vol. 10(2), pp. 100–103 (2002)

[4] Luz P. M., Codeço C. T., Massad, E. Struchiner C. J., Uncertainties regarding dengue modeling in Rio de Janeiro, Brazil, *Memrias do Instituto Oswaldo Cruz*, vol. 98(7), pp. 871–878 (2003)

Influence of the fractal dimension of a fungal colonial area in its population dynamics

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Keywords: fractal, mathematical model, population dynamics, fungi.

Fungi are found in nature in several structures and places, being part in the production of food and medicines as well as in disease propagation. The filamentous fungal colony is consisted of a mycelium mass whose elements are hyphae. The colonial area increases through apical growth and hyphal networks that are also responsible for the production of spores, which will produce new fungal colonies.

The spores, when they find a suitable environment, begin to grow forming the mycelium that grows through the combination of two factors: the apical growth of hyphae, which makes possible the exploration of new areas in search of nutrients, and the ramification which makes possible the filling of these areas already reached. In favorable conditions, this growth of the colonial area occurs with a high grade of radial symmetry [1, 2].

Nevertheless, in heterogeneous environments, concerning about the distribution of nutrients that are necessary for the growth of the fungi, the morphology of the frontier of the fungal colony may be very irregular, for the quantity of the environmental nutrients is directly related to the ramification of the hyphae [3]. An immediate consequence of this irregularity, of the frontier of the colonies, is in the production of spores and, consequently in the population dynamic of the fungal colonies. The study about the growth of fungal colonies is of major importance in the propagation of foliar lesions caused by fungi, for example. In this case, the growth of the colony, besides intensifying the level of severity of the disease also contributes for the increase of the production of spores that disseminate the disease [4].

This assignment proposes a mathematical model that aims to describe the population dynamic of fungal colonies that develop in non-homogeneous environments, concerning about the distribution of nutrients, which consequently take irregular forms. The proposed model considers von Foersters equation [5] to describe the growth of colonial areas and to use the fractal dimension of the frontier of the colonies to characterize the appearing of new colonies. We considered that the production of spores depends on the fractal perimeter of the colonies. Numerical simulations were done, aiming to obtain qualitative information concerning about the population dynamic in fungal colonies.

References

[1] L. Edelstein, The propagation of fungal colonies: a model for tissue growth. J. Theor. Biology, 98, 679-701, 1982.

[2] F. Soddell, R. Seviour, J. Soddell, Using Lindenmayer systems to investigate how filamentous fungi may produce round colonies. Complexity International, v. 2, 1995.

[3] S. M. Parkinson, M. Wainwright, K. Killham, Observations on oligotrophic growth of fungi on silica gel. Mycological Research, 93, 529-534, 1989.

[4] R. D. Berger, A. Bergamin Filho, L Amorim, Lesion Expansion as an epidemic component. The American Phytopathological Society V. 87, n. 10, 1005-1013, 1997.

[5] N. Shigesada, K. Kawasaki, Y. Takeda, Biological Invasions: Theory and Pratice. Oxford Series in Ecology and Evolution. Oxford University, 1997.

Qualitative Study of Transmission Dynamics of Antibiotic-resistant Malaria

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Keywords: Malaria - Antibiotic - Resistant

Malaria is a mosquito-borne disease caused by protozoa of the genus plasmodium. Four species of malaria parasites can infect humans under natural conditions: P. *falciparum*, P. *vivax*, P. *ovale* and P. *malariae*. The first two species cause the most infections worldwide. P. *falciparum* is the agent of severe, potentially fatal malaria. The parasites are transmitted by the bite of infected female mosquitoes of the genus Anopheles.

Malaria is one of the most severe public health problems worldwide. It is estimated that between 350 and 500 million of clinical episodes of malaria occur every year with at least one million of deaths.

Treatment and control have become more difficult in recent years with the emergence and spread of drug resistant strains of malaria parasites. Since early 60s the sensitivity of the parasites to chloroquine, the best and most widely used drug for treating malaria, has been on the decline. Newer antimalarials were discovered in an effort to tackle this problem, but all these drugs are either expensive or have undesirable side effects. Moreover, after a variable length of time, the parasites, especially the *falciparum* species, have started showing resistance to these drugs.

In this work we present a deterministic model for exploring the evolution and transmission of drug-resistant malaria in a given population.

The model is based on the classical Ross-McDonald model for the spread of malaria. Here, the human and vector populations are divided into a number of mutually-exclusive compartments which differ according to treatment status and infection type (sensitive or resistant strains). We use qualitative analysis and numerical simulations to study the impact of various treatment scenarios on the emergence of resistance.

References

[1] P.B. Bioland, Drug resistance in malaria, World Health Organization, 2001.

[2] McDonald, G., The Epidemiology and Control of Malaria, Oxford University Press, London, 1957.

Consequences of increase the CO_2 atmospheric for plants' ecology

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Keywords: CO_2 atmospheric concentration, Photosynthesis Rate, Ecological Modelling.

The mathematical modelling of ecology started at the twenties, with the Lotka-Volterra prey-predator models and to describe competion between predators. These models were adapted to include spatial variation and to vegetable ecology [1]. In this way, we consider that increase atmospheric CO_2 concentration can change the variables related to plant's reproduction, leading to a different arrangement between species.

For two species, the hierarchical model is given by the following equation system:

$$\frac{dY}{dt} = \lambda_y Y(1-Y) - Y \tag{1}$$

$$\frac{dZ}{dt} = \lambda_z Z(1-Y-Z) - Z - \lambda_y YZ \tag{2}$$

where $Y(t)$ or $Z(t)$ is the the specie's density at time t.

Y propagates by contact, so it is $\lambda_y > 1$ (at mean field theory). Z also has the contact process, but it's space can be filled by Y's prole, if there is. Y is called superior competitor, and Z is called colonizer specie. The condition to Z coexist with Y is

$$\lambda_z Z \geq (\lambda_y Y)^2 \tag{3}$$

, that is, the birth rate to Z species must be bigger than the square birth rate of Y.

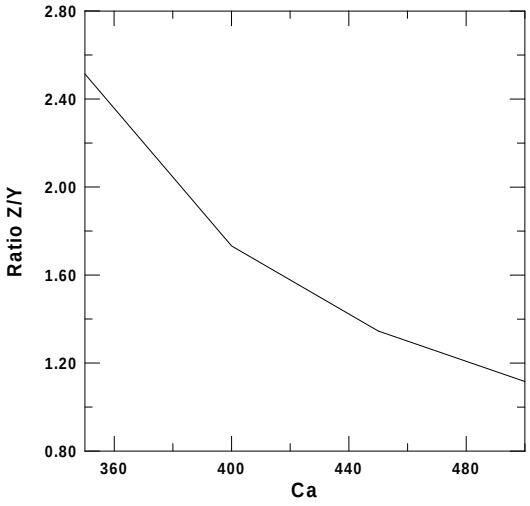
In the present work, the values λ_z and λ_y are proportional to the photosynthesis rate $A(Ca, T)$:

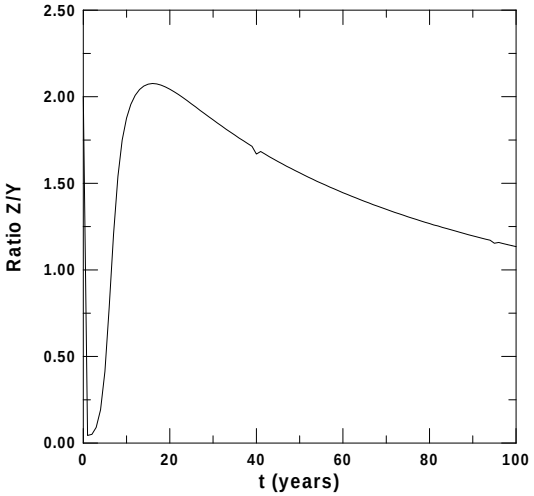
$$\lambda_{Y,Z} = c_{Y,Z} A(Ca, T) \tag{4}$$

where $c_{Y,Z}$ is a constant, Ca is the atmospheric carbon concentration (in parts per million, ppm) and T is the temperature ($T = 25^oC$), in this work. We use [2]

$$A(Ca, T = 25^oC) = \frac{Ca - 63}{Ca + 126} \tag{5}$$

. To understand the consequences of increase Ca, we worked many different cases, including some at edge of 3. The pictures in the following show the biomass ratio Z/Y for the case with constant value to Ca (on the top) and the time evolution of this ratio, if Ca is growing according the equation $Ca = 350 + 1.15t$, (below).





References

[1] C. Neuhauser e S. W. Pacala, Ann. Appl. Probab 9 1226-1259 (1999)

[2] M.U.F. Kirshbaum , Plant, Cell and Environment., 17:747-754; 1994.

Allee Effects in the Pattern Formation in Prey-Predator Systems

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Keywords: Allee effects, pattern formation, coupled map lattices.

Many observational data show that some species present an increase in the per capita growth rate at low densities, what is called Allee effect. In the context of difference equations, a strong Allee effect occurs when there is a positive equilibrium density such that the per capita growth rate is less than one for densities lower than this equilibrium and is greater than one for densities greater than it. That is, a population must overcome this threshold to grow. On the other hand, a population with weak Allee effect does not have a threshold, the per capita growth rate reaches its maximum at an intermediate population size and is greater than one when individuals are rare.

In this work, we have constructed a discrete model in order to analyze heterogeneous pattern formation in prey-predator system with Allee effect dynamic in prey population. The model proposed uses Coupled Map Lattices, where the time and the space are discrete variables and the state variable is continuous.

The simulations developed indicated that, for strong Allee effect, fixed heterogeneous spatial patterns for both species occur when the prey population decay below the Allee threshold in some regions of the habitat. We have noted that the pattern generated was directly related to the perturbation imposed on prey distribution near the population homogeneous steady state. For weak Allee effect, heterogeneous pattern formation occurs according to the Turing diffusive instability process.

References

[1] J. D. Murray, Mathematical Biology, Springer-Verlag, Berlin, 2003.

[2] S. Schreiber, Allee effect, extinctions, and chaotic transients in simple population models. Theoretical Population Biology, 64, 201-209, 2003.

[3] L. A. Segel and S. A. Jackson, Dissipative structure: an explanation and an ecological example. Journal of Theoretical Biology, 37, 545-559, 1972.

[4] D. J. Seidel, Modelos Matemáticos para Formação de Padrões em Sistemas Biológicos. Dissertação de Mestrado, UNIJUÍ, 2006.

[5] A. M. Turing, The chemical basis of morphogenesis. Phil. Trans. Roy. Soc. Lond., 237, 37-72, 1952.

[6] M. H. Wang and M. Kot, Speeds of invasion in a model with strong or weak Allee effects. Mathematical Biosciences, 171, 83-97, 2001.

Mathematical Analysis of a Diffusive Predator-Prey System with Non-Local Intra-Specific Competition

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Keywords: Predator-Prey, Reaction-Diffusion, Non-locality, Traveling Waves.

The aim of this work is the study of the following diffusive predator-prey system of equations, which is an extension of the one studied in previous works ([1], [2]):

$$\frac{\partial U}{\partial T} = D_1 \frac{\partial^2 U}{\partial X^2} + AU\left[1 - \frac{1}{K}(g \star U)\right] - BUV$$
$$\frac{\partial V}{\partial T} = D_2 \frac{\partial^2 V}{\partial X^2} + CUV - DV,$$

where

$$g \star U = \int_{-\infty}^{\infty} g(x-y)U(y,t)\,dy. \qquad (1)$$

The constants D_1, D_2, A, K, B, C, and D, as well as the weight function g are all positive. This system models the dynamics of two interacting populations, where the first one, the prey, obeys a nonlocal logistic growth; and the second one, the predator, interacts with the prey, with exponencial decrease of its density in the absence of first species, which indicates that the predation of the second species is specific. We also assume that the predator diffuses more rapidly than the prey, a fact which simplifies the analysis. The key feature in these equations is the presence of the convolution term (1), which models a nonlocal intra-specific interaction among the prey individuals, just as it was indicated in [3]. After a suitable approximation of the first equation, we analyse the possibility of coexistence of these species, by investigating the conditions under which a travelling wave solution, which connects trivial steady states and a nontrivial one, exists. We obtain the existence of this solution for any combination of the constants, a fact which is not verified in [1], [2], where a minimal wave speed exists. Besides, the stabilization towards the nontrivial equilibrium allways occurs in a oscillationg pattern, which is in accordance with the results in [3], with the one-equation model, where a non-monotonic wave connectiong the steady states is obtained.

References

[1] S.R. Dunbar, Travelling wave solution of diffusive Lotka-Volterra equations. Journal of Mathematical Biology 17, 11-32, 1983.

[2] S.R. Dunbar, Travelling wave solutions of diffusive Lotka-Volterra equations: a heteroclinic connection in R^4. Transactions of the American Mathematical Society 268, 577-594, 1984.

[3] S. Gourley, Travelling front solutions of a nonlocal FIsher equation. Journal of Mathematical Biology 41, 272-284, 2000.

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Predator-prey systems subject to threshold policy with hysteresis

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Keywords: predator-prey system; threshold policy with hysteresis

This paper introduces a *threshold policy with hysteresis* (TPH) for the control of predator-prey models of one and two species. The models studied are the single species Noy-Meir model, as well as the Lotka–Volterra and Rosenzweig–MacArthur two species density-dependent predator-prey models. The proposed policy (TPH) changes the dynamics of the system in such a way that a bounded oscillation is achieved. The policy can be designed by a suitable choice of so called virtual equilibrium points in a simple and intuitive manner.

In the engineering control literature, TPH has been shown to change the dynamics of a system to which it is applied in such a way that a bounded oscillation (limit cycle) can be achieved [1]. In the context of ecology stabilizes a system at a point is a rather nonrealistic goal, this being the main motivation for the proposal of the threshold management policy with hysteresis that changes the dynamics of the system in such a way that a bounded oscillation is achieved.

Mathematical definitions of threshold policy with hysteresis

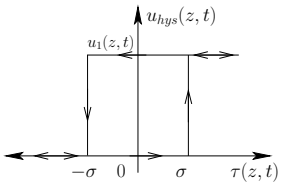

Figure 1: Graphical representation of the threshold policy with hysteresis, where z is the state vector, τ is the threshold variable that should be chosen adequately, depending on the problem to be solved and $\sigma > 0$ is the hysteresis parameter.

In the context of real systems, there is one important assumption that makes the threshold policy used in [2] a little unrealistic: namely that as soon as the system crosses a threshold, the mode of control changes instantaneously. This allows the model to closely follow the threshold (sliding mode), and reach a stable equilibrium (sliding equilibrium). In practice, it is likely that the threshold from the region with control towards the region without control has a different position than the threshold from the region without control towards the region with control. In control language, this means that we should consider hysteresis in the threshold, see Figure 1.

Predator-prey two species Rosenzweig–MacArthur model subject to a TPH

In this section is shown that the proposed approach is successful in the control of the classical Rosenzweig–MacArthur predator-prey model. We choose this model because it can be regarded as the simplest nontrivial paradigm that was proposed after the more classical but biologically unrealistic Lotka-Volterra model. We have outlined how the approach being formalized in this paper and it can also be used to control the Lotka-Volterra model

In managing renewable resources stabilizing a system at a point is a rather nonrealistic goal, this being the main motivation for the proposal of the threshold policy with hysteresis that changes the dynamics of the system in such a way that a bounded oscillation is achieved, i.e. the system stabilizes in a limit cycle. It must be stressed that when it is considered a threshold control with hysteresis effect, we are considered errors and delays in the implementation of the policy, i.e. it is considered errors and delays in the measurement of the species density.

References

[1] A. A. Andronov, A. A. Vitt, and S. E. Khaikin, *The Theory of Oscillators*. UK: Pergamon Press, 1966.

[2] M. E. M. Meza, A. Bhaya, E. Kaszkurewicz, and M. I. S. Costa, "Threshold policies control for predator-prey systems using a control Liapunov function approach," *Theoretical Population Biology*, vol. 67, no. 4, pp. 273–284, 2005.

The deterministically chaotic dynamics in a realistic multi-strain dengue model with temporary cross-immunity.

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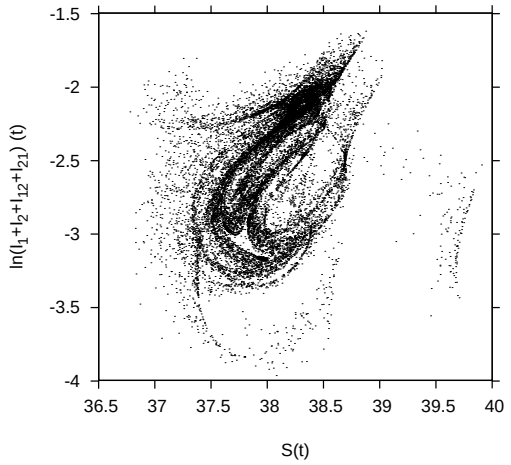
Keywords: Dengue, Antibody-dependent Enhancement, Cross-immunity, Chaos.



The map of maxima of the overall infected and the respective values for the susceptibles.

Dengue is a mosquito-borne infection which in recent years has become a major international public health concern. Two fifths of the world's population is at risk from dengue [1].

Two forms of the disease exist: classic dengue, often benign, and hemorrhagic dengue, which is more severe, often fatal. [2, 3].

The infective agent is the Dengue virus of the family Flaviviridae. Four serotypes have been distinguished, type 1, 2, 3, and 4. Infection by one serotype confers life-long immunity to only that serotype. Only temporary cross-immunity to other serotypes exists [4].

Mathematical models describing the transmission of dengue viruses appeared focusing on different aspects, simulating the abundance of the mosquito vector and its effect upon transmission [5, 6, 7]. Or in multi-strain dynamics, chaos was found when the antibody-dependent enhancement (ADE) was included [8, 9, 10].

In this work, we investigate a more realistic multi-strain dengue model, including the temporary cross-immunity, and describe the dynamic behavior from a fixed point via limit cycles to chaotic attractors.

References

[1] Word Health Organization. (2002) *Dengue and Dengue Hemorrhagic Fever*. (World Health Org., Geneva, Fact Sheet **117**).

[2] Halstead, S. B. (1982) Immune enhancement of viral infection. Progress in Allergy. *Progress in Allergy*. **31**, 301–64.

[3] Halstead S. B. (2003) Neutralization and antibody-dependent enhancement of dengue viruses. *Advances in Virus Research*. **60**, 421–67.

[4] Esteva, L., & Vargas, C. (2003) Coexistence of different serotypes of dengue virus. *Math. Biosci*. **46**, 31-47.

[5] Esteva, L., & Vargas, C. (1998) Analysis of a dengue disease transmission model. *Math. Biosci*. **150**, 131–151.

[6] Focks, D. A. et al. (2000) Transmission Threshold For Dengue In Terms Of Aedes aegypti Pupae Per Person With Discussion Of Their Utility In Source Reduction Efforts. *Am. J. Trop. Med. Hyg*. **62**, 11–18.

[7] Twiddy, S. S. et al. (2003) Comparative population dynamics of mosquito-borne falviviruses. *Infect. Genet. Evol*. **3**, 87–95.

[8] Ferguson, N. et al. (1999) The effect of antibody-dependent enhancement on the transmission dynamics and persistence of multiple-strain pathogens. *Proc. Natl. Acad. Sci*. **96**, 790–94.

[9] Cummings, D. A. T. et al. (2005) Dynamic effect of antibody-dependent enhancement on the fitness of viruses. *PNAS*. **102**, 15259–64.

[10] Adams, B., & Boots, M. (2006) Modelling the relationship between antibody-dependent enhancement and immunological distance with application to dengue. *J. Theor. Biol*. **242**, 337–46.

Optimal Control Strategy of Malaria Vector Using Genetically Modified Mosquitoes

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Keywords: mathematical modeling, malaria, transgenic mosquitoes.

Malaria is a tropical infectious disease, transmitted from infected person to susceptible one through *Anopheles* female bite. Scientists of several countries are trying to create an effective vaccine to prevent malaria, but it has been difficult. Now, it has been suggested, and there are some research initiatives in this direction that genetically modified mosquitoes introduced in the environment can exterminate malaria once for all [1]-[3]. These transgenic mosquitoes carry a specific code that inhibits the plasmodium evolution in his organism. It is said that this characteristics is hereditary and consequently the disease fades away after some time. The difficulty in this case is that the introduction of transgenic species in the environment is very questionable, particularly for this kind of genetic manipulation. However, as indicated by mathematical modeling, if it would be possible to increase the latency period of the infected mosquitoes, the treatment efficiency could be much higher. Maybe a slight genetic manipulation could be enough to introduce this characteristics or even a properly induced selection process.

The mathematical model considered in [4] applies to the Brazilian Amazon region, where the seasonal fluctuation of the mosquito density is clearly observed. The main vector of this region is the *Anopheles darlingi*, and its latent period is directly linked to the environment temperature [5].

In this work the vector control was formulated and solved as an optimal control problem indicating how the genetically modified mosquitoes should be introduced in the environment. The numerical simulations show the effectiveness of the proposed control.

References

[1] F. Catteruccia, H. C. J. Godfray, A. Crisanti,. Impact of genetic manipulation on the fitness of *Anopheles stephensi* mosquitoes. *Science* 299, 1225–1227, 2003.

[2] L. A. Moreira, A. K. Ghosh, E. G. Abraham, M. Jacobs-Lorena. Genetic transformation of mosquitoes: a quest for malaria control. *International Journal for Parasitology* 32, 1599–1605, 2002.

[3] A. C. Pinkerton, K. Michel, D. A. O'Brochta, P. W. Atkinson. Green fluorescent protein as a genetic marker in transgenic *Aedes aegypti*. *Insect Mol.Biol.* 9, 1–10, 2000.

[4] A.P.P. Wyse, L. Bevilacqua, M. Rafikov. Simulating Malaria Model for Different Treatment Intensities in a Variable Environment, Ecological Modelling (In press)

[5] W. Tadei. Entomologia da malária em áreas de colonização da Amazônia. Chapter 4, pp. 157–168, MCT, PPD–Programa de Pesquisa Dirigida, 1997.

Aedes aegypti dispersal. A survival strategy?

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Keywords: Mathematical Ecology, Stochastic dynamics, Aedes aegypti.

Aedes aegypti is the main vector for dengue fever and urban yellow fever. It is extended around the world not only in tropical regions but also beyond them, reaching temperate climates zones such as Buenos Aires city. In this work the biology and environment of Aedes aegypti is incorporated into the framework of a stochastic spatial population dynamical model able to describe seasonal variations of the populations, adult dispersal and local and total population extinctions1. The evolution of the immature and adult mosquito populations is modeled by a state dependent Poisson Process whose rates depend on time through seasonal temperature variations and that incorporates the spatiality through an explicit spatial model. In order to design a realistic dispersal model in a city with temperate climate such as Buenos Aires city, many factors have been taken into account such as the biology of the mosquito, the temperature seasonal variations, the spatial heterogeneity of the environment represented by the availability of breeding sites and of course the dispersal of female adult mosquito populations, which is supposed to be driven by the availability of oviposition sites. The model is able to deal with the mosquito dispersal and can be used to estimate the amount of effective breeding sites of an homogeneous place from the fraction of positive ovitraps in that region, obtained as a result of a surveillance study without altering the environment and without perturbation of the mosquito populations. Our validation corresponds to a monitoring study of mosquito egg-traps (ovitraps) performed in Mataderos quarter of Buenos Aires city between July 2001 and July 2002. We estimated the density of breeding sites in Mataderos as 20 BS/ha. With a previous homogeneous model2 without dispersal we show that this density of breeding sites is not enough to sustain the populations of Aedes aegypti. We analyzed the mosquito dispersal as an advantageous strategy of persistence in Buenos Aires city and simulated the dispersion of females from a source to the surroundings along a three years period, observing that several dynamical processes occur simultaneously such as local extinctions, diffusion-like recolonization processes (resulting from the flight and the oviposition performed by flying females) and local colonization processes resulting from the emerged adults born from survival remaining eggs during the Winter season.

References

[1] M. Otero, N. Schweigmann, H. Solari. A stochastic spatial dynamical model for Aedes aegypti. Submitted to the Bulletin of Mathematical Biology.

[2] M. Otero, H. G. Solari, and N. Schweigmann. A stochastic population dynamics model for Aedes aegypti: formulation and application to a city with temperate climate. Bulletin of Mathematical Biology 68 (8), 1945-1974, 2006.

A Tinnitus´ Models Using McKendrick´s Equations.

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Keywords: Tinnitus, McKendrick´s Equations, Numerical Analysis.

Tinnitus can be defined as an auditive sensation that does not come from external sources. According to north-american researches about 15% of population has tinnitus, with severe impairment in about 20% of the patients. Although tinnitus´ physiopatology is not completely understood, it is believed that initial damage occurs at the cochlea, with later development to the central auditory pathways. The main known causes of tinnitus include professional noise exposure, aging, methabolic and ischemic diseases. Based on that, we propose, in this article, a model based in Mckendrick´s equations to calculate the probability for workers with ages [0,x] and exposed to intense noise during a spam of time [0,T]. it is taken two states, the first one is for assintomatic patients and second for those who have tinittus. The states´ transitions are made by treatment and illness rates. In order to do that we use the McKendrick´s equations including a term of an unexpected event (a more intense noise, metabolic descompensantion, use of medications) that has started in a given time t° was given by convolution. A numerical method is presented as solution for some systems as the above, based in Euler Iterative Method.[2]

References

[1] A.A. Azevedo, R.R.Figueiredo, Atualização em Zumbido . Rev. Brasileira Otorrino. 70, 27-40, 2004

[2] M.O. Pinho, A.C.M Alvim, J.L. Fernandes, P.F.F.Frutuoso e Melo , Discussion on the Convergence of Euler's Iterative Method for Calculating Failure Probabilities for a System Modeled by Supplementary Variables, Annals of EMC-2005,2005

VARIABLE INTERACTIONS BETWEEN FRESHWATER AMPHIPODS AND ALGAE

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Keywords: Ecology

The *Hyalella* complex is a group of crustacean found in all the plain streams and ponds from the American continent. In particular, in the Pampasic region of Argentina, *Hyalella curvispina* and *H. pseudoazteca* are the two most frequent species.
These two species form a stable ecological set with microalgaes of the phytobentos and periphyton (microscopic algae stick to solid substrata and submersed plants respectively). The more common genera of submersed plants in this region are *Myriophyllum*, *Ceratophyllum* and *Egeria*. They are important as structuring acuatic habitats. The macrophytes constitute an importan refuge and, at the same time, a large substratum where the algae can growth. Amphipods eat mainly algae biofilms.
However, it was found that *H.curvispina* can become a predator of *H. pseudoazteca* when algae are scarce. Is this system dinamically stable? To answer this question in a general form we propose a model to study the behavior of the system under different conditions and values of the parameters involved in the model . We considerer direct effects, as predation and competition, and indirect effect as refuge or support. The existence of refuges can be modeled with a strong Allee effect that “kidnapp” from predation a fix number of prey, and the effect of feeding with a function reporting the relation between the available biomass of algaes and macrophytes. The empirical evidence shows that even in those brooks where sudden flow growth sweep away periodically the community of macrophytes, the system is restablished, recolonizing the backwaters. These anphipods play a relevant role in the ecosystem of fresh water linking the energy of algae with the fish stock.

Regions of endemicity for disease in metapopulations

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Keywords: metapopulation

A simple stochastic metapopulation model proposed by Ezanno et al [1] couples the spread of infectious disease within a subpopulation to the dynamics of transmission between subpopulations. The dynamic behaviour of the model is not yet fully understood. One feature of the model is that the duration of an outbreak that begins with a single infectious individual has a strongly non-linear relationship with migration rate. For very high migration rates the model exhibits endemic behaviour, i.e. the disease persists for at least 10 years in the population, but there is also a peak in outbreak length for relatively low migration rates. Questions arising are for which regions of parameter space endemic behaviour occurs and whether this can be explained by synchronous disease dynamics in the subpopulations. One of the model assumptions is that individuals migrate randomly from one patch to any other patch, i.e. it is a fully connected network. This assumption can be replaced by using more realistic networks that reflect the migration patterns typical of wildlife or the movement patterns observed for livestock. Hence this model can be used, for example, to understand the dynamics of sylvatic plague in great gerbil populations in central Asia or to model the dynamics of disease transmission between and within different farms.

References

[1] P. Ezanno, S. Davis, J.A.P. Heesterbeek, Epidemics in a metapopulation: coupling within- and between-patch dynamics (submitted)

Towards understanding Natural Killer cell development in the bone marrow: combining experiments and mathematical modeling

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Keywords: Cell populations, immunology, NK cell development, model fitting.

Recent studies have described different in vivo stages of Natural Killer (NK) cell maturation in the bone marrow [1, 2]. However, a solid description of the basic population dynamics of NK cell development is still lacking. It is not clear how processes as differentiation, proliferation, and cell death shape the distribution of NK cells over the different maturation stages. Furthermore, the exact developmental pathway the cells follow has not been clarified yet. It is impossible to obtain complete kinetic information of NK cell maturation using only experimental tools; a combination with mathematical modeling is indispensable. In this project, the distribution of NK cells over the different stages and the acquisition of BrdU-label, incorporated in cells that have divided during the label administration, were determined experimentally. These data were then used to test different mathematical models and comparison of the results gave insight in which of the models is more likely to describe the developmental path. Furthermore, differentiation, proliferation and death rates were estimated for each of the stages. This resulted in a better understanding of the function the different developmental stages play in the maturation of NK cells. A solid description of the basic population dynamics of NK development will enable us to understand the shaping of the repertoire, help us specify important stages in NK cell selection and evaluate the effect of disease on the development of NK cells. Results will be discussed with respect to this aim.

References

[1] [1]J.P. DiSanto (2006). Natural killer cell developmental pathways: A question of balance. Ann. Rev. Imm. 24:257.

[2] S. Kim et al. (2002). In vivo developmental stages in murine natural killer cell maturation. Nature immunology 3:523.

Modeling the effect of chemical information and resources abundance on colonization of an orchard by Drosophila melanogaster

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Keywords: Chemical information, colonization, spatial population dynamics, Drosophila melanogaster.

Chemical communication is widely used in insects. Chemical information can hold cues on the availability of food and mates, as well as the presence of competitors and natural enemies. To study the effect of the use of infochemicals on spatial population dynamics, we chose Drosophila melanogaster as a model organism. D. melanogaster uses its aggregation pheromone and food odors to aggregate at suitable resources to avoid the negative effects of low population densities (finding a mate, larval exploitation of the resource). However, forming aggregations can also have costs, at high local larval densities there is an increased risk of competition for resources.

In a previous model study we found that infochemicals mainly have a positive effect on population numbers and persistence during the spatial colonization of an area, starting from the center of the domain [1]. After the spatial expansion, however, the negative effects of increased mortality due to competition were higher when fruit flies could use chemical information, resulting in a negative effect of the infochemicals on the population numbers. Thus the use of infochemicals is mainly beneficial for the fruit flies during the colonization of an area. Drosophila melanogaster cannot survive the winter in the field in the natural climate of the Netherlands. Therefore, they have to re-colonize the orchards from the boundaries in spring. In this situation, determining the position of the resources is of vital importance, and here the guidance of the fruit flies by infochemical information can play a significant role.

We developed a spatio-temporal model that incorporates odor dispersion and population responses, to study to what extent the presence of infochemicals, specifically food odors and aggregation pheromone, affects the ability of a fruit fly population to colonize an orchard from the boundaries, when resources are scarce or abundant. We investigate the costs and benefits of the use of infochemicals on population numbers, by looking at larval mortality due to Allee effect or competition and the net effect on population numbers.

References

[1] M. Lof, R. Etienne, J. Powell, M. de Gee, L. Hemerik, The effect of chemical information on the spatial distribution of fruit flies: I Model results, Bulletin of Mathematical Biology, submitted.

Size-Structured Modelling of Aquatic Ecosystems

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Keywords: body size, spectrum, life history, bioenergetic, food web, ecosystem

We present a mechanistic multi-species size-structured dynamic modelling framework for more realistic modelling of fish populations based on an individual-level bioenergetic growth model. The model is defined as a simple model where only one parameter, asymptotic body mass M, is used to characterise a species.

Each species population density spectrum (numbers per volume per mass) N_i is modelled by modelling individual growth from offspring size m_0 to the asymptotic size M. Growth $g(m)$ is obtained by eating individuals of an appropriate size in the community spectrum N_c, which is the sum of all species spectra N_i and the background spectrum N_b that contains the basal resources (plankton) in the ecosystem. The predation intake of all individuals in the community returns the mortality $\mu(m)$ that each m sized individual experiences. The dynamics of the species spectrum is obtained via g and μ by solving the McKendrick-von Foerster equation $\partial N_i/\partial t + \partial/\partial m[\, gN_i\,] = -\mu N_i$, and the dynamics of the ecosystem is obtained by the coupling of such PDEs (and the ODEs describing the resources). Fig. 1 illustrates the components of the model. Reproduction enters the system as a boundary condition on the PDEs at m_0, where a flux of offspring is routed to from the spawning stock of the species.

The model is conceptually similar to [1, 2], with the addition of an individual-level functional response, a background spectrum, and further specification of reproduction and energy allocation. [1] can be seen as steady-state solutions to the presented model.

We will use the model to i.e. demonstrate coexistence in an aquatic environment where any individual is capable of eating any other individual if it is of an appropriate size. A mechanism termed the *trophic ladder* is used to explain coexistence among different sized species by allowing larger species to exist only by using smaller species as a ladder to reach a higher position in the trophic hierarchy. Bistability plays an important role for the existence of trophic ladder states. The trophic ladder may play an important role in the assembly and composition of size-structured food webs, but cannot alone account for high biodiversity. Having additional species in between the steps of the ladder are possible, but actual food web configurations remains to be of importance.

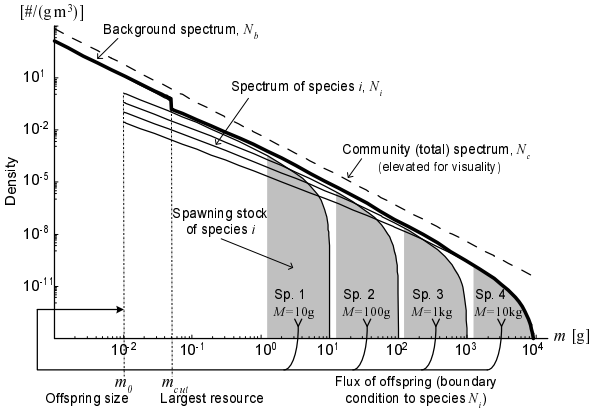

Figure 1: Ideal steady-state illustration of the different components in the ecosystem model with the example of four species having $M = 10,\ 100,\ 1.000$ and 10.000 g.

Our goal is to use the presented framework as a general framework for size-structured food webs as the Yodzis and Innes model [3] in classical food webs.

References

[1] K. H. Andersen and J. E. Beyer. Asymptotic size determines species abundance in the marine size spectrum. The American Naturalist, 168(1):54-61, 2006.

[2] K. H. Andersen, J. E. Beyer, M. Pedersen, N. G. Andersen, and H. Gislason. Life history constraints on asymptotic size in marine ecosystems. Submitted, 2007.

[3] P. Yodzis and S. Innes. Body Size and Consumer-Resource Dynamics. The American Naturalist, 139(6):1151-1175, 1992.

Mathematical modelling of cell population dynamics in the colonic crypt and in colorectal cancer

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Keywords: age-structure, feedback, mutations, structural stability

Colorectal cancer is initiated in colonic crypts. A succession of genetic mutations or epigenetic changes can lead to homeostasis in the crypt being overcome, and subsequent unbounded growth.

We examine the dynamics of a single colorectal crypt based on the model by Tomlinson and Bodmer [1], which accounts for populations of stem cells, differentiated cells, and transit cells. That original model made the simplifying assumption that each cell population divides synchronously, but the model we present relaxes this assumption by adopting an age-structured approach that models asynchronous cell division, and we compare this to a continuum model.

We present two feedback mechanisms that could regulate the growth of cell numbers. The first maintains the homeostasis that is normally observed in the crypt, and the second can be used to explain the long lag phases in tumour growth, where new, higher equilibria are reached, before unlimited growth in cell numbers ensues.

Results show that an increase in cell renewal, which is equivalent to a failure of programmed cell death or of differentiation, can lead to the growth of cancers.

References

[1] Tomlinson, I. P. M. & Bodmer, W. F. *Proc. Natl. Acad. Sci. USA* **92**, 11130-11134 (1995).

[2] Johnston, M. D., Edwards, C. M., Bodmer, W. F., Maini, P.K. & Chapman, S. J. *Proc. Natl. Acad. Sci. USA* **104** 4008-4013 (2007).

Modelling transmission of Toxoplasma sp. in Amazonia

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Keywords: predator-prey system, transmission by predation, indirect transmission, SIR type model, Human contamination, toxoplasma gondii.

Toxoplasma gondii was first observed in 1908 by C. Nicolle and L. Manceaux in a rodent population, *Ctenodactylus gundi*, (Tunis) and by A. Splendore in rabbits (Sao Paulo). A large literature has been devoted to the understanding, study and modelling of this parasite since. To summarize it is generally acknowledged, cf. [4], that its propagation goes through a multispecies system involving mammal and bird species as intermediate hosts and domestic cats as final hosts. Parasite sexual reproduction takes place within the final host and then oocysts are released in the environment by infected hosts through their feces, a free stage. Transmission to intermediate hosts occurs through contaminated food – vegetal – and water while contamination of final hosts is related to predation. Human contamination occurs through contaminated vegetal, water and raw or undercooked meat. This poses severe health problems to offspring of infected pregnant women, while it is benign for other individuals.

Later in the XX century several evidences were found of the likely existence of a wild cycle of Toxoplasma sp. – as opposed to the previous domestic cycle – closely related to Toxoplasma gondii. Among others, some native populations living in French Guiana without any contact with domestic cats proved to have a prevalence identical to that of European populations, cf. [3]. There it poses severe health problems to offspring of infected pregnant women but also to HIV infected individuals and immunocompetent individuals, cf. [2].
The goal of this talk is to present a deterministic mathematical model for the propagation of Toxoplasma sp. in this tropical environment. Final host populations are wild feline predators feeding upon wild prey playing the role of intermediate hosts; compared to the domestic cycle these predators have no other food supply than wild prey. Parasite transmission in the prey-predator/Human system occurs through contaminated food – vegetal, prey – and water. Contamination of the environment is caused by excrements of final hosts.
Ignoring for the time being any spatial or any other structuring variable this leads to a multi-component ODEs system whose state variables are prey, predator and Human species, and the environment. It is derived upon using standard functional responses to predation to model, (1), interspecific interactions between prey and both predators and humans, and, (2) to model efficient contacts allowing parasite transmission through the prey-predator/Human system. Indirect contamination through the environment follows a methodology from [1].
Introducing simplifying assumptions concerning, (1), the impact of the virus on either or both prey and predator populations, and, (2) the impact of predation on the prey population, allow to reduce the original system to a simpler one. Then some stability analysis can be performed. Continuity arguments will support numerical simulations in more generic cases.

This work is supported by Programme CNRS Amazonie, *Théories, modélisation et simulation des écosystèmes amazoniens.*

References

[1] K. Berthier, M. Langlais, P. Auger et D. Pontier. Dynamics of a feline virus with two transmission modes within exponentially growing host populations. Proc. R. Soc. London, series B, 267, 2049-2056, 2000.

[2] B. Carme, F. Bissuel, D. Ajzenberg, R. Bouyne, C. Aznar, M. Demar, S. Bichat, D. Louvel, A.M. Bourbigot, C. Peneau, P. Neron, M.L. Darde. Severe acquired toxoplasmosis in immunocompetent adult patients in French Guiana. J. Clin. Microbiol. 40, 4037-4044, 2003.

[3] A. Fribourg-Blanc, E. Bois et J. Feingold. Bilan épidémiologique des Amérindiens de Haute-Guyane Franaise : Sérologie de la Toxoplasmose. Médecine et Maladies infectieuses. 5(10), 502-507, 1975.

[4] J. P. Webster, Rats, cats, people and parasites: the impact of latent toxoplasmosis on behaviour. Microbes and infection, 3, 1037-1045, 2001.

Analytical models of forest dynamics and tree species coexistence: from field data to theory

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Keywords: individual based models, mechanistic theory, scaling.

The identification of the mechanisms underlying forest dynamics and community assembly is a central goal in ecology. Individual-based models (IBM) of forest succession typically track the life cycle of each tree and simulate the feedback between resource depletion and tree performance. IBM can be parameterized from field data and have been shown to provide critical insights on the mechanismsmaintaining biodiversity [1]. Despite of this, lack of a proper analytical framework to understand the predictions of IBM has hindered the development of a mechanistic (resource based) theory of forest dynamics. Also gathering of field data for each species can be costly and time consuming. In this contribution we formulate a two tree species IBM and apply a general method to derive a partial differential equations (PDE) representing the limiting behaviour of the IBM [2]. We then solve numerically the model [3, 4] to identify conditions for coexistence among the two species. Finally a maximum likelihood based parameterization from available forest inventory data is shown.

References

[1] Pacala, S.W., Canham, C.D., Saponara, J., Silander, J.A., Kobe, R.K. & Ribbens, E., 1996. Forest models defined by field measurements: estimation, error analysis and dynamics. Ecol. Monogr. 66: 143.

[2] Gómez-Mourelo, P., 2005. From individual-based models to partial differential equations. An application to the upstream movement of elvers, Ecol. Model. 188: 93-111.

[3] Angulo, O. & López-Marcos, J.C., 1999. Numerical schemes for sizestructured population equations. Math. Biosci. 157: 169188.

[4] Zavala, M.A., Angulo, O., Bravo, R. & J.C. López-Marcos. 2007. An analytical model of stand dynamics as a function of tree growth, mortality and recruitment: the shade tolerance-stand structure hypothesis revisited. J. Theor. Biol. 244: 440450.

The study of the viability domain for a fishing problem with reserve

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Keywords: Maritime reserves, domain of viability.

The approach followed in this paper is completely different from the objectives mentioned in previous work [3, 4, 5], for examples, the research of the optimal strategy and the study of the asymptotical stability of the equilibrium point of the model. Because we want that the final result of work interests the fishermen and the ecologists in the way that it is applicable and satisfies simultaneously their economic and ecological interests.

Thus, the aim of our approach is to find a viability domain which verifies two different constraints using the viability theory. The first constraint is ecological: guarantying the stocks perenniality for protecting the environment, the second constraint is economic: guarantying a minimal income for the fishermen. These results will allow the decision-makers and the managers to start reflecting on others different ways than that used now like the application of the quota.

References

[1] D. Ami, P. Cartigny and A. Rapaport. Can marine protected areas enhance both economic and biological situations? C. R. Biologies 328(2005) 357-366.

[2] C. W. Clark. Mathematical Bioeconomics: The optimal Management of Renewable Ressources, second ed., John Wiley and Sons, New York, 1990.

[3] M. Jerry and N. Raïssi. A policy of fisheries management based on continuous fishing effort, Journal of Biological System, Vol. 9, No. 4, 2001, pp. 247-254.

[4] M. Jerry and N. Raïssi. The optimal strategy for a bioeconomical model of harvesting renewable resource problem, Mathematical and Computer Modeling, 36, 2002, pp. 1293-1306.

[5] M. Jerry and N. Raïssi. Optimal Strategy of a fishing problem based on a structured model, Comptes Rendus Biologies, Vol. 328, No. 4, 2005, pp 351-356.

Robust feedback control design for a nonlinear wastewater treatement model

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Keywords: wastewater treatement, stability, robust control, observer, dynamical systems.

In this work we study a model of wastewater treatment formulated by a nonlinear ordinary differential system. The model describes an activated sludge process in which the substrate is removed by one suspended bacterial population. The main biological reaction in the aeration holds. Three phenomenas are considered: The reaction kinetics in the aerator linked to microbial growth, The substrate degradation and the recycle of the biomass from the settler. The basic model is developed in

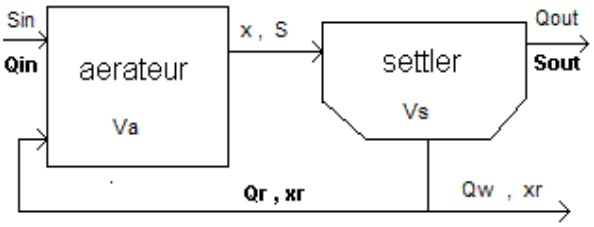

Figure 1: Diagram of the activated sludge process.

litterature where various of aspects are studied (observers [1], ...). The mass balance of the various constituents gives the following set of equations:

$$(S) \quad \begin{cases} \dot{s} = -\dfrac{\mu(.)x}{Y} - (1+r(t))Ds + Ds_{in}(.) \\ \dot{x} = \mu(.)x - (1+r(t))Dx + r(t)Dx_r \\ \dot{x}_r = \nu(1+r(t))Dx - \nu(w+r(t))Dx_r \\ s(0) = s_0 \ ; \ x(0) = x_0 \ ; \ x_r(0) = x_{r0}; \end{cases}$$

with

$$D = \frac{Q_{in}}{V_a}; \ \ r(t) = \frac{Q_r(t)}{Q_{in}}; \ \ w = \frac{Q_w}{Q_{in}}; \ \ \nu = \frac{V_a}{V_s}$$

where s, x and x_r are the states variables representing the substrate, the biomass and recycled biomass concentration, respectively. Q_{in}, Q_{out}, Q_r, Q_w are the influent, effluent, recycle and waste flow rates, respectively. V_a and V_s represent the aerator and settler volumes. $s_{in}(t)$ corresponds to the substrate concentrations in the feed stream. Y refer to the yield coefficient of the growth of biomass on substrate. $\mu(.)$ is the specific growth rate of the bactery.

Hypotheses :
We assume that $\mu(.)$ as well as $s_{in}(.)$ are not well known but there exists $s_{in}^{+}(t)$ and constant ξ^{+} such that :

$$s_{in}(t) \leq s_{in}^{+}(t) \leq \xi^{+}, \forall \ t \geq 0$$

As shown in our earlier work [2], the study of robustness leads to the fact that, when the specific growth rate function $\mu(.)$ of the bactery is not well known, the system admit an interior domain as a domain of stability, instead a unique stable equilibrium point arising when $\mu(.)$ is perfectly known. Our goal, is to propose a design of robust feedback control in term of recycle rate, in order to keep the system below a suitable level s_d fixed by the environmental authorities. Since the specific growth function and the substrate concentration in the feed stream are not well known, the substrate (polluant) concentration s is unmeasured and hence we can't use it to build the feedback control. Our approach consist to provide a upper observer of s and by using theory of monotone dinamical systems, we prove global partial stabilizability of the upper observer about s_d and hence keeping $s \leq s_d$. Our approach is different from litterature, since firstly, our system contain a recycle phenomena. Furthermore, we use the recycle rate as control instead dillution rate D, often used as control, since this last variable is praticaly very hard to manipulate, and varing D implies variation of somes other parameters. On the other hand, we not require any information on the specific growth function of the bactery. Finaly, our control not require the knowledgment of online measurement of substrat concentration s.

References

[1] M.Z. Hadj-Sadok and J.L. Gouzé, Estimation of uncertain models of activated sludge processes with interval observers. J. of Process Control, 11, 299-310, 2001.

[2] M. Serhani and J.L. Gouzé, Dynamical study and robustness of an activated sludge process problem. Submitted.

Analysis of management measures effects on the fisheries

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We consider a specific fishery bioeconomical model where the dynamic of resource price depends on the gap between the demand and the supply (the catch) in a specific market. The goal of this work is to identify management measures leading the system (resource stock / price) to a bionomic equilibrium at a reasonable level. In a first step, the authors analyse the existence conditions of equilibrium, when the harvest period is indefinite and the catch at each time is constraint to be less than a given level, that is an instantaneous quota. The obtained results depend on biological and economical parameters of the model, and even when the existence conditions of equilibrium are satisfied, it is reached asymptotically and then in an infinite period of time. Moreover, when the catch is over than a critical level, the risk of stock collapse is possible. In order to avoid this critical situation, the authors suggest in a second step of the work to consider an additional classical fishery measure, biological rest, at a specific time computed as the solution of an optimal time problem.

In this situation, the quota becomes the cumulate catch over the entire fishery unknown period T and the problem consists on leading the system state from an initial position (x^0,p^0) to a desired final position (x_T,p_T) in the optimal time T, and then to claim a biological rest period in order to permit the natural stock's reconstitution.

The solution of this problem corresponds to the optimal fishery period T as well as the beginning of the period of biolo gical rest. The value of the biological rest period labelled T' is then evaluated as the required time for the stock/price « natural » growth to reach the desired final position$(x_{T'},p_{T'})$ level.

In a third step of the work, we define a cyclical regulation of the fishery activity when the level of quota is caught, cut out periodically by a biological rest period in order to allow the stock level reconstitution. Some numerical simulations based on specific data related to biological and economical parameters of the south Moroccan Atlantic stock of sardines are computed. The obtained calculations permit to valid the analytical results. This numerical part of the work contribute to a larger project of decisions help in the building of a management plane of small pelagic fisheries in the Moroccan south Atlantic coast.

[1] Clark C.W. (1990), Mathematical Bioeconomics: the optimal management of renewable resouirces, second ed: a Wiley interscience
[2] Jerry M. Raïssi N. (2002) The optimal strategy for a bioeconomical model of harvesting renewable resource problem, Mathematical and Computer Modelling 36, pp.1293-1306.
[3]Mchich R.,Charouki N.,Auger P.M.,Raissi N and Ettahiri O., (2006) Optuial Spatial distribution of the fishing effort in a multi fishing zone model, Ecological Modelling, Vol 197, Issues 3-4, pp.274-280.

Epidemiological signatures of alternative cross-immune responses to BCG

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Keywords: tuberculosis, reinfection, dynamics, cross-immunity, BCG.

Tuberculosis vaccine efficacy presents striking variability in efficacy [1]. Elucidating the cause of this variability could help to improve tuberculosis control strategies. Two of the most widely accepted hypotheses proposed stem from the existing cross-immunity between species of the mycobacterium genus [2], to which tuberculosis belongs. Diversity and abundance of Mycobacteria present in the environment follow a gradient with latitude, which has been associated to vaccine efficacy [1]. However, it is still unclear whether the so-called Environmental Mycobacteria prevent (*block*) development of an immune response following vaccination, or whether they protect against tuberculosis themselves, confounding (*masking*) assessment of vaccine impact. Moreover, differences in the relative contribution of reinfection to active disease burden result from differences in transmission rate and may also underlie contrasting responses to vaccination [3]. Since reinfection is known to occur in tuberculosis, we use a model that incorporates this mechanism to explore the signature of cross-immune (masking and blocking) mechanisms in tuberculosis epidemiology.

References

[1] Fine, P. E. M., Variation in protection by BCG: implications of and for heterologous immunity. The Lancet, 346, 1339-1345, 1995.

[2] Palmer, C. E. and Long, M. W., Effects of infection with atypical mycobacteria on BCG vaccination and tuberculosis, American Journal of Respiratory Diseases, 94, 553-568, 1966.

[3] Gomes, M. G. M. and Franco, A. O. and Gomes, M. C. and Medley, G. F., The reinfection threshold promotes variability in tuberculosis epidemiology and vaccine efficacy, Proceedings of the Royal Society of London Series B-Biological Sciences, 271, 617-623, 2004.

Rich dynamics in multi-strain epidemiological models: from evolution towards criticality to reinfection threshold and new deterministically chaotic attractors

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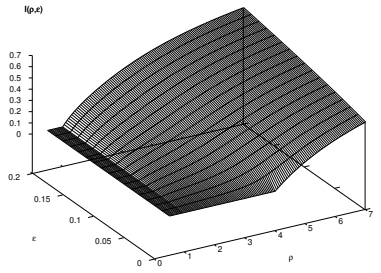
Keywords: Multi-strain models, reinfection threshold, deterministic chaos

As compared to classical epidemiological models multi-strain models show a rich dynamic behaviour, reaching from new thresholds, namely the reinfection threshold in models of partial immunity, for example in influenza, to deterministically chaotic dynamics, in dengue fever models, and can show evolution to critical thresholds including its large fluctuations, in Neisseria menigitidis epidemiology:

1) We describe a model for **Neisseria menigitidis** epidemiology with mutant strains evolving from a harmless resident strain and eventually causing severe disease [1]. The evolutionary disadvantage of causing severe disease leads to a system with mainly harmless carriage and strains which only infrequently cause disease, leading the epidemiological system to a critical state. In this way we can explain the large fluctuations observed in meningitis case data which simpler models without the multi-strain dynamics fail to describe [2].

2) Recently, the notion of a **reinfection threshold** in epidemiological models of only partial immunity has been debated in the literature. We present a rigorous analysis of a model of reinfection which shows a clear threshold behaviour at the parameter point where the reinfection threshold was originally described [3].



The reinfection threshold appears for vanishing demographic parameter ε.

Furthermore, we demonstrate that this threshold is the mean field version of a transition in corresponding spatial models of immunization. The reinfection threshold corresponds to the transition between annular growth of an epidemics spreading into a susceptible area leaving recovered behind and compact growth of a susceptible-infected-susceptible region growing into a susceptible area. This transition between annular growth and compact growth was described in the physics literature [4] long before the reinfection threshold debate broke out in the theoretical biology literature.

3) In models for **dengue fever** we include temporary cross-immunity and find new chaotic attractors in parameter regions previously thought to only give fixed point behaviour [5]. Hence such models are of much wider interest than previously reported.

References

[1] Stollenwerk, N., Maiden, M.C.J., & Jansen, V.A.A. (2004) Diversity in pathogenicity can cause outbreaks of menigococcal disease, *Proc. Natl. Acad. Sci. USA* **101**, 10229–10234.

[2] Stollenwerk, N., & Jansen, V.A.A. (2007) Criticality in epidemiology, in *Complex Population Dynamics: Nonlinear Modeling in Ecology, Epidemiology and Genetics*, eds. Bernd Blasius, Lewi Stone, Jürgen Kurths, (World Scientific Lecture Notes in Complex Systems, to appear).

[3] Stollenwerk, N., Gökaydin, D., Hilker, F., van Noort, S., & Gomes, G. (2007) The reinfection threshold corresponds to the mean field version of the transition between annular and compact growth in spatial epidemic models, *submitted*.

[4] Grassberger, P., Chaté, H., & Rousseau, G. (1997) Spreading in media with long-time memory, *Phys. Rev.* **E 55**, 2488–2495.

[5] Aguiar, M., & Stollenwerk, N. (2007) A new chaotic attractor in a basic multi-strain epidemiological model with temporary cross-immunity, *available as* arXiv:nlin/0704.3174.

A theoretical framework for biological control

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Keywords: Epidemiology, Biological Control, R_0, Thresholds, Stochasticity, Take-all Decline.

Epidemics in plant crops can result in financial losses for growers, increased prices for consumers and can lead to reduced food security and associated hardships, particularly in developing countries. There have even been recent suggestions that plant pathogens could be used as agents of biological terrorism. Plant disease may be controlled by altering cultural practices; by applying fungicides or other agrochemicals; or by planting genetically modified varieties; but each approach has well-known disadvantages. Despite its obvious potential, biological control (in which the natural enemies of a pathogen are manipulated to effect a reduction in disease prevalence) remains beguilingly unsuccessful.

A generic compartmental model for the epidemics caused by soil-borne plant pathogens will be presented, and the standard epidemiological threshold R_0 determined. An antagonist species is then coupled into the system, where in addition to potentially bulking up on either or both susceptible and diseased host tissue, together with the pathogen itself, the antagonist alters one or both of the rates of infection or the infectious period of the pathogen or infected tissue. The differences in R_0 and so potential control efficacy, contingent upon differing effects of antagonism on the pathogen and different reproductive strategies of the antagonist, are investigated and explained. The model is first analysed in deterministic form to derive criteria for invasion; thereafter the robustness of the invasion criteria together with conditions for persistence are tested and confirmed in a version of the model that includes demographic stochasticity.

The framework is made more realistic by introducing the temporal interruptions associated with cultivation and modelling the spatial spread of the pathogen and of the antagonists. It is then applied to model Take-all Decline, in which the size of epidemics of Take-all in wheat initially increase then decrease over successive seasons of monoculture. Understanding the mechanisms by which natural enemies of this economically devastating pathogen cause soils to become suppressive is extremely important, both intellectually and economically.

Travelling Waves Describing the Spatial West Nile Virus Epidemic

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Keywords: Travelling waves - West Nile Virus - Control

West Nile Virus (WNV) is an arthropod-borne flavivirus. WNV disease appears at first time in New York City in the summer of 1999 and then spread prolifically within birds (200 species has been infected). Mammals (including humans) do not develop sufficiently high bloodstream titers of WNV to play a significant role in trasmission. [6].

In this work we study the WNV propagation across the USA. The model proposed to study this propagation is a system of partial differential reaction-diffusion equations, considering the vector (mosquito) and the avian population.

In the avian population the susceptible, infective and recovered subpopulation are considered. In the vector popualtion is only considered the susceptible and infected subpopulation, since, in its short life, the mosquito never recover from the infection [4].

The temporal dynamic considered is the same as in [2]. The cross infection is modeled with the mass action law, considering the number of bites per mosquito, the transmission probability and the number of susceptible and infective of each species.

Parameters concerned to important WNV disease factor are considered as the birds recovered rate, the specific death rate associated with WNV and the vertical transmission in the vector population. The mortality is considered in both population.

The diffusion movement is considered, as in [5], only in the avian population. In our work is considered an advection movement, a preferencial direction of birds.

When the the threshold value $\tilde{R}_0$ determined in [2] is greater than 1, the disease remains endemic, and a infection front could propagete to free disease regions. We study this propagation using the travelling wave solutions [1], [3] to determine the speed wave as function of the essential paramters. This allow us to describe the propagation across the USA, as well as, to propose control strategies.

References

[1] Murray, J. D., Mathematical Biology, Springer, Berlin, 2002.

[2] Cruz-Pacheco, G., Esteva, L., Montaño-Hirose, J.A., Vargas. C., Modelling the dynamics of West Nile Virus. *Bulletin of Mathematical Biology*, **67**, 1157-1172, 2005.

[3] Murray, J. D., Stanley, F. R. S., Brown, D.L. On the spatial spread of rabies amog foxes. *Proc. R. Soc. Lond.* B229, pp. 111-150, 1986.

[4] Gubler, D. J., Dengue. In: Monath,T.P.(Ed.), The arboviruses: Epidemiology and Ecology, Vol II. CRC Press, Florida, pp. 213-161, 1986.

[5] 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.

[6] DeBiasi, R. L. and Tyler, K. West Nile Virus Meningoencephalitis. *Nature Clinical Pratice Neurology*, **Vol. 3 NO 5** pp. 264-275, 2006.

Interference in Classically Entangled Discrete Populations

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Keywords: Stochastic population models, correlations, fluctuations, monitoring.

Discrete population models provide a means of studying the behaviour and possible extinction/survival characteristics of a population in the case where it is inappropriate to approximate the population size as a continuous variable due to its small size. A particularly useful method of solving such discrete models is that of the moment generating function. The generating function solution contains within it all of the information about the process and enables all moments of the distribution that arises to be calculated, where they exist [1]. This method has the advantage over mean field approaches since it permits the consideration and effects of fluctuations. Furthermore, these fluctuations arise directly from the stochastic process itself, rather than from the externally imposed noise that is often used to generate fluctuations in mean field approaches.

The situation under consideration here is that of two death-immigration models coupled by an exchange process whereby occupants of one population may "migrate" into the other and vice versa. In each population, deaths occur in proportion to the instantaneous population size and single immigrants arrive spontaneously and independently of the population size. The exchange processes that couple the two populations occur at a rate that is proportional to the instantaneous population size in the population that is being migrated away from. These processes result in a rate equation that can be solved analytically using the generating function approach. The solution obtained for the entangled system, containing all the information about the process, is significantly modified from that describing the two uncoupled populations. One manifestation of the exchange process is seen in the mean total population size, despite the fact that the process itself is conservative of individuals within the coupled population. With appropriate choices of parameters, the mean total population size for the coupled process can achieve values both above, equal to and below that of the sum of the means of the two separate death-immigration processes: the exchange process introduces a property analogous to interference between the two population sizes.

The impact of the exchange process is also apparent in higher order quantities. By constructing the 2-point generating function it is possible to study the correlation properties of the coupled populations at differing times $t = 0$ and $t = \tau$. The effect of the exchange process is to introduce correlations between each population at different times, with a maximum value of the correlation occurring at a non-zero value of τ. Prior to the introduction of the exchange, the two populations are completely uncorrelated, being Poisson in nature. This peak in the correlation indicates that there is a time delay before fluctuations in one population impact upon the other via the exchange process. It is the fact that there are two timescales involved with the coupled process that gives rise to these interesting correlation properties, as compared to the single timescale encountered in more simple models, for example [2].

A monitoring process, taking the form of additional deaths, is applied to the coupled model, converting fluctuations in population size into a time series of events. The distribution in time between these events is derived and used to calculate such measurable quantities as the time to the first event and the inter-event times. These quantities are discussed in light of the correlation properties mentioned previously.

References

[1] J. O. Matthews, K. I. Hopcraft and E. Jakeman, Generation and monitoring of discrete stable random processes using multiple immigration population models. J. Phys. A: Math. Gen. **38** (2003) 11585-11603.

[2] T. Shepherd, A model for photodetection of single-mode cavity radiation, Optica Acta **28** (1981) 567-583.

A Numerical Method for Separable Models with Finite Maximum Age

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Keywords: Numerical integration, maximum finite age-structured populations

We will study the numerical integration of a nonlinear model which describe the dynamics of a age-structured population with finite maximum age $a_\dagger$. More precisely, we consider a separable model,

$$u_t + u_a = -(m(a) + \mu(a, I_\mu(t), t))\, u, \quad 0 < a < a_\dagger,\ t > 0, \tag{1}$$

$$u(0,t) = \int_0^{a_\dagger} \alpha(a, I_\alpha(t), t)\, u(a,t)\, dx\ , \quad t > 0, \tag{2}$$

$$u(a,0) = \phi(a), \quad 0 \le a \le a_\dagger, \tag{3}$$

$$I_s(t) = \int_0^{a_\dagger} \gamma_s(\sigma)\, u(\sigma, t)\, d\sigma, \quad 0 < a < a_\dagger, \quad t > 0, \quad s = \mu, \alpha. \tag{4}$$

The independent variables a and t represent, respectively, age and time, where $a_\dagger$ is the maximum finite age reached by a given population. The functions $u(a,t)$ is the population density with age a at time t. The functionals I_s, $s = \mu, \alpha$, are employed to describe the competition among individuals for available resources.

In this model, the mortality rate is in a separable form. Function μ is a bounded function in which the nonlinearity is considered, and funcion m is unbounded at the maximum finite age.

The mathematical theory to study such models is well developed in [2]. However, the choice of a numerical method for simulation of this problem, there are hardly any available. On the one hand Iannelli and Milner [3] point out that most classical methods do not work well for such mortality functions; on the other hand Kim and Kwon [4] analyzed a numerical method for a specific type of mortality function. Finally, in [1], we analysed a scheme that works efficiently with unbounded mortality rates.

In the present work, we carry out the numerical integration of the nonlinear model proposed in equations (1)-(4) by means of a new second order numerical method that integrate along the characteristics curves.

References

[1] O. Angulo, F. A. Milner and J. C. López-Marcos, The application of an age-structured model with unbounded mortality to demography, Math. Biosci., doi: 10.1016/j.mbs.2006.11.001.

[2] M. Iannelli, Mathematical Theory of Age-Structured Population Dynamics, Applied Mathematics Monographs. C.N.R., Giardini Editori e Stampatori, Pisa, 1994.

[3] M. Iannelli, F.A. Milner, On the approximation of the Lotka-McKendrick equation with finite life-span, J. Comput. Appl. Math. 136 (2001) 245-254.

[4] M. Y. Kim, Y. Kwon. A collocation method for the Gurtin-MacCamy equation with finite life-span. SIAM J. Numer. Anal. 39(6) (2002) 1914–1937.

Modelling demographic processes of wild mosquito populations

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Keywords: mathematical modeling, data analysis, wild mosquito.

For contributing to the control of arbovirial, parasitical and other diseases transmitted by mosquitoes populations, we develop two different ways of vectors population dynamics modelling close to blood feeding process modelling approach. They produced a stochastic and a determistic model determined respectively by a poissonian and a Dirac distribution. Each one takes account implicitly some environmental factors or conditions which have important impacts in infection and transmission processes of adults female mosquito population to vertebrates such as mortality and emergence. We found a great usefulness of the second way by showing a well and precise mathematical background of the Clements Paterson Method for analyzing and estimating survivals rates (essential in vectorial capacities evaluation) of wild mosquito population [1] which induced a more general related algorithm. The application of our algorithm to a wild cohort data gave good results in survival rates estimation with simple linear regressions according to some remarkable mortality rates models. The model of a single emergence is characterized by duration of mosquito aquatic stages which depend on environmental conditions and the total emerged females. Finally, in the particular case of *Aedes vexans* population in temporary ponds area like sahelian domain, multiple emergence processes during a rainy season is related to the sequence of rain, the wetness of ponds and the duration between rain evens with a easy use relational scheme by analysing fields data and biological knowledge.

References

[1] A.N. Clements and G.D Paterson, The analysis of mortality and survival rates in wild population of mosquitoes. J. Applied Biology. 18, 373 - 399 ,1981.

Aedes aegypti population model for analysis of dengue control strategies in Rio de Janeiro, Brazil

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Keywords: dengue, *Aedes aegypti*, vector contol, modeling.

Four antigenically distinct serotypes of dengue viruses are transmitted by *Aedes aegypti*, a mosquito well adapted to breeding in a range of containers present near and inside houses. The emergence of dengue fever and its severe forms (DHF, dengue hemorrhagic fever and DSS, dengue shock syndrome) is linked to population growth and unplanned urbanization. There is no specific treatment available for dengue; disease activity can be monitored in humans or vectors. Entomologic surveillance in Rio de Janeiro (RJ) is carried out by measuring larval infestation levels (the house and Breteau indices). Dengue prevention can be achieved by vector control [2].

The objective of this work is to simulate *Ae. aegypti* population in RJ for comparative analysis of vector control strategies. We developed a continuous time life stage structured model that incorporates a population genetic framework (for resistance to chemical control). Resistance is modeled as a single locus with two alleles; there is a fitness cost to resistance which acts on phenotypically resistant mosquitoes. Biological and environmental features are incorporated: larval mortality is density dependent and mosquito population dynamics is seasonal [1]. Seasonality is incorporated mechanistically [4] using environmental data from RJ. The model output is fitted to the observed indices data for RJ.

The vector control strategies considered are source reduction and chemical control of larvae and adults, as well as combined strategies. Important covariates examined for each strategy are their maximum efficacy and persistence. Strategies are implemented as continuous health programs or discrete interventions (that is, specific interventions being executed at scheduled times). The model outcome for comparative analysis is the relative reduction of adult population determined by each strategy. Also, since resistance evolution should be minimized, chemical control strategies are compared with respect to the time until a fraction of the population is resistant.

We find that only through the targeting of adults can immediate and efficient reduction of the adult population be achieved. Chemical control of larvae has a paradoxical effect: larval mortality leads to the "removal" of the biological regulation of the population through density dependence. The overall effect is a new dynamic equilibrium determined by the fitness cost of resistance. Efficiency of each strategy is a function of when (during the one calendar year) they are applied.

In RJ vector control is a complex task. Dengue epidemics of increasing magnitude reveal the inefficiency of the current vector control strategies; also, resistance is observed [3]. This model shows that an optimal timing for each intervention exists. Currently, a fixed schedule of vector control and resistance monitoring is carried out by health agents. An adaptive control strategy might be more effective.

References

[1] R. Barrera, M. Amador and G. G. Clark, Ecological factors influencing *Aedes aegypti* (Diptera: Culicidae) productivity in artificial containers in Salinas, Puerto Rico. Journal of Medical Entomology 43(3):484-492, 2006.

[2] D. J. Gubler and G. Kuno, Dengue and dengue hemorrhagic fever, CAB International, New York, 1997.

[3] J. B. P. Lima, M. P. da-Cunha, R. C. S. Junior, A. K. R. Galardo, S. S. Soares, I. A. Braga, R. P. Ramos and D. Valle. Resistance of *Aedes aegypti* to organophosphates in several municipalities in the state of Rio de Janeiro and Espirito Santo, Brazil. American Journal of Tropical Medicine and Hygiene 68(3):329-333, 2003.

[4] C. C. Lord, Seasonal population dynamics and behaviour of insects in models of vector-borne pathogens. Physiological Entomolgy 29:214-222, 2004.

Patterns of genetic variation in pathogen populations

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Keywords: genetic variation, pathogens, SIS, networks

The analysis of genetic variation in pathogen populations may help us understand their epidemiology and evolution. We introduce a model for assessing the levels and patterns of genetic diversity in pathogen populations, whose epidemiology follows a susceptible-infected-susceptible (SIS) model. We assume a population which is structured into many small subpopulations (hosts) that exchange migrants (transmission) between their neighbors, that are connected according to a specific type of contact network. We considered different types of networks: the fully connected network (island model) and scale free networks [2], which have been considered as a model that captures properties of real contact networks. Pathogens transmit between hosts, through migration, where they grow and mutate until elimination by the host immune system.

In the classical SIS model, individuals can be in of two states: susceptible (S) and infected (I). The time evolution of the fraction of individuals in both states is determined by

$$\frac{dS}{dT} = -\beta SI + \alpha I \tag{1}$$

$$\frac{dI}{dT} = \beta SI - \alpha I \tag{2}$$

where β is the transmission rate, and α is the rate at which infected individuals become susceptible. The relevant parameter of the model is the basic reproductive number $R_0 = \beta/\alpha$. When $R_0 < 1$ the epidemics does not spread (i.e., $I = 0$), whereas for $R_0 > 1$ the non-null solution $I = 1 - 1/R_0$ becomes stable.

In our model, we consider a metapopulation of hosts (demes). Each host can carry at most N_d pathogens. Each node in the network corresponds to a given host, and the total number of hosts is D. Therefore, the maximum number of pathogens in the metapopulation is $N_t = DN_d$. Each host can clear the pathogens it carries with probability e at each generation. When this occurs it becomes empty. However, since we also assume migration, empty hosts can be recolonized. This latter step makes our model distinct from previous metapopulation models where migration and recolonization are distinct processes [1]). At each generation, the number of emigrants n_e of a given deme j (which is not empty) is taken from a Poisson distribution of mean $N_d m k_j$, where m is the migration rate and k_j is the connectivity of deme j.

Making the analogy between our model and the classical SIS, we check that the parameter e corresponds to α and that the transmission rate in Eq. (1) corresponds to $N_d m K$, where K is the mean connectivity of the network under consideration. The relevant parameter R_0 is then given by

$$R_0 = \frac{N_d m K}{e}. \tag{3}$$

From our simulations we find that: depending on the relation between the rate at which pathogens are eliminated by the immune system and the within host effective population size, pathogen genetic diversity increases with R_0 or peaks at intermediate R_0 levels. This is independent of the type of host contact structure. Patterns of genetic diversity in the model are in general similar to those expected under the standard neutral model, but in a scale free network and for low values of R_0 a distortion in the neutral mutation frequency spectrum can be observed; highly connected hosts (hubs in the network) show patterns of diversity different from poorly connected individuals, namely higher levels of genetic variation, lower levels of genetic differentiation and larger values of Tajima's D.

References

[1] J. R. Pannell and B. Charlesworth, Effects of metapopulation processes on measures of genetic diversity. Phil. Trans. R. Soc. Lond. B 355, 1851-1864, 2000.

[2] R. Albert R and A-L Barabási, Statistical mechanics of complex networks. Rev Mod Phys 74, 47–97 (2002).

Genetic linkage and the subversion of natural selection

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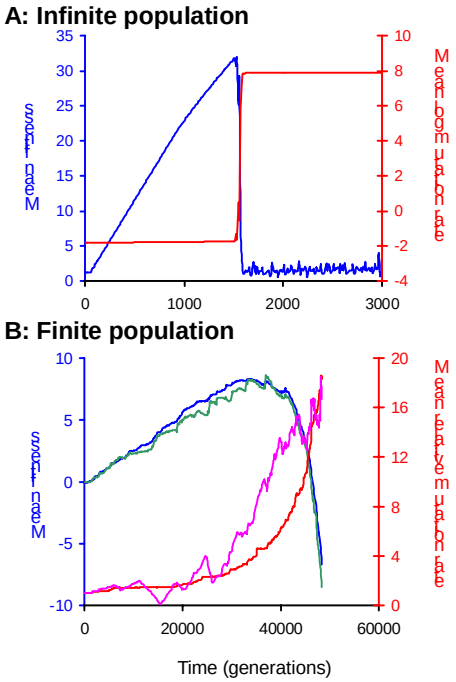
Keywords: evolution, population dynamics, biophysics, mutation rate, asexual, clonal.

The intricate adjustment of organisms to their environment demonstrates the effectiveness of natural selection. But Darwin himself recognized that certain biological features could limit this effectiveness – features that generally reduce the efficiency of natural selection or yield sub-optimal adaptation. Genetic linkage is known to be one such feature, and here we show theoretically that it can introduce a more sinister flaw: when there is complete linkage between loci affecting fitness and loci affecting mutation rate, positive natural selection and recurrent mutation can drive mutation rates in an adapting population to intolerable levels. We discuss potential implications of this finding for the early establishment of recombination, the evolutionary fate of asexual populations, and immunological clearance of clonal pathogens.



References

[1] P.D. Sniegowski, P.J. Gerrish, R.E. Lenski, Evolution of high mutation rates in experimental populations of *Escherichia coli*, Nature 387:703-705, 1998.

[2] P.J. Gerrish, A. Colato, A.S. Perelson, P.D. Sniegowski, Complete genetic linkage can subvert natural selection, PNAS 104:6626-6271, 2007.

Conditions for the existence of a convergence stable strategy in a discrete stage-structured population model

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Keywords: Stage-structured Population, Matrix Model, Convergence, Stability, and Management Strategy

There has been a fair amount of previous work on stage-structured models of population growth consisting of immature and mature individuals for single species, where the stage-structure $\mathbf{n}(t)$ is modeled by the introduction of a transition matrix. In a linear matrix population model $\mathbf{n}(t+1)=\mathbf{n}(t)\mathbf{P}$, population performance can be measured by the population growth rate λ, given by the dominant eigenvalue of $\mathbf{P}$. The sensitivity and elasticity of λ to changes in the entries of $\mathbf{P}$ provide a wealth of information about the effects of environmental change, the action of natural selection, and the efficacy of management strategies.

In the present work, we consider the stage-structured population where in general migratory effects play an essential role. We try to extend the linear matrix population model to nonlinear models $\mathbf{n}(t+1)=\mathbf{n}(t)(\mathbf{Q}+\mathbf{e}^{T}\mathbf{r})+M(t)\mathbf{r}$, where $\mathbf{Q}$ is a semi-transition matrix, vector $\mathbf{e}$ is the emigration structure, $\mathbf{r}$ is the vector of proportions of immigrants, and $M(t)$ is the increment of population size. The conditions under which a vector of the model has a steady population structure are identified, as well as those under which the stage structure converges to a given steady state, through a series of decisions or controls of letting immigrants in or forbidding them entry into the country. The decisions are expressed as vectors of proportions of immigrants. In the steady state, when the increment of population is proportional to its size, the stage-structure remains unchanged between transitions.

References

[1] J. Ackleh, A.S. and Deng, K. Monotone approximation for a hierarchical age-structured population model. Dynamics of Continuous Discrete and Impulsive Systems-Series, B-Applications & Algorithms 12(2): 203-214, 2005.

[2] Caswell, H. Matrix Population Models. Construction, Analysis, and Interpretation. 2nd Edition. Sunderland, MA: Sinauer Associates, 2001.

[3] Davydova, N. V., Diekmann, O. and van Gils, S. A. Year Class Coexistence or Competitive Exclusion for Strict Biennials? Journal of Mathematical Biology 46,95-131, 2003.

[4] Gourley, S. A. and Wu, J. H. Extinction and periodic oscillations in an age-structured population model in a patchy environment. Journal of Mathematical Analysis and Applications 289(2): 431-445, 2004.

[5] Grant, A. and Benton, T. G. Elasticity analysis for density dependent populations in stochastic environments. Ecology 81: 680-693, 2000.

[6] He, Z. R., Wang, M. S. and Ma, Z. E. Optimal birth control problems for nonlinear age-structured population dynamics. Discrete and Continuous Dynamical Systems-Series B 4(3):589-594, 2004.

[7] Jensen, A. L. Matrix population model with density-dependent recruitment for assessment of age-structured wildlife populations. Bulletin of Mathematical Biology 59(2): 255-262, 1997.

[8] Zhang, Qingguo, Hu, Bingmin. A matrix model for population dynamics within open system with immigration and emigration of individuals. In: Chen, L., Ruan, S., and Zhu, J., Advanced topics in biomathematics. New York: World Scientific Publication Company, pp. 303-308, 1998.

Some bio-economic models of fisheries: Stabilizability of a controlled system and Optimal spatial distribution

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Keywords: Population dynamics, fisheries, aggregation, stability, Control.

Abstract: We present some stock-effort dynamical models describing the evolution of a fish population growing and moving between several fishing zones, on which it is harvested by a fishing fleet, distributed on the different zones. The bio-economical models are sets of ODE's governing the fishing efforts and the stocks on the different fishing zones. We take profit from the existence of two time scales (a fast one for fish migration and fleets movements, and a slow one for fish growth and mortality and fleets revenue) to construct reduced (aggregated) models. Aggregated models describe the global evolution of the harvested stock as well as the total fishing effort.

In equations of one of the models, a control function is introduced as the proportion of the revenue to be invested, for each fleet. The stabilizability analysis of the aggregated model, in the neighbourhood of the equilibrium point, enables the determination of a Lyapunov function, which ensures the existence of a stabilizing discontinuous feedback for this model. This enables us to control the system and to lead, in an uniform way, any solution of this system towards this desired equilibrium point.

In the other models, the mathematical analysis of the aggregated models allows the optimization of the spatial distribution of the fishing effort and the identification of an efficient set of management measures, which corresponds in one hand to set an appropriate system of tax and/or subsidies, and on the other hand to control the displacement of the fleets between the fishing zones, in order to increase the total activity.

References

[1] P. Auger, A. El Abdllaoui & R. Mchich, Méthode d'agrégation de variables appliquée à la dynamique des populations, ARIMA, numéro spécial TAM TAM'05, Vol. 5, 26-36, 2006.

[2] P. Auger & J.C. Poggiale, Aggregation and Emergence in Systems of Ordinary Differential Equations, Math. Computer Modelling. Special issue "Aggregation and Emergence in Population Dynamics". P. Antonelli & P. Auger guest-Editors. 27(4), 1-21, 1998.

[3] P. Auger & R. Bravo de la Parra, Methods of aggregation of variables in population dynamics, CRAS, Sciences de la Vie, 323, 665-674, 2000.

[4] R. Mchich, P. Auger, R. Bravo de la Parra & 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, 51-62, 2002.

[5] R. Mchich, P. Auger & N. Raïssi, The dynamics of a fish stock exploited on two fishing zones, Acta Biotheoretica. Vol. 48, N. ¾, 207-218, 2000.

[6] R. Mchich, P. Auger & N. Raïssi, The stabilizability of a controlled system describing the dynamics of a fishery, Comptes Rendus Biologies, Vol. 328 (4), 337-350, 2005.

[7] R. Mchich, N. Charouki, P. Auger, N. Raïssi & O. Ettahiri, Optimal spatial distribution of the fishing effort in a multi fishing zone model, Ecological Modelling, Vol. 197, Issues 3-4, 274-280, 2006.

Estimating rubella incidence from deterministic models

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Keywords: incidence, mathematical model, rubella.

Mathematical models have been used to estimate the impact of the introduction of vaccines in populations mainly by determining how the force of infection is changed. From this kind of parameter, it is possible to estimate the average age of first infection, and the proportions of susceptible, infected or immune individuals over time. Deterministic models usually do not differentiate clinical and subclinical infections. This creates a difficulty to establish a parallel between model results and incidence recorded by surveillance systems, which take into account the number of new cases (clinical). Moreover, subnotification is more likely, contributing to a greater difference between models and official data.

The purpose of this paper is to propose a method to estimate incidence from a SIR model, using rubella data.

Data from a previous serological survey for rubella published elsewhere were modeled to calculate the age- and time-dependent force of infection before the vaccination program was introduced [1].

Based on the SIR model, the proportion of immune individuals at age a and time t, $r(a,t)$, was calculated [1]. The proportion of new infections, $\Psi(a, t)$, was then estimated by

$$\Psi(a,t) = r(a,t) - r(a-\Delta t, t-\Delta t) \quad (1)$$

where Δt is a time interval (Figure 1).

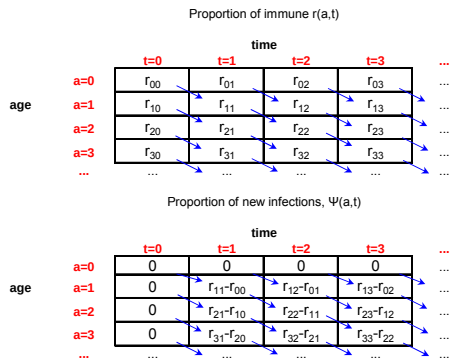

Figure 1 – Schematic illustration of equation (1)

This formulation was adopted because the proportion of immune individuals in the model has a similarity with what can be measured in a population by seroprevalence, allowing the comparison between data and model results. The increase in seroprevalence for a given age cohort can be followed for each time interval. This increase offers the possibility to estimate the incidence in the period.

Applying this method for rubella data considering immunization between 1 and 2 years old children with 80% effective coverage, it was estimated the impact on incidence after calculating $\Psi(a,t)$ (Figure 2).

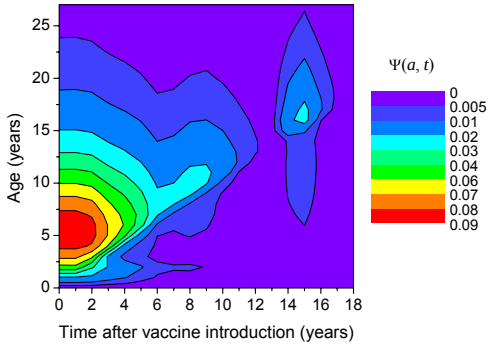

Figure 2 – Contour plot of $\Psi(a,t)$

Thus, after 11 years of vaccine introduction, the 15 year-old cohort would have a $\Psi(a, t)$=0.005, corresponding to an incidence of 500 cases / 100,000 inhabitants. Next step is to compare these modeling estimates of incidence with notification data.

References

[1] M. Amaku et al., Vaccination against rubella: Analysis of the temporal evolution of the age-dependent force of infection and the effects of different contact patterns. Physical Review E 67, 051907, 2003.

Models to directly transmitted diseases based on fuzzy rules

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Keywords: fuzzy controller, phase-plane, epidemiological models.

Traditionally, mathematical models to describe the dynamic of directly transmitted infectious diseases are described by an autonomous system of differential equations and its qualitative behaviour can be analyzed through phase-plane methods ([1],[2]). The model proposed in this work uses fuzzy controllers of the Mamdani type, which considers the knowledge of specialists to formulate the base of rules ([3],[4]). These rules can be compared to the direction field of a deterministic system, being able to be also represented in a phase-plane. To obtain a solution (trajectory) of the fuzzy system we join the fuzzy controllers theory to methods of numerical analysis (Runge-Kutta) [5]. Besides representing the disease evolution, the solution obtained from fuzzy system can be used to estimate parameters of epidemiological deterministic systems, even in the absence of statistical data.

References

[1] L. Edelstein-Keshet, Mathematical Models in Biology, New York: Random House, 1988.

[2] H. M. Yang – Epidemiologia matemática – Editora da Unicamp, 2001.

[3] L.C. Barros, R.C. Bassanezi, Tópicos de Lógica Fuzzy e Biomatemática, 1ª.ed., Campinas, IMECC-UNICAMP, 2006.

[4] M.S.Peixoto, L.C. Barros, R. C. Bassanezi. Predator-Prey Fuzzy Model. (aceito para publicação no Bulletin of Mathematical Biology, 2007.

[5] W.Pedrycz, F. Gomide, An introduction to fuzzy sets: analysis and design, The MIT Press, Massachusets, 1998.

OPTIMAL CONTROL OF MOSQUITO *Aedes aegypti* BY THE STERILE INSECT TECHNIQUE

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Keywords: Optimal Control; minimum Pontryagin principle; Aedes aegypti; Mathematical modeling; Sterile insect technique.

The life cycle of mosquito Aedes aegypti is comprised by two phases: aquatic (egg, larvae and pupae) and winged form (adult mosquitoes). We present a model to describe the dynamics of mosquito population in which sterile male mosquitoes (prodeced by irradiation) are introduced as a form of biological control, besides the traditional application of insecticide. Our model is formulated taking into account a system of ordinary differential equations which describes the dynamics of the mosquitoes in the aquatic phase, of the immature female mosquitoes (before mating), of the fertilized female mosquitoes (after mating), of the unfertilized female mosquitoes (after mating), of the natural male mosquitoes (wild) and of the sterile male mosquitoes (irradiated). In order to analyze the minimal effort to reduce the fertile female mosquitoes, we search for the optimal control considering the cost of insecticide application, the cost of the production of irradiated mosquitoes and their delivery and the social cost (number of fertilized female mosquitoes, potential transmissor of dengue). The optimal control is obtained by applying the minimum Pontryagin principle.

References

[1] L. Esteva & H. M. Yang, Mathematical Model to Asses the Control of Aedes aegypti Mosquitoes by the Sterile Insect Technique. Mathematical Biosciences, 198: 132-147 (2005).

[2] W. Fleming & R. Rishel, Deterministic and Stochastic Optimal Control, Springer-Verlag, New York, 1975.

[3] D. E. Kirk, Optimal Control Theory: An Introduction, Prentice-Hall, Englewood Cliffs, 1970.

[4] E. F. Knipling, Possibilities of insect control or eradication through the use of sexually sterile males. Journal of Economic Entomology, 48: 459-462 (1955)

Y-linked bisexual branching models

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Keywords: Sex-linked inheritance; Y-linked genes; Bidimensional bisexual stochastic model; Perfect fidelity mating; Survival.

It is well-known that in some animal populations the sex of the individuals is determined by a pair of chromosomes X (or Z) and Y (or W). A female will have XX chromosomes, while a male will have XY chromosomes. Certain characteristics are due to genes carried on the X chromosome (X-linked). Others due to genes carried on the Y chromosome (Y-linked) and still others by genes on both chromosome types (XY-linked). Taking into account this fact, females and males with different genotypes and/or phenotypes will be present in the population. Moreover, females and males in a generation form mating units in order to produce offspring. Therefore, each mating unit has a genotype which is formed from the genotypes of the two individuals in the unit. Consequently, an individual offspring will receive its genetic structure as specified by the inheritance rules associated to that species.

From a practical viewpoint, it is of interest to model and to analyze the evolution of sex-linked genes from generation to generation. But first, a sexual reproduction scheme must be developed since we are assuming that offspring are generated by mating units.

A first approach has been provided in [1] by considering Y-linked genes having two alleles, say R and r, so that only males can be carriers of such a gene. Under this genetic context and following the inheritance rules, the male descendants of a mating unit have the same allele that their father. Moreover in that paper it is assumed that the carriers of the R allele are preferred by females as mates because the r allele is considered pernicious or of a negative character. Furthermore, perfect fidelity mating is the only mating mechanism allowed, that is, an individual may mate with no more than one individual of the opposite sex from that generation. Therefore, a female mates with a male carrying the r allele if there is no R-carrier available.

Implicitly, this scheme considers that males carrying the r allele and males carrying the R allele have different phenotypes. However, for many Y-linked genes both alleles give rise to the same phenotype. Therefore, selective mating is not possible and females must choose randomly males as mate.

Considering that both alleles produce the same phenotype and perfect fidelity mating, in this work we will propose a two-type bisexual branching process adequate to describe the behaviour of genotypes defined by Y-linked genes. We will study its basic properties and will provide some conditions for the extinction and/or survival of Y-linked genes in the population. Such conditions will depend on the magnitude of the average number of female and male descendants per mating unit. Finally, we will illustrate the theoretical results by means of simulated examples and will propose some open problems as conjectures.

References

[1] M. González, D. M. Hull, R. Martínez, M. Mota. Bisexual branching processes in a genetic context: The extinction problem for Y-linked genes. Mathematical Biosciences 202, 227-247, 2006.

A Simple SI Model with Two Age Groups and Its Applications to HIV

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Keywords: Structured SI model, global stability, HIV epidemic..

First, we review simpler S-I epidemic models that differ from the others in the fact that an infected individual could produce a susceptible newborn, takes into account the density dependence due competition for resources and maintain sufficient simplicity to the model to be used in practical empirical work. We make an introduction to the classical population model with demographic structure, as a first step to combine the epidemic and demographic structures. Finally, we will pay attention to the demographic variation of the former SI epidemic model, it means, we first divide our total population into juvenile and adult groups and immediately we consider the S-I model in each subpopulation, taking into account the maturity concept (transition from juvenile to adult), the restriction that only adults are sexually active individuals, the assumption that infected adults could produce susceptible newborns and the condition of density dependence effect. Simulations of the model have been constructed to check the analytical results of local and global stability.

References

[1] R.M. Anderson, The Role of mathematical models in the study of HIV transmission and the epidemiology of AIDS, J. AIDS 1:214-256, 1988.

[2] F. Brauer, A Model for an SI Disease in an Age-Structured population, Discrete and Continuous Dynamical Systems-Serie B, Vol 2, May 2002

[3] O. Dieckmann and A.P. Heesterbeek, Mathematical Epidemiology of Infectious Diseases: Model Building, Wiley, 2000

[4] Y. Kuang and E. Beretta, Global Qualitative Analysis of a ratio-dependent predator-prey system, J. Math. Biol., 36, 389-406, 1998

[5] R. López Cruz, Structured SI Epidemic Model with Applications to HIV Epidemic, Doctoral Thesis, Department of mathematics, Arizona State University, May 2006

On the Fractional Predator-Prey Model

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Keywords: Fractional Calculus, Predator-Prey Model, Population Dynamics, Lotka-Volterra System, Fractional Differential Equations, Mittag-Leffler Functions.

Probably the multiple non equivalent definitions of fractional derivatives and a non evident geometrical interpretation contribute to the non utilization in large scale of the concepts of fractional calculus, that involves fractional integration and fractional derivatives [1, 2]. Here, we present only one of the non equivalent definitions of the fractional derivatives, the so-called Caputo fractional derivative. There are several ways to introduce the fractional derivatives as a generalization to the ordinary derivative, particularly, the Riemann-Liouville and Caputo ones. We also mention the Weyl and Grünwald-Letnikov methods [1]. The derivative in the Caputo's sense is appropriate to discuss fractional differential system because there exists a physical interpretation for the initial conditions, which doesn't happen, for example, with the Riemann-Liouville formulation. This paper proposes a generalization for a dynamical system [3] utilizing fractional derivatives [1]. The aim of this generalization is to improve the description of the phenomena in an analogous way to the one that was made in works concerning viscoelastic systems, such as human blood [4]. The classical predator-prey model, *i.e.*, the Lotka-Volterra system with derivatives to integer order is discussed and, using a linearization technique, a solution is obtained in terms of the constant parameters. In addition a solution to the so-called Lotka-Volterra fractional differential system, which is a system of two non linear fractional differential equations where each fractional derivative has order lower than one, is obtained in terms of the Mittag-Leffler function [5, 6, 7]. Here, we use the Laplace transform methodology associated with the linearization system to obtain a solution of the Lotka-Volterra fractional differential system [8].

References

[1] I. Podlubny, *Fractional Differential Equations*, Mathematics in Science and Engineering, Vol.198, Academic Press, San Diego, (1999).

[2] K. S. Miller and B. Ross, *An Introduction to the Fractional Calculus and Fractional Differential Equations*, John Wiley & Sons, Inc., New York, (1993).

[3] S. S. Ray, B. P. Podar and R. K. Bera, *Analytical Solution of a Dynamical System Containing Fractional Derivative of Order One-Half y Adomian Decomposition Method*, Trans. of the ASME, **72**, 290-295, (2005).

[4] R. L. Bagley and P. J. Torvik, *A Theoretical Basis for the Application of Fractional Calculus to Viscoelasticity*, J. Rheol., **27**, 201-210, (1983).

[5] F. Mainardi and R. Gorenflo, *On Mittag-Leffler-Type Functions in Fractional Evolution Processes*, J. Comput. Appl. Math., **118**, 283-299, (2000).

[6] F. Mainardi, R. Gorenflo and A. Vivoli, *Renewal Processes of Mittag-Leffler and Wright*, Fractional Calculus and Applied Analysis, **4**, 1-38, (2005).

[7] R. Figueiredo Camargo, E. Capelas de Oliveira e Ary O. Chiacchio (in Portuguese) *Sobre a Função de Mittag-Leffler*, Technical Report RP15/6 - Abril de 2006.(Available at *http* : *//www.ime.unicamp.br/rel_pesq/*2006/*rp*15−06.*html*)

[8] R. Figueiredo Camargo, E. Capelas de Oliveira and F. A. M. Gomes, *Replacement of Lotka-Volterra Model by a Formulation Involving Fractional Derivatives*, To be submited for publication (2007).

EPIDEMIOLOGICAL CHARACTERISTICS OF LYME-SIMILE IN CHILDREN

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Keywords: Lyme-simile disease, exanthem, epidemiological study.

Exanthematic febrile diseases are a frequent cause of cases seen at paediatric emergency services, where immediate attention is sometimes necessary due to a large number of diagnostic possibilities and the need for antibiotic treatment for diseases such as Lyme-simile. The first cases of this disease were identified in 1992, in Itapevi, SP, and isolated cases have been found in adults, though rarely in children. From 15/07/98 to 18/11/2000 a cohort study was carried out in a reference hospital, involving children with exanthematic diseases aged between 0 and 15, to determine the incidence, age distribution, and seasonality of exanthematic infections including Lyme-simile disease.

333 children were included in the study. Borreliosis tests were only carried out in the 193 samples with negative results to other pathogens (B19, HHV6, measles, Rubella, Scarlet fever and Enterovirus). They were analyzed using the ELISA and Western Blot methods. The incidence of serum-positiveness for Lyme-simile was of 3.6% (12/333). Age distribution: 0a 2a: 41,6%(5/12); 3a 6a: 41.6%(5/12) and 7ª e+ 16.7%(2/12), mostly from the town of Franco da Rocha (58.3%). Bivariate analysis showed that there was a predominance of cases during autumn-summer, [$p(x^2)=0.08$] and ORB=1,41 ($IC_{95\%}$ 0-354-5.626), the presence of itching [$p(x^2)=0.04$] and ORB=0.05 ($IC_{95\%}$ 0-06-1.221) and the absence of lip fissure [$p(x^2)=0.004$]. There was predominance in females 8/12 (66.67%), but without any significance. There were no clinical suspicions of Lyme-simile and its prevalence was of 3.6%.

With this field data, infection transmission models can be validated, and the dependency of incidence in relation to seasonal variations can be explored.

References

1. Cherry JD Contemporary nfectious exanthems.
2. Clin Infect Dis. 1993 Feb;16:199-205
3. Gable EK, Liu G, Morrell DS. Pediatric exanthems. Prim Care. 2000 Jun;27(2):353-69.
4. Yoshinari NH; Barros PJL; Gauditano G; Fonseca AH et al. Report of 57 cases of Lyme-like disease in Brazil. Arthritis Rheum 2000; 43: S188.
5. Steere AC; Malawista SE; Hardin JA et al. Erythema chronicum migrans and Lyme arthritis: the enlargingclinical spectrum. Ann Intern Med 1977; 86:685-98.
6. Azulay RD; Abulafia-Azulay L; Sodré CT et al. Lyme Disease in Rio de Janeiro, Brazil. Int J Dermatol 1991; 30:569-71.
7. Yoshinari NH; Oyafuso LK; Barros PJL et al. Doença de Lyme. Relato de um caso observado no Brasil. Rev Hosp Clin Fac Med São Paulo 1993; 48 (4): 170-4.
8. Yoshinari NH; Barros PJL; Stelling R; Baggio D; Yassuda P; Cossermelli W. Epidemiological study of Lyme disease in Cotia. Rev Especial Reumatol 1993; 20: 474.

Influenza A evolution paradigm shift: how can we explain the recurrence of influenza A epidemics in humans?

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Keywords: multi strain pathogen dynamics, antigenic drift, extinction-coexistence, transient dynamics, seasonality, cross-immunity.

Until recently, it was assumed that the recurrence of influenza A epidemics in humans resulted from the continuous appearance of positively selected immune-escape virus mutants. Thus, the susceptible host pool depleted after an epidemic was supposedly renewed by the progressive evolution of the virus [1].

This progressive evolutionary-epidemiological mechanism (called antigenic drift) has been challenged by new sequencing data [2], antigenic mapping [3] and theoretical development [4]. It is now being considered that influenza A main antigen seasonal evolution is driven by « long intervals of stasis punctuated by burst of positive selection » [5]. Such a view highlights the critical role of rare mutations (or reasortments) with strong antigenic effects in the evolution of influenza A.

Starting from this new paradigm for influenza A epidemiology and evolution, and neglecting the previously used SIRS framework, we present a minimal stochastic serial SIR model capturing the invasion of a rare immune-escape mutant with strong antigenic effect. We show that for influenza A parameter values, the evolutionary process proposed in [5] alone results in the local extinction of both resident and mutant viruses with a trade-off between invasion and persistence success. Thus, in order to understand the recurrence of influenza A in humans, others factors than those included in our serial SIR model are needed. We explore the role of seasonality in mediating resource (susceptible hosts) exhaustion; the role of local extinction/recolonisation spatial dynamics; the role of ecological interference (e.g. quarantine, isolement), immune mediated interactions (e.g. due to short time heterosubtypic cross-immunity) between co-circulating influenza A subtypes and, the role of progressive evolution. Our study therefore illustrate the feedback loop between immunology, epidemiology and evolution that, influenced by external factors, gives rise to the recurrence of influenza A epidemics.

References

[1] M.I. Nelson, E.C. Holmes, The evolution of epidemic influenza. Nature Reviews Genetics 8(3): 196, 2007.

[2] M.I. Nelson, L. Simonsen, C. Viboud, M.A. Miller, J. Taylor, K.S. George, S.B. Griesemer, E. Ghedin, N.A. Sengamalay, D.J. Spiro, Stochastic Processes Are Key Determinants of Short-Term Evolution in Influenza A Virus. PLoS Pathogens 2(12): e125, 2006.

[3] D.J. Smith, A.S. Lapedes, J.C. de Jong, T.M. Bestebroer, G.F. Rimmelzwaan, A.D. Osterhaus, R.A. Fouchier, Mapping the antigenic and genetic evolution of influenza virus. Science 305(5682):371-376, 2004.

[4] K. Koelle, S. Cobey, B. Grenfell, M. Pascual, Epochal evolution shapes the phylodynamics of influenza A (H3N2) in humans. Science 314: 1898-1903, 2006.

[5] Y.I. Wolf, C. Viboud, E.C. Holmes, E.V. Koonin, D.J. Lipman, Long intervals of stasis punctuated by bursts of positive selection in the seasonal evolution of influenza A virus. Biology Direct 1:34, 2006.

Epidemiological modelling for managed herds

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Keywords : epidemiological model, stability analysis, singularly perturbated systems, theoretical identifiability, handled flocks, health qualification.

Mathematical modeling is a useful tool when studying different strategies in the management of sheep flock or cattle herd. Previous works presented some simulation models to study the spread of bovine viral-diarrhoea (BVD) in a cattle herd or the spread of scrapie in a sheep flock : a stochastic individual based model for BVD, see [1], requiring more mathematical formalism. and a finely structured model, based on PDEs, for the scrapie, see [2], limited to a within-herd approach.

Our aim is to design and study simplified epidemiological models for BVD and scrapie which allow us to evaluate health qualification strategies by a modeling approach, using systems of non-linear ODEs. In further works, these results will be helpfull in a model coupling approach, giving simplification of mathematical within-herd models.

This study is based on a SIR model which describes the dynamics of a generic disease in a handled flock. We built a deterministic compartmental model in continuous time, incorporating epidemiological processes and management of the flock.
The flock is structured according to epidemiological status (susceptible or infected) and two discrete structures : a fixed one (genotype/lot) and a variable structure (age class). Depending on the disease, we consider one or more infectious status. Moreover, we consider entry flows to modelize exchanges between herds.
>From this generic model, we derive two specific ODE models related to scrapie and BVD.

On these specific models, the stability analysis underline the existence of an endemic state. Using results from the singular perturbation theory, we bring to the fore fast-low dynamics. Consequently, we can simplify these specific models. Moreover, when several infectious states exist, as in BVD, we can throw light on the epidemiological dynamics, and on the relative importance of the infectious states.
Finally, the theoretical identifiability of parameters, performed with algorithms from [3] and [4], showed that few parameters are required to describe the model.

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Références

[1] A.F. Viet, C. Fourichon, H. Seegers, C. Jacob, C. Guihenneuc-Jouyaux. A model of the spread of the bovine viral-diarrhoea virus within a dairy herd. Preventive Veterinary Medicine, 63, 211-236. 2004.

[2] S. Touzeau, M. E. Chase-Topping, L. Matthews, D. Lajous, F. Eychenne, N. Hunter, J. D. Foster, G. Simm, J.-M. Elsen and M. E. J. Woolhouse. Modelling the spread of scrapie in a sheep flock : evidence for increased transmission during lambing seasons. Archives of Virology, 151(4) :735-751, 2006.

[3] S.Audoly, G. Bellu, L.D'Angiò, M.P. Saccomani, and C. Cobelli. Global Identifiability of Nonlinear Models of Biological Systems. IEEE Transactions on biomedical engineering, 48(1), 2001.

[4] A. Sedoglavic. A probabilistic algorithm to test local algebraic observability in polynomial time. Journal of Symbolic Computation 33(5), 735-755. 2002.

The effects of the intersexual competition on the sex-ratio of hermaphrodite

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Keywords: Sex inversion, Sex-ratio, Discrete Dynamical Systems, Bifurcation, Chaos.

We propose in this work to modelise the mechanism of sexual inversion for hermaphrodite species. More precisely we are interested on the case where sexual inversion depends on the intraspecific competition between adults and juvenile. For that, we generalize a model of J. Mr. Cushing and Jia Li [3, 4] and we applies it to the case of the sexual inversion. In our case, we present and analyse a model for iteroparous hermaphrodite population, whose adults can survive by time and the transition between adult class is possible.

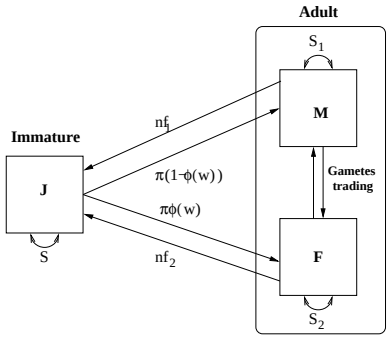

Figure 1: Simultaneous hermaphroditism.

We assume that hermaphrodites attempt to copulate in a gendre that optimises their fitness gain and their fertilization. Hence, when the expected male and female fitness gains diverge, we have an increasing preference bias towards the more profitable sex function.

Generally, an increase in male allocation, r, automatically decreases female allocation $(1-r)$ because both are drawn from a common resource pool [2, 1].

We will consider that each hermaphrodite individual of the population passes through two stages in his life : Immature : Juvenile and Adult.

At the moment of reproduction, the juvenile, who mature to the adulthood, is faced to a mating opportunity it needs to take a decision about its sexual status. It has the preference between two gender, female or male. Indeed, we assume that the mature juvenile will invest more initialy in the sex role profitable for all population and that generally offers the higher fertilization certainty.

let $\Phi(W)$ be the fraction of surviving juvenile that mature to the female class. This fraction depend upon the amount of competitive pressure experienced during the hormone juvenile growth, which is measured by, $W = J + \beta_1 M + \beta_2 F$, the total population density balanced by the competition. Where, J is the number of juveniles, M is the number of males and F is the number of females. Then $\beta_i \geq 0$ are the competition coefficient, that measure the pressure effect of the male or female presence on the sexual status choice of a juvenile.

$$\begin{cases} J(t+1) = nf_1 M(t) + nf_2 F(t) + sJ(t) \\ M(t+1) = \pi(1 - \Phi(W(t)))J(t) + s_1 M(t) \\ F(t+1) = \pi\Phi(W(t))J(t) + s_2 F(t) \end{cases}$$

An analytical study of the stability and robustness is made. We show that the model display a period doubling bifurcation as well as a chaotic region. This region deseppear with a high birth rate. The model is then generalize to the case where birth rate is density dependence, and we show that the density dependance stabilize the system.

References

[1] E. L Charnov. Simultaneous hermaphroditism and sexual selection. *Proc Natl Acad Sci U S A*, 76(5):2480–2484, May 1979.

[2] E. L. Charnov. *The theory of sex allocation.*, volume 18 of *Monogr. Popul. Biol.* Princeton University Press, november 1982.

[3] J. M. Cushing and Jia Li. *Individual Based Modeling: Concepts and Models*, chapter Chapter 6: The dynamics of a size-structured intraspecific competition model with density dependent juvenile growth rates, pages 112–125. Routledge, Chapman, and Hall, New York, 1992.

[4] J. M. Cushing and Jia Li. Intra-specific competition and density dependent juvenile growth. *Bulletin of Mathematical Biology*, 53(4):503–519, 1992.

A Convex Approach for Controlled Lotka Volterra Multi-Species Models

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Keywords: Multi-species models, Lotka Volterra systems, control systems, coexistence, LMIs.

Recently, the coexistence of the multi-species, where the models consider switching or not, are analyzed in the references [1, 2]. The latter results employ a traditional approach and the coexistence of the species is taken into account by means of rational functions (switching terms) as in the references [3, 4, 5]. Since Lotka Volterra systems have more than one equilibrium point, an estimate of the region in which the species coexist is an important issue not yet fully addressed. One can provide an estimate of the coexistence region through the Linear Matrix Inequality (LMI [6]) framework and a Differential-Algebraic representation (DAR) of the system [7, 8]. It turns out that Lotka Volterra systems with no switching have no stable equilibrium points. In this work, a control term to stabilized the system is imposed extending the result obtained in [2]. An example illustrates the proposed methodology, where a system with three species (two prey and one predator in a two trophic level food chain) is considered.

References

[1] S. Palomino Bean, A. C. S. Vilcarromero, O. Bonato, and J. Fernandes, Co-existência de espécies em sistemas presa-predador com switching [Coexistence of species in predator-prey systems with switching], Tendências em Matemática Aplicada, TEMA/SBMAC, vol. 7(2), 317–326, 2006 (in Portuguese).

[2] S. Palomino Bean and L. M. Porto, Coexistence on predator-prey multi-species with control, 4th Workshop in Mathematical Methods and Modeling of Biophysical Phenomena, (IMPA, Angra dos Reis, RJ), 2006.

[3] M. Tansky, Switching effect in prey-predator system, J. Theoretical Biology, vol. 70, 263–271, 1978.

[4] E. Kuno, Principles of predator-prey interaction in theoretical, experimental and natural population systems, Advances in Ecological Research, vol. 16, 249–337, 1987.

[5] M. Saleem, A. K. Tripathi, and A. H. Sadiyal, Coexistence of species in a defensive switching model, Mathematical Biosciencies, vol. 181, 145–164, 2003.

[6] S. Boyd, L. Ghaoui, E. Feron, and V. Balakrishnan, Linear matrix inequalities in systems and control theory. Philadelphia: SIAM Books, 1994.

[7] S. Palomino Bean, D. F. Coutinho, and K. A. Barbosa, Coexistence of Lotka Volterra Multi-species: an LMI Approach, 6th International Congress on Industrial and Applied Mathematics, (submited), 2007.

[8] D. F. Coutinho, C. E. de Souza, and A. Trofino, Regional stability analysis of implicit polynomial systems, Proc. 45th IEEE Conf. Decision and Control, (San Diego, CA), 2006.

Parameter identifiability and identification of a hierarchical epidemiological model

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Keywords: structural identifiability, parameter identification, PDE model, epidemiological model.

Our study is based on a PDE model which describes the dynamics of a scrapie outbreak in a sheep flock. Scrapie is a transmissible spongiform encephalopathy that affects sheep (prion disease), a fatal neuro-degenerative disorder characterised by a genetic susceptibility factor and a long incubation period, during which infected animals cannot be detected. The epidemiology of scrapie and particularly the transmission mechanisms are still incompletely understood. The confounding effects of incubation period, lifespan, age-dependent susceptibility and the changing force of infection make direct analyses of the case data difficult, hence the interest of a modelling approach.

The model we used was initially elaborated by Woolhouse et al. [1] and refined to fit the natural outbreak that occurred in the Langlade flock (INRA Toulouse), for which data are available [2].

The flock is structured according to scrapie status (susceptible or infected), PrP genotype (major susceptibility gene for scrapie), sheep age, and infection load. When susceptible sheep become infected, they are given a variable initial load, which is assumed to grow during the incubation period, until a fixed maximum value corresponding to the onset of clinical signs; animals are then culled.

To represent the outbreak, demographic and epidemiological processes need to be taken into account. The model incorporates the following components: seasonal breeding and routine culling, genetic susceptibility, a long and variable incubation period, and seasonal horizontal transmission. The available observations are the birth inflow, the routine culling outflow and the incidence (scrapie mortality) outflow, per genotype and age.

Our aim was to design a hierarchical method for the structural identifiability of the model parameters and their identification, so as to check the model parametrisation and consistency, given the available observations, and to perform accurate numerical simulations. Many methods have been developed to identify ODE model parameters, in the linear and non linear cases [3]. However, there are only few studies that consider PDE-based models.

To tackle this problem, we took advantage of the hyperbolic structure of the PDEs and built three overlapping models [4]. Changes of variables and an approximation due to cohort aggregation were used to transform the original problem into ODE subproblems. Moreover, the incidence observation was expressed as a function of the aggregated variables.

The parameter identifiability could then be addressed. We showed that demographic parameters can be easily obtained. Transmission parameters are identifiable, but the incubation parameters not necessarily, depending on the initial infection load function. Finally parameters were identified from the Langlade data.

References

[1] S. M. Stringer, N. Hunter and M. E. J. Woolhouse, A mathematical model of the dynamics of scrapie in a sheep flock. Mathematical Biosciences, 153(2):79–98, 1998.

[2] S. Touzeau, M. E. Chase-Topping, L. Matthews, D. Lajous, F. Eychenne, N. Hunter, J. D. Foster, G. Simm, J.-M. Elsen and M. E. J. Woolhouse, Modelling the spread of scrapie in a sheep flock: evidence for increased transmission during lambing seasons. Archives of Virology, 151(4):735–751, 2006.

[3] E. Walter and L. Pronzato, Identification of parametric models: from experimental data, Springer, 1997.

[4] B. Laroche and S. Touzeau, Parameter identification for a PDE model representing scrapie transmission in a sheep flock. In: 44th IEEE Conference on Decision and Control and European Control Conference, Sevilla, Spain, 2005.

Probabilistic cellular automata describing a predator-prey system and the dynamics of an epidemic

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Keywords: predator-prey; cellular automata; spatial models; stochastic dynamics; numerical simulations; men-field approximations; spreading of an epidemic.

We consider probabilistic cellular automata to describe two population dynamic processes where interacting individuals of different species or classes are spatially distributed. The individuals are discrete and localized over the sites of a lattice. The interactions between them are local and described by stochastic rules similar to the ones of the contact process. We set up pair mean-field approximations for the cellular automata and perform numerical simulations. We consider first the dynamics of a predator-prey system in which each site of a lattice can be occupied by individuals of each specie and the local predator-prey interactions are based on the processes of the Lotka-Volterra model. From the pair approximation we derive a quasi-spatial model which is able display stable self-sustained oscillations of the populational densities. The other population dynamic problem that we consider is the spreading of an epidemic in a community. In that case, the interacting individuals belong in different classes, susceptible (S), infective (I) and removed (R) and our probabilistic cellular automaton is defined by a set of local rules which incorporates the processes of the most simple SIR model. With the pair approximation for the cellular automaton we introduce here a SIR model which takes into account spatial correlations. With this model we deduce the epidemic threshold and epidemic curves

References

[1] E. Arashiro and T. Tomé, J. Phys. A 40, 887 (2007).
[2] K. C. de Carvalho and T. Tomé, Int. J. Mod. Phys. C 17, 1647 (2006).

New contributions to fish growth studies: from classical parameter estimation to new stochastic growth models

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Keywords: von Bertalanffy, individual based model, length-frequency analysis.

Growth studies are a fundamental tool in stock assessment and for the management of fisheries resources[1]. The **von Bertalanffy growth function (VBGF)** is almost universally used to describe growth in fish and in many other organisms, as its constants can be readily incorporated into early stock assessment models[2]. The VBGF has also been the conceptual reference for the development of tools aimed to estimate growth parameters and to detect the components of a population (i.e. the cohorts). Nevertheless, the deterministic nature of the VBGF represents a caveat[3]. Since the equation does not implement a model for growth variability, the observed pattern of growth can not be efficiently predicted. Moreover, the classical analyses are based on "*a priori*" assumptions, such as the normal distribution of cohorts and the monotone increase of variance. For these reasons, the algorithms for estimation of growth parameters (e.g. the ELEFAN I, the SLCA and the PROJMAT methods) and mixture decomposition (e.g. the Bhattacharya method, MIX and MULTIFAN) could lead to incorrect results, as they require a subjective inputs and arbitrary steps along the data analysis procedure. The aim of our research was an attempt to overcome some of the above-mentioned limits. A first work path was devoted to implement recent mathematical advances into data massaging procedures to give objectivity and reliability to the estimates. Two algorithms were tested. The first is a generalization of Akaike's theorem to choose the number of intervals to be used for building a regular histogram for a length-frequency analysis[4]. The second, the Expectation-Maximization algorithm[5], was used as an alternative density estimator with respect to the histogram used in the length-frequency analysis. Both algorithms improved the performance of estimates.

The second step of our research was devoted to overcome the theoretical limits of the VBGF. A number of models have been developed in the past with the aim to describe the growth pattern of a population by considering the growth of each individual[6-7]. Generally, these **individual based models (IBM)** introduce a stochastic part in the VBGF, which gives rise to the considerable differences in fish growth that are observed in the field. Nevertheless, all previous models present limitations, since they do not consider all the sources of variability or lack of properties that are desirable to model fish growth. We introduced two stochastic processes that obey to the following properties: (1) take into account all the sources of randomness in growth; (2) are always increasing and positive and (3) assume that the size average of the population is a function of time following the VBGF.

References

[1] A.A. Rosenberg and J.R. Beddington. Length-based methods of fish stock assessment. In: Fish Population Dynamics (ed. J.A. Gulland). John Wiley & Sons, London: 83-103, 1988.

[2] L. von Bertalanffy. A quantitative theory of organic growth (Inquiries on growth laws. II). Hum. Biol., 10: 181-213, 1938.

[3] V.J. Isaac. The accuracy of some length-based methods for fish population studies. ICLARM Tech. Rep., 27: 81 p, 1990.

[4] L. Birgé and Y. Rozenholc. How many bins should be put in a regular histogram. Thechnological report, Laboratoire Probabilités et Modèles Aléatoires, Univ. Pierre et Marie Curie, Paris, France, PMA-721, 2002.

[5] A.P. Dempster, N.M. Laird and D.B. Rubin. Maximum likelihood from incomplete data via the EM algorithm (with discussion). J. R. Stat. Soc. (B), 9: 1-38, 1977.

[6] Q. Lv and J.W. Pitchford. Stochastic von Bertalanffy models, with applications to fish recruitment. J. theor. Biol., 244: 640–655, 2007.

[7] K.J. Sainsbury. Effect of individual variability on the von Bertalanffy growth equation. Can. J. Fish. Aquat. Sci., 37(2): 241–247, 1980.

Modeling approach to explore influenza pandemic profiles and their associated interventions

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Keywords: epidemiological modeling, pandemic profile, metapopulation model, sensitivity analysis, clustering methods.

Recent modelling studies have focused on exploring the local and global spread of a potential influenza pandemic and the effect of interventions in reducing its burden. But little research has examined different potential profiles of spatial and temporal influenza pandemic diffusion, and how the impact of control strategies may depend on these profiles. In this study, we focus on these two questions using a modeling approach.

We used a refined version of a previously published deterministic compartmental model in discrete time [1-3], incorporating diffusion by air travel on a network of 52 world major cities, and six different intervention strategies to simulate spatial and temporal pandemic spread. Model analysis and simulation of different scenarios included three key components: (i) first, using the Latin Hypercube Sampling (LHS) scheme, the parameters related to the pandemic characteristics (transmission rate, etc) were sampled from appropriate distributions and 1,000 simulations using these inputs were performed; (ii) second, clustering methods were applied to the set of simulated dynamics to identify typical influenza pandemic profiles, and a multivariate Sensitivity Analysis (SA) on this same set of simulations, implying calculation of Partial Rank Correlation Coefficients (PRCC) between input parameters and model output criteria (global burden, etc), determined which input parameters had the greatest influence on the profiles; (iii) third, for two extremes pandemic profiles previously identified, parameters related to the control measures were sampled using the LHS method and the independent and relative effects of each intervention was assessed by calculating PRCCs. We considered significant PRCCs greater than 0.4 as indicating a moderate to important effect.

Five different groups of profiles that could occur in the absence of any control measures were identified and two of them were retained for further analysis: in the first one, 83% of individuals in the initial population would be susceptible resulting in a very short (52 days) and massive pandemic (close to 50% of people infected worldwide); conversely, the second profile, with 30% of initial susceptibles corresponds to a moderate (11% of people infected) and long lasting pandemic (more than one year). In the absence of any intervention, the two key parameters revealed by the SA were the rate of transmission and the proportion of susceptible individuals in the initial population (PRCC $\geq$ 0.74 with the pandemic final size). SA conducted on each of two extreme profiles showed that, regardless of the profile, restricting air travel was of little effect (no significant correlation with any of output variables), and early introduction of each control measure was the most important factor to reduce the number of people infected. Conversely, the effectiveness of other control measures seemed to vary depending on the pandemic profile. In the case of a short and massive pandemic, all intervention parameters played a clear role (with an emphasis on those related to antiviral prophylaxis, therapy and vaccination), whereas in case of a progressive and long lasting pandemic, only the date of interventions introduction was correlated with the outcome variables.

Our key findings concerning the great variation in possible profiles of a potential influenza pandemic and the dependence of the intervention efficacies on these profiles show that it is of critical importance to develop tools for early-stage identification of the pandemic profile, in order to adapt the public health response.

References

[1] L. Rvachev, I. M. Jr. Longini IM, A mathematical model for the global spread of influenza. Math Biosci 75, 3-22, 1985.

[2] R. F. Grais, J. H. Ellis, G. E. Glass, Assessing the impact of airline travel on the geographic spread of pandemic influenza. Eur J Epidemiol 18, 1065-72, 2003

[3] A. Flahault, E. Vergu, L. Coudeville, R. Grais, Strategies for containing a global influenza pandemic. Vaccine 24, 6751-5, 2006.

MODELLING OF A ONE-SEX AGE-STRUCTURED POPULATION

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Keywords: structured population, spatial difusion, child care.

Many species of animals produce a small number of osprings and take care of them. This phenomenon is natural for many species of mammals and birds and forms the main difference between the behavior of a population taking child care and that of a population without maternal (or parental) duties. Mammals and birds feed, warm, and defend their young offsprings from enemies. If one of these native duties is not realized, young offsprings die, and the population vanishes. For many species of mammals only females take care of their young offsprings. Some species of mammals and birds care of their offsprings in couples. In last years, to describe the population dynamics with child care some models were proposed and examined [1, 2, 3]. Numerical results of one-sex age-structured population model [4] taking into account a discrete set of offsprings, their care, and an environmental pressure will be discussed. All individuals have pre- reproductive, reproductive and post-reproductive age intervals. Individuals of pre-reproductive age are divided into young (under maternal care) and juvenile (who can live without maternal care but cannot reproduce offsprings) groups. Individuals of reproductive age are divided into the single and tak- ing child care classes. The model consists of integro-partial differential equations subject to conditions of integral type. The number of these equations depends on the biologically possible maximal new- borns number of the same generation produced by an individual. Both nondispersing and migrating population cases will be considered.

References

[1] V. Skakauskas, An age structured population dynamics model with females pregnancy and child care. Lith. Math. J. 44 (3), 251-315, 2004.

[2] V. Skakauskas, A two sex population dynamics model with strong parental care. Nonlinear analysis: RWA 6(4), 609-636, 2005.

[3] V. Skakauskas, A pair-formation model with a discret set of offsprings and child care. Lith. Math. J. 47 (1), 93-159, 2007.

[4] S. Repsys, V. Skakauskas, Modelling of a one-sex age structured population dynamics with child care. Nonlinear Analysis, Modelling and Control (Lith. Acad. Sci), 12 (11), 77-94, 2007.

The Multiple Faces and Feats of Diffusion Models in Biology: From Bets to Bits and Beyond

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Keywords: Diffusion processes, population dynamics

This conference is intended to present some fresh and innovative views and recent applications of an old subject. Diffusion processes in population dynamics is has being used for the description of transport, signaling, search, organization, and smoothing, among many other phenomena, from the molecular to the geographic level. Nowadays, also, diffusion models have become increasingly important as a flexible and efficient tool for the analysis of biological data, which is a quite opposite point of view to its classical uses.

Biological protection of plants — a model and its parameters

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Keywords: predator-prey model, stability, parameters estimation, plant protection.

A biological protection of plants against a pest consists in introduction a natural enemy of a pest into an agricultural site (field, greenhouse, orchard, vineyard etc.) According to the classical textbook [4], the dynamics of phytophagous population and predator substantially reducing its density can be modeled by the Gause-type predator-prey model

$$\dot{x} = rx - \varphi(x)y, \quad \dot{y} = -dy + \kappa\varphi(x)y,$$

where x and y denote the pest (prey) and its natural enemy (predator) population densities, respectively, r and d denote the prey growth and predator death rates, respectively, φ denote the functional response of predator to prey density (trophic function) and κ represents an effectiveness of conversion of the destroyed prey population into the predator population growth. The analysis of the system shows that there is one interior equilibrium point which may be locally asymptotically stable for the Holling-type-III functional response. The stability depends on particular shape of φ: the equilibrium point (x^*, y^*) is stable if $K_S^* = \varphi(x^*) - x^*\varphi'(x^*) < 0$.

From the point of view of plant protection, the key questions are whether the equilibrium is stable, i.e. whether the predator is able to reduce the pest population, and, if so, where is the equilibrium basin of attraction, i.e. which initial population sizes do not indicate a necessity of chemical treatment. To answer these questions, the particular form of the function φ should be chosen, the parameters of the model should be estimated and the identified model should be reliable, i.e. projections done by it should correspond to field data.

The parameters of a particular model can be estimated by methods of regression analysis, cf. [3], but a problem arises in the case of unknown analytical form of the trophic function. Even establishing the type of the functional response from field data is very often a hopeless task, cf. [2].

The aim of the contribution is to show that, in spite of the mentioned difficulties, the question on a population equilibrium stability can be answered in a sufficiently reliable way. Two methods of parameter estimation were considered and they were tested on simulated data. The provided simulations indicate that:

(i) the both methods uses to estimate the prey growth rate r correctly and with sufficiently small variance;

(ii) the predator death rate d uses to be estimated correctly but the variance of estimates is excessively large;

(iii) the precision of estimation the trophic function parameters do not allow to distinguish between different particular functions — the data fit for different choices of the function φ are equally good (or bad);

(iv) the criterion of stability K_S^* uses to have the correct sign independently on the choice of particular function φ and of particular method of estimation.

The result (iv) demonstrates that the attempt to estimate parameters of the considered model from field observations can support a decision to utilize a particular predator as a bioagens for plant protection.

The mentioned model had formed a theoretical basis for the method of integrated protection of grapes against phytophagous mites [1]. At that time, the model parameters had been estimated by a "semi by-eyes" method. Hence, the recent analyses complete the ones provided some twenty years ago and they represent late justification of South-Moravian vineyards protection method provided since early 90's.

References

[1] M. Hluchý and Z. Pospíšil, Damage and economic injury levels of eriophyid and tetranychid mites on grapes in Czechoslovakia. Exp.Appl.Acarol., 14, 95–106, 1992.

[2] H. McCallum, Population parameters: Estimation for ecological models, Blackwell Science, Oxford, 2000.

[3] K. Schittkowski, Numerical Data Fitting in Dynamical Systems, Kluwer Academic Publishers, Dordrecht, 2002.

[4] Ю. М. Свирежев, Д. О. Логофет, Устойчивость биологическич сообществ, Наука, Moscow, 1978.

Mean-field analysis of a replicator dynamics in a lattice

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Keywords: Replicator Dynamics; Spatial localization; Nonequilibrium Phase Transitions

The requisites for the persistence of small colonies of self-replicating molecules living in a two-dimensional lattice are investigated analytically using the independent site and the pair approximation [1]. The molecules are modeled by replicators A that are capable of replicating via binary fission $A + E \to 2A$ with production rate s, as well as via catalytically assisted replication $2A + E \to 3A$ with rate c. In addition, a molecule can degrade into its source materials E with rate γ. The stationary regime of this system is characterized by the presence (alive phase) and the absence (death phase) of replicators in the lattice.

We find that for small values of the ratio c/γ these phases are separated by a second-order phase transition, whereas for small values of s/γ the phase transition is of first order. Thus the phase diagram exhibits a tricritical point that separates these two types of phase transitions. In this contribution we expand and correct the results of a previous analysis of this model (see [2]) in order to avoid multiple occupance of empty cells surrounded by several particles [3]. In addition to the stationary properties of the lattice system, we investigate the stochastic dynamics of the replicators using the Gillespie algorithm [4], which allows us to obtain the (mean-field) dynamic critical exponents that characterize the time dependence of the average number of replicators as well as of the survival probability at the transition lines.

References

[1] D. A. Rand, Correlation equations and pair approximation for spatial ecologies, in: J. McGlade (Ed.), Advanced Ecological Theory, Blackwell Science, Oxford, pp. 100-142, 1999.

[2] C.P. Ferreira and J.F. Fontanari, Non-equilibrium phase transition in a model for the origin of life. Phys. Rev. E 65, 021902, 2002.

[3] M.A.M. de Aguiar, E.M. Rauch and Y. Bar-Yam, Mean Field Approximation To a Spatial Host-Pathogen Model. Phys. Rev. E 67, 047102, 2003.

[4] D. T. Gillespie, A General Method for Numerically Simulating the Stochastic Time Evolution of Coupled Chemical Reactions. J. Comp. Phys. 22, 403-434, 1976.

A Mathematical Model for the Dynamics of Rotavirus Infection

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Keywords: rotavirus, immune system, mathematical model, infection.

In infants and young children, rotavirus is the major cause of severe inflammation of the intestine (gastroenteritis). Rotavirus infection frequently results in fever, vomiting and diarrhea, wich symptoms are so intense that they can lead to death. This virus causes nearly a million deaths each year worldwide, mostly in developing countries.

Rotavirus attacks the epithelial cells of the thin intestine and replicates in the cytoplasm and do not fully uncoat during the process of replication. The reason for their failure to fully uncoat is that the coat is resistant to protease digestion, which prevents them from being completely destroyed by the infected cell and of readily being seen by the immune system.

This complex biology of rotavirus and its interaction with the immune system are the motivation of this work, that presents a model for this interaction, structured by non-linear ordinary differential equations of first-order that describes the action of the innate immune system to eliminate rotavirus. From this model, we find the trivial and non-trivial equilibrium points and analyze its stability.

References

[1] S. Barrozo, H.M Yang, Desenvolvimento de um modelo para resposta imunlógica primária célula-mediada, *Tendências em Matemática Aplicada e Computacional*, **7**, No. 1 (2006), 31-41.

[2] S. Barrozo, H.M Yang, Mecanismos da interação antígeno-anticorpo em uma resposta primária célula T-mediada, *Tendências em Matemática Aplicada e Computacional*, **7**, No. 1 (2006), 43-52.

[3] L. Edelstein-Keshet, Mathematical Models in Biology, *Birkhauser Mathematics Series*, 1988.

[4] S. Lauren, How Pathogenic Viruses Work, Janes and Bartlett. Canadá, 2002.

[5] J.P. Mascarenhas, Caracterizao molecular de genotipos de rotavrus do grupo A em Belm,Par, no perodo de 1990 a 2003: estudo em neonatos, crianas diarricas de 0 e 3 anos e ensaio clnico com vacinas, Tese (Doutorado em Biologia Parasitria). Rio de Janeiro: Instituto Oswaldo Cruz, 2006.

Population dynamics of a prey-predator model in a cassava system

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Keywords: stability analysis, functional response, *M. tanajoa*, *T. aripo*, biological control, distributed delay.

Phytophagous mites belonging to Tetranychidae family are one of the major constraints for cassava crop. Among them, the cassava green mite Mononychellus tanajoa is the most economically important in South America where it comes from, and in Africa where it was accidentally introduced in the 1970ies. In Brazil, M. Tanajoa is most abundant in the Northeast region and high yield losses (until 50% of dry matter) are reported. Ecological and biological studies suggested that phytoseiid mites would be the most appropriate predators to control M. Tanajoa.

In order to study the population's interactions, the stability of a predator-prey model was analyzed with special reference to cassava system where M. Tanajoa is the prey and Typhlodromalus aripo the predator. T. aripo, native from Brazil, is a phytoseiid frequently found in association with M. tanajoa in Brazilian cassava fields. Besides, in the context of M. tanajoa control programs, this predator was released and established with success in Africa. Our study consisted to analyze mathematically the effect of changing the functional response in the predator-prey Rosenzweig-MacArthur type model [5]: $dx/dt = rx(1-x/k) - ayf(x)$ and $dy/dt = -sy + byf(x)$, where x and y are the populations of prey and predator, respectively; a, b, r, s are constants; f(x) is the functional response. Three functional responses were chosen: Ivlev, Holling and Rosenzweig. Numerical simulations were done to show qualitative differences between the functional responses. Mathematical analyses of equations allowed interpretation of numerical results and exploration of structural stability. Using the Holling function the system showed high sensibility to the parameters used. Rosenzweig function also showed numerous oscillations and/or extinction of populations. From a mathematical point of view these two functions are inappropriate to represent interactions between predator and prey because the populations never reach equilibrium or extinguish. The Ivlev model showed little oscillations, stabilized very quickly and seems to be the best for representing interactions. This function was inserted in the distributed delay model with time varying to represent the population's development [1, 2, 3, 4].

References

[1] J. Bonato, O. These de Doctorat, Univ. Montpellier II, pp. 146 (1993).

[2] Vansickle, J. Attrition in distributed delay models, IEEE Transactions on systems man and cybernetics, vol. 7(9), pp.635-638 (1977).

[3] Manetsch, T. J. Time-varying distributed delays and their use in aggregative models of large systems, IEEE Transactions on systems man and cybernetics, pp.547-553 (1976).

[4] Severini, M.; Baumgärtner, J. and Ricci, M., Theory and practice of parameter estimation of distributed delay models for insect and plant phonologies, Meteoroly and environmental sciences: Proceedings of the course on Physical climatology and meteorology for environmental application, pp: 674-719, (1990).

[5] Holling, C.S. Mem. Entom. Soc. Can, 48, pp 86 (1966).

Agent-based Model for Migration of Individuals in Metapopulation

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Keywords: metapopulation, patch migration, population dynamics, agent-based model, Ricker equation.

The study of metapopulation dynamics generally deals with migration among patches in which resources are homogeneous and populational internal dynamics of such patches are modelled by using logistic equations. Usually, the rate of migration among patches is a fixed value and it does not take into account internal heterogeneity of individuals related to migration decision. Then, in this work, we propose an agent-based model for metapopulation migration among patches. In our approach, migration occurs according to non-coordinate individuals decisions of agents and we also consider that patches are heterogeneous with respect to resources.

Population dynamics of each patch will be modelled by the Ricker equation:

$$n_{t+1} = n_t \exp\left[r\left(\frac{1-n_t}{K}\right)\right], \qquad (1)$$

where n_t is population size at time t, r is the intrinsic growth rate of the population and K is the carrying capacity. In order to model heterogeneity of available resources, all patches p will have a random viability function $\mathcal{F}_p$ which can represent, for example, water, food or both.

As mentioned above, the decision of migrate or not will be taken individually. To simulate such a process, we split the agent-based migration process in two stages: potential migration and effective migration. In the first stage, a fraction a of individuals will be chosen randomly in their current patch to become potential migrants. All potential migrant i select randomly a potential patch for migration. Next, the potential migrant i will assess its satisfaction level by using equation:

$$S_i = \mathcal{F}_p - \Delta x, \qquad (2)$$

where Δx is the distance between the current patch and the potential patch considering periodic boundary conditions. The probability that an individual migrates to a patch randomly select previously is given by:

$$Pr_i = \frac{1}{1+e^{-S_i}}. \qquad (3)$$

In the second stage, effective migration, individuals will leave their current patch according to the probability calculated in equation (3). After migration process is complete, all patches will have their population updated according to their own dynamics by using equation (1). Whole process described before will be repeated according to the number of steps defined in the simulation.

The results of the simulations will be compared to those obtained in the models different from our approach. All relevant factors to the simulation will be checked such as: r, the intrinsic growth rate of the population in eq. (1), several functions for the patch viability $\mathcal{F}_p$ and the impact of other probability distribution in equation (3).

References

[1] Sugat Sutirth Dey, Amitabh Joshi Dabbholkllar, The effect of migration on metapopulation stability is qualitatively unaffected by demographic and spatial heterogeneity. J. Theor. Biol. 238, 78-84, 2006.

[2] J. J. Silveira, Aquino L. Espíndola, T. J. P. Penna, An agent-based model to rural-urban migration analysis. Physica A 364, 445-456, 2006.

[3] Ilkka Hanski, Juha Alho and Atte Moilanen, Estimating the parameters of survival and migration of individuals in metapopulations. Ecology 81(1), 239-251, 2000.

[4] Lenore Fahrig, Gray Merriam, Habitat connectivity and population survival. Ecology 66(6), 1762-1768, 1985.

[5] Larry S. Liebovitch and Ira B. Schwartz, Migration induced epidemics: Dynamics of flux-based multipatch models. Phys. Lett. A 332, 256-267, 2004.

Interactions between traps and other reservoirs in Aedes aegypti oviposition behavior

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Keywords: Individual-based modeling, Aedes aegypti, oviposition, control programs.

Though the efforts for controlling Aedes aegypti populations have been very intensive in the last years, many uncertainties are associated with field measurements of mosquito abundances, due to the erratic dynamics of both mosquitoes and oviposition sites availability. Questions such as the reliance of infestation indices under different rainy conditions and whether the impact of trapping on mosquito abundance is significant are yet to be answered. In order to address these questions, a simulation model in which individual female mosquitoes were allowed to live in a virtual space filled with oviposition traps as well as others oviposition sites was created. Aedes aegypti life-cycle processes as aging, daily motion, gonodotrophic state and their corresponding parameters were passed to mosquitoes individuals. Traps attractiveness was introduced as a traps parameter, which reflects mosquitoes relative preference for traps in comparison to natural oviposition sites. Each simulation last ten steps (approximately the number of days a trap is exposed), during which mosquitoes age, stochastically move and oviposit (depending on its gonodotrophic stage). Ten replicates were done for each set of parameters and number of captures in traps and in other sites were recorded. From these numbers, we calculated the Trap Oviposition Index (IPO), defined as the ratio between the number of traps containing eggs and the total of traps. Trap relative attractiveness showed to be important only at high density of oviposition sites. The reason for this is that if, during its oviposition behavior, a mosquito finds only one or few places to laid its eggs, the mosquito will oviposit there, no matter the places quality (traps cattractiviness, in the model). The odds that a mosquito will be able to choose between traps or natural reservoirs increases at high oviposition sites densities. As desired for an infestation index, we found that IPO increases linearly with mosquito abundance up to ca. IPO=80%. At high mosquito densities, IPO tend to saturate and loses its linear behavior. Presence of natural oviposition sites affected IPO negatively, resulting in a source of imprecision for mosquito abundance estimation based on traps. For example, in a scenario with 200 traps (50% attractiveness), with 200 natural breeding sites, we obtain an estimate of IPO 1% lower than in the absence of these breeding sites. In real situations, the abundance of natural breeding sites often exceeds in many figures the number of traps, so the expected error would be greater. This behavior of IPO must be considered when comparing areas with temporal or heterogeneous distribution of natural breeding sites (due to seasonal precipitation, for example). In areas/periods with abundant natural reservoirs, mosquito abundance would be underestimated. This implies that special care should be taken when using IPO in regions with marked rainy seasons, were reservoir abundance show wide variation, introducing uncertainties in the estimates. We suggest that, to be used for surveillance, covariates should be considered in the development of a trap based infestation index, based on the expected number of breeding sites and their response to rainfall.

A short history of mathematical population dynamics

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Keywords: History, mathematical models, pop- ulation dynamics.

This is a presentation of a book written in French on the history of mathematical population dynamics. The publication of the book is planned for the middle of the year 2007. It contains short biographies of most of the scientists mentioned, including pictures. It is also intended to be an introduction of the subject for students: the mathematical models are explained in some detail. Web sites from which some of the original papers can be downloaded are listed.

The book may also be of interest to professionals. A number of historical mistakes still circulate in the literature, e.g. on what Verhulst really did with his logistic equation. An English translation of the book is not yet scheduled. Comments for improving the content are welcome.

Table of contents (translated): Foreword The Fibonacci sequence (1202) Halleys life table (1693) Euler on the geometric growth of populations (1748) Eulers equation (1760) Daniel Bernoulli and smallpox inoculation (1760) Critic by dAlembert (1760) Süssmilch, Euler and the divine order (1761) Malthus on the obstacles to geometric growth (1798) Verhulst on the logistic equation (1838) Bienaym on the extinction of families (1845) Mendel on heredity (1865) Galton, Watson and the extinction of families (1873) The Hardy-Weinberg law (1908) Ross on malaria (1911) Fisher on natural selection (1922) Yule on evolution (1924) Lotka on physical biology (1925) McKendrick on epidemics (1926) Haldane on mutations (1927) The Wright-Fisher model (1930) Erlang, Steffensen and the extinction problem (1930) Volterra on the mathematical theory of the struggle for life (1931) The difusion of genes (1937) Lotka on demography (1939) The Leslie matrix (1945) Percolation and epi- demics (1957) Game theory and evolution (1973) Chaotic populations (1974) The one-child policy (1980) Some contemporary problems Bibliogra- phy.

References

[1] N. Bacaër, Histoires de mathématiques et de populations. To appear in 2007, Éditions Cassini, Paris, 225 pages, 10 Euros.

An environment for knowledge discovery on eukaryotic cell cycle and population dynamic regulations – a multiscale model

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Keywords: cell cycle, population dynamics, multiscale model, cancer.

Regulation of cell number and organ size is an important principle in biology. Experimental data in developmental biology indicate that there are mechanisms by which organisms sense their total mass and they are related to regulation of cell proliferation. In physiological conditions, the tissues are maintained in a dynamic equilibrium, called homeostasis, where the cell number is kept essentially constant and is regulated in function of production and death rates and half-life of cellular population. The cell cycle regulation is one of the central mechanisms of this homeostasis. Furthermore, resources supply and territorial space limit determine the population size, and hormones and components of sophisticated signal-transduction pathways regulate growth and cell division establishing a type of "social behavior". In this scenario, molecular alterations that disturb the homeostasis can be potentially dangerous. like in cancer, where the uncontrolled growth and spread of cells can affect almost any tissue of the body.

While modeling tissue and/or tumor growth is one of the most active areas of research within the Mathematical and Theoretical Biology community, much work remains to be done to create models that resemble physiological conditions, with predicted value. One obstacle that must be overcome is the intrinsic multiple scale nature of tissue growth. It involves processes occurring over a variety of time and length scales: from the tissue scale (for example, vascular re-modeling) to intra-cellular processes (for example, progression through the cell cycle of both normal and cancer cells). Most existing models focus on one scale. Whilst this may provide valuable insight into processes occurring at that scale, it does not address the fundamental problem of how phenomena at different scales are coupled.

Here, we present the initial results from a research we are carrying out with the aim of eventually formulating a multiple scale model of tissue growth capable of integrating a hierarchy of processes occurring at different scales. We expect to create an environment to design and simulate genetic and cell communication networks from available biological knowledge and data. The available information comes from molecular biology (genes, proteins, pathways and dynamical signal expression) and literature summaries data sets, besides expert knowledge. The control system that represent the cell cycle genetic networks will be expressed by a set of Boolean or differential equations that should be estimated from the available information. Cells will be represented by their control system model in the cell communication network. This environment will be applied to study the phenomena inside the cell and at the tissue level, in a physiological environment. After validation studies, the system control could be modified to simulate random mutations as occur in cancer. These studies may help to better understand issues related to cell proliferation that represent one of the most difficult challenges in cancer research.

Mathematical Model to study the spread of information from educative campaigns and rumors

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Keywords: Meme; Memetics; Epidemiological Models; Mathematical Models; Psychological Models; Information Exchange

The application of deterministic mathematical models to analyze the spread of ideas may represent an important tool to evaluate the efficacy of educative campaigns, especially in the health field. In this work we developed a mathematical model based on the mass-action principle, in analogy to the works that study the dynamics of infectious diseases in Epidemiology. We analyzed the dynamics of rumors spreading, taking into account the symmetry of contacts among "susceptible" and "infected" individuals and studied the implications of an educative advertising campaign in the model.

The analysis of the model shows that in the presence of educative campaign there is not a trivial equilibrium point and the non trivial equilibrium point is globally asymptotically stable. In this case the rumor improves the spread of ideas. On the other hand, the exclusion of the educative campaigns shows that the trivial equilibrium point is unstable and we studied the threshold values to the spread of the innovation only by the rumor.

Afterwards, we proposed a simplification of the model excluding a particular contact among "susceptible" and "infected" individuals. We present the analysis of the stability of equilibrium points and the threshold values to the spread of the innovation in both communication methods. The implications to the spread of rumor and its connection with parameters describing social behavior are discussed.

$$\frac{dS_1}{dt} = (1-p)\pi N - \lambda S_1 - \frac{\beta_1 S_1 I_1}{N} - \frac{\beta_3 S_1 I_2}{N} - \mu S_1$$
$$\frac{dS_2}{dt} = p\pi N - \lambda S_2 - \frac{\beta_4 S_2 I_2}{N} - \frac{\beta_2 S_2 I_1}{N} - \mu S_2$$
$$\frac{dI_1}{dt} = \lambda S_1 + \frac{\beta_1 S_1 I_1}{N} + \frac{\beta_3 S_1 I_2}{N} - (\gamma_1 + \mu) I_1$$
$$\frac{dI_2}{dt} = \lambda S_2 + \frac{\beta_4 S_2 I_2}{N} + \frac{\beta_2 S_2 I_1}{N} - (\gamma_2 + \mu) I_2$$
$$\frac{dR_1}{dt} = \gamma_1 I_1 - \mu R_1$$
$$\frac{dR_2}{dt} = \gamma_2 I_2 - \mu R_2$$

Figure 1: Ordinary Differential Equation System

References

[1] Massad E, Rocha AF, Lopez LF. Modeling the spread of memes: How units of knowledge are transferred from brain to brain. Journal of Information Science-submitted. 2006.

[2] S.M. Raimundo and H.M. Yang, Trasnmission of tuberculosis with exogenous re-infection and Endogenous Reativation, Mathematical Population Studies,13(4), pp 181-203, 2006.

A mathematical model to describe the *lethargic crab disease*

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Keywords: networks, *lethargic crab disease*, differential equations

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. For example, 38% of the households of 21 communities located around the estuary of the Caeté River (Pará State, North of Brazil) rely on collection and commercialization of *U. cordatus*. Beginning in 1988, massive mortalities of *U. cordatus*, such as 84% reduction in collection rates, have been report by crab-collectors. 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). Several potential etiological agents have been linked in unpublished acconts on LCD, including of exotic metazoans and chemical poisoning. Finally, in 2005, there were several evidences showing that LCD is caused by a fungus of phylum Ascomycota [1]. In this work we developed a mathematical model to describe the LCD disease transmission between the mangrove complexes. The dynamic system is given by the following differential equations:

$$\begin{aligned}
\frac{dA(t)}{dt} &= E(t) - \gamma A(t), \\
\frac{dS(t)}{dt} &= \gamma A(t)\left(1 - \frac{S(t)}{C}\right) - \mu_s S(t) - \\
&\qquad \beta(t)(F(t) - F_0)(S(t) - S_0), \\
\frac{dI(t)}{dt} &= \beta(t)(F(t) - F_0)(S(t) - S_0) - \mu I(t),
\end{aligned}$$

where

$$E(t) = \begin{cases} \phi S(t) + K, & t_1 \le t \le t_2 \\ 0, & 0 \le t < t_1 \text{ and } t > t_2, \end{cases}$$

$$\beta(t) = \begin{cases} \beta, \quad F(t) > F_0, \ S(t) > S_0, \ t_3 \le t \le t_4 \\ 0, \qquad\qquad\quad 0 \le t < t_3 \text{ e } t > t_4, \end{cases}$$

and $A(t), S(t), I(t)$ are, respectively, not susceptible, susceptible and infected population crabs. The $F(t)$ is related to the fungus population and is proportional to the dead crabs population caused by the disease.

To simulate the spread and evolution of the disease we constructed a network using the algoritm proposed by Watts-Strogats [2]. In each lattice site we observe the temporal evolution of the dynamic system proposed before. The links between the nodes promove fungus dispersion and is not static. Comparing simulated and experimental measures we are able to discuss several aspects of the disease transmission observed by the biologists and crab-collector communities.

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, Rio de Janeiro, vol. 100(2):161-167 (2005).

[2] D. J. Watts S. H. Strogatz Collective dynamics of small worldnetworks. Nature, 393: 440-442 (1998).

New Lyapunov functions for epidemic models with variable population size

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Keywords: Epidemic models, Lyapunov functions, $LaSalle's$ invariance principle, Global stability.

Abstract

In this work we deal with global stability properties of classic SIS, SIR and $SIRS$ epidemic models with constant recruitment rate and variable population size. For each model we analyze two cases according the incidence type: mass action law and homogeneous mixing. The usual approach to determine global stability of equilibria is the direct Lyapunov method which requires the construction of a function with specific properties. In this work we construct Lyapunov functions for the systems mentioned above using combinations of suitable logarithmic and quadratic functions.

References

[1] R. M. Anderson, R. M. May, Infectious Diseases of Humans: Dynamics and Control, Oxford University, Berlin, 1991.

[2] H. W. Hethcote, The Mathematics of Infectious Diseases. SIAM Review, 42(4), 599-653, 2000.

[3] A. Korobeinikov, G. C. Wake, Lyapunov functions and global stability for SIR, SIRS, and SIS epidemiological models. Applied Mathematics Letters, 15(8), 955-960, 2002.

[4] J. P. LaSalle, The Stability of Dynamical Systems, SIAM, Philadelphia, PA, 1976.

[5] J. Mena-Lorca, H. W. Hethcote, Dynamic models of infectious diseases as regulators of population sizes. Journal of Mathematical Biology, 30(7), 693-716, 1992.

Global stability of host-vector disease models via the Lyapunov method

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Abstract

In this work we deal with global stability properties of $host - vector$ disease models by construction of Lyapunov functions and $LaSalle's$ invariance principle. We prove that the global dynamics is completely determined by the basic reproduction number R_0. If $R_0 \leq 1$, the disease-free steady state is globally asymptotically stable. If $R_0 > 1$, a unique endemically infected steady state exists and is globally asymptotically stable in the interior of the feasible region.

References

[1] N. T. J. Bailey, The Mathematical Theory of Infectious Diseases, Griffin, London, 1975.

[2] C. Bowman, A. B. Gumel, P. van den Driessche, J. Wu, H. Zhu, A mathematical model for assessing control strategies againstWest Nile virus. Bulletin of Mathematical Biology, 67, 1107-1133, 2005.

[3] K. Dietz, Transmission and control of arbovirus diseases in: D. Ludwig *et al*. (Eds.), Epidemiology, Proceedings of the Society for Industrial and Applied Mathematics, Philadelphia, PA, 1974.

[4] L. Esteva, C. Vargas, Analysis of a Dengue disease transmission model. Mathematical Biosciences, 150, 131-151, 1998.

[5] L. Esteva, C. Vargas, A model for dengue disease with variable human population. Journal of Mathematical Biology, 38, 220-240, 1999.

[6] L. Esteva, C. Vargas, Influence of vertical and mechanical transmission on the dynamics of dengue disease. Mathematical Biosciences, 167, 51-64, 2000.

[7] L. Esteva, C. Vargas, Coexistence of different serotypes of dengue virus. Journal of Mathematical Biology, 46, 31-47, 2003.

[8] Z. Feng, J. X. VelascoHernández, Competitive exclusion in a vectorhost model for dengue fever. Journal of Mathematical Biology 35, 523-544, 1997.

[9] H. Guo, M. Y. Li, Global dynamics of a staged progression model for infectious diseases. Mathematical Biosciences and Engineering, 3, 513-525, 2006.

[10] A. Korobeinikov, G. C. Wake, Lyapunov functions and global stability for SIR, SIRS, and SIS epidemiological models. Applied Mathematics Letters, 15(8), 955-960, 2002.

[11] A. Korobeinikov, Lyapunov functions and global properties for SEIR and SEIS epidemic models. IMA Mathematical Medicine and Biology , 21, 75-83,2004.

[12] J. P. LaSalle, The Stability of Dynamical Systems, SIAM, Philadelphia, PA, 1976.

[13] M. A. Lewis, J. Renclawowicz, P. van den Driessche, M. Wonham, A comparison of continuous and discrete-time West Nile virus models. Bulletin of Mathematical Biology, 68, 491-510, 2006.

[14] C. McCluskey, Lyapunov functions for tuberculosis models with fast and slow progression. Mathematical Biosciences and Engineering, 3, 603-614, 2006.

[15] D. M. Thomas, B. Urena, A model describing the evolution of West Nile-like Encephalitis in New York City. Mathematical and Computer Modelling, 34, 771-781, 2001.

Small World Effect Using Cellular Automata: A Case Study for Epidemics

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Keywords: small world, cellular automata, epidemic.

One of the most important features to be concerned on the development of an epidemic model is the heterogeneity of interactions amongst population individuals. By the way, a very interesting model in terms of social interactions was introduced recently by Watts and Strogatz [1], trying to cover such heterogeneity of interactions. However, this model generally does it in a static way, with a fixed graph of interactions between individuals, which in the real life isn't so realistic by don't consider new friendships or enmity acquaintances.

In front of this scenario, we propose here an alternative model based on a probabilistic cellular automata [2] to obtain an equivalent behavior as that observed by Watts and Strogatz [1]. Particularly, the novel distinct feature of this alternative model (compared to the traditionally cellular automata used in epidemiology), is the use of two types of interactions between its individuals: the local interactions (performed by the neighborhood influence over each individual) and the global interactions, wherein all individuals have equal likelihood to establish contact with everyone of the grid and, by this way, reach further regions akin the small-world networks [1].

Thus, the probability of every individual becomes infected is modeled as the superposition of these two kinds of interactions through the following equation:

$$p_S = \Gamma p_G + \Lambda p_L \qquad (1)$$

in which $0 \le P_S \le 1$ and Γ and Λ are parameters utilized to tune the interactions of short (clustering formation) and long distance (of mean-field type) among individuals of the same population, like $\Gamma + \Lambda = 1$.

The global influence is modeled considering the total number of infective individuals in the grid, i.e. due the presence and mobility of every infective individual, which can be calculated as:

$$p_G = \frac{\rho}{N} \sum_{\{k,l\}} \delta_{i,\sigma(k,l)} \ , \qquad (2)$$

in which $0 \le \rho \le 1$ is a model parameter that limits the maximum value of p_G and is related with the intrinsic populational mobility. The local influence can be calculated through the neighborhood of a single individual and can comprise one or more infective individual. If this occurs, the probability of the local disease transmission can be calculated by the following equation:

$$p_L = 1 - (1 - \lambda)^n \ , \qquad (3)$$

in which $\lambda \in [0,1]$ and represents the probability of an individual become infected due the presence of *n* infective elements at his neighborhood in a SIR model, for instance

Therefore, through these two types of contacts described by Equations 2 and 3 we can adjust the mobility parameters Γ and Λ to transit from a traditional individual based model scenario (for $\Gamma = 0$) to a random scenario (for $\Gamma = 1$, where one individual can interact all the others as in a compartimental model which has no space), passing through another very interesting scenario, which both interaction approaches works together (for $0 < \Gamma < 1$).

In particular, this last scenario ascribes to the system a behavior akin such generated by the reconnection protocol of the small-world model [1] without, however, the need to discriminate those connections (due the probabilistic feature of the alternative model), which can change at any time.

References

[1] D. J. Watts, S. H. Strogatz, Collective Dynamics of 'Small-World' Networks, *Nature*, Vol. 393, 440-442, 1998.

[2] D. Alves, V. Hass, A. Caliri, The Predictive Power of R0 in an Epidemic Probabilistic Model, *Journal of Biological Physics*, Vol. 29, 2003.

Dynamics of the Interaction Between the Immune System and Measles Systemic Infection

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Keywords: Population Dynamics, Mathematical Modeling, Biomathematics, Measles.

The measles virus usually causes no serious disease symptoms until about ten days after infection. In this period the innate immune system deals very well with the infection, keeping the antigen concentration level low. However, the measles virus is able to get a productive infection of the dendritic cells, that after this, moves to nearby lymph nodes. These infected dendritic cells infect other cells of the lymph node. Viruses produced in the lymph node are then transported via lymph to the bloodstream. After this process, because of the ubiquity of the celular receptor for the measles virus, the viral infection rapidly spreads throughout the body. The virus then estabilishes a systemic infection.

In this work we develop and present a mathematical model to describe the population dynamics of the measles virus, host's cells and killer T cells during this period of the systemic infection. We intended to describe what happens after the productive infection of dendritic cells, which spreads the virus througout the whole body of the host with the help of the host's lymph node

The model consists of a system of ordinary differential equations with non-linearities. The variables considered are the concentrations of measles virus S, free (non-infected) host cells H, infected host cells H^*, killer T cells (non-activated) T_c and activated killer T cells T_c^*.

Once we have the model, we search for equilibrium points. Initially we verify the existence of one trivial equilibrium point, corresponding to the case where the concentration of the virus is null ($S = 0$). Let P_0 be that trivial equilibrium point, we have $P_0 = \left(0, \frac{k_H}{\mu_H}, 0, \frac{k_{T_c}}{\mu_{T_c}}, 0\right)$. It means that if the concentrations maintain its values at $S = 0$, $H = \frac{k_H}{\mu_H}$, $H^* = 0$, $T_c = \frac{k_{T_c}}{\mu_{T_c}}$ and $T_c^* = 0$, we will get all the derivatives in the model constants and equal zero.

The signal of the eigenvalues of the Jacobian matrix evaluated at P_0 would return us the kind of stability we have for this particular point. From the characteristic polynomial of that Jacobian matrix evaluated at P_0 we obtain the eigenvalues in terms of the parameters of the model. In order to get all these eigenvalues strictly lesser than zero, and hence assure the asymptotic stability of the trivial equilibrium point, we should impose that all the coefficients of the polynomial related to the eigenvalues obey the conditions of Routh-Hourwitz.

By calculating the variables H, H^*, T_c and T_c^* in terms of the variable S and the parameters of the model, we can also obtain the non-trivial equilibrium points. This non-trivial equilibrium corresponds to non-zero concentrations of measles virus ($S > 0$). They are obtained from a polynomial, whose coefficients can be described in terms of the parameters of the model. We also characterize this point with respect to its stability.

References

[1] S. Barrozo e H. M. Yang, Desenvolvimento de um Modelo para Resposta Imunológica Primária Célula-mediada, *TEMA Tend. Mat. Apl. Comput.*, **7**, No. 1 (2006), 31–41.

[2] S. Barrozo e H. M. Yang, Mecanismos da Interação Antígeno-anticorpo em uma Resposta Primária Célula T-mediada, *TEMA Tend. Mat. Apl. Comput.*, **7**, No. 1 (2006), 31–41.

[3] S. Barrozo, H. M. Yang e C. H. Dezotti, Uma Abordagem Matemática em Imunologia, *Notas em Matemática Aplicada: Matemática Aplicada à Fisiologia*, **7**, No. 1 (2003), 93–117.

[4] L. Edelstein Keshet, Mathematical Models in Biology, Soc. Industrial and App., 2005.

[5] J. D. Murray, Mathematical Biology, Springer Verlag, Berlin Heildelberg, 1989.

[6] L. Sompayrac, How Pathogenic Viruses Work, Jones and Bartlett Publishers, 2002.

A cellular automata model to describe the postfeeding larval dispersion in blowfly species

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Keywords: Chrysomia sp.; Cellular Automata; Larval Dispersion.

Blowflies use discrete and ephemeral substrate to place eggs and feed larvae. After the larval period, when each larva seeks to ingest the most food before the complete exhaustion of the resources, the larvae start looking for a site to pupate, or for additional food sources, if the larva has not reached the minimum weight for pupation. This process is called postfeeding larval dispersion. The study of the blowfly larva dispersion is very important for ecology (decomposition of animal organic substance), epidemiology (transmission of illnesses for animals and man) and in forensic entomology (interval post mortem).

Laboratory results of pupae distribution in the substrate indicate three important characteristics related to the larva migration: the number of pupae exponentially decreases in relation to food source; the interaction among the larvae can or cannot cause oscillation in the observed dispersion standard; interaction between species can change the aggregation standard of each one of the species. The experimental data have been collected at the department of parasitology of IBB/Unesp, Botucatu, for the postfeeding dispersion of the *Chrysomya alcebiceps* and *Chrysomya megacephala*, which are essential because they are bacteria and virus vectors, and responsible for myiasis in humans and other animals.

In this work, we aim to investigate the theoretical standards of blowfly larva migration using the formalism of cellular automata. The simulation results will be compared with the experimental data The results that we have simulated until now represent two important characteristics of pupae distribution in the substrate: the pupae number exponentially decreases in relation to the food source; the interaction among the larvae can or cannot cause oscillation in the observed dispersion standard.

References

[1] BASSANEZI, R. C. et al.; Diffusion model applied to postfeeding larval dispersal in blowflies (Diptera: Calliphoridae). Mem. Inst. Oswaldo Cruz, 92(2) 281-286, 1997.

[2] BOLDRINI, J. L. et al.; Non-local interactions and the dynamics of dispersal in immature insects. J. Theor. Biol. 185, 523-531, 1997.

[3] FELSENSTEIN, J.; Theoretical evolutionary genetics. Seattle: University of Washington Press, 1994.

[4] GAINES, S. D., BERTNESS, M.; The dynamics of juvenile dispersal: why field ecologists must integrate. Ecology 74: 2430-2435, 1993.

[5] GOMES, L.; GODOY, W. A. C.; VON ZUBEN, C. J.; A review of posfeeding larval dispersal in blowflies: implications for forensic entomology. Naturwissenschaften 93 207-215, 2006.

[6] REIGADA, C.; GODOY, W. A. C.; Dispersal and predation behavior in larvae of *Chrysomya albiceps* and *Chrysomya megacephala*. Journal of Insect Behavior, 18(4) 543-555, 2005.

[7] ROHANI, T; MIRAMONTES, O.; Immigration and the persistence of chaos in populaton models. J. Theor. Biol. 175:203-206,1995.

Conceptual spatio-temporal models of marine viral infections

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Keywords: Plankton, lytic viral infection, reaction-diffusion system, spatiotemporal structures, patchiness, multiplicative noise.

During the last two decades, marine biologists have gained deeper insights in the important role of viruses in the sea. However, the role of viruses is still far from being understood [1]. Thus, there is a need for conceptual models in order to explore the potentials of the dynamics.

[2], [3] and [4] investigated stochastic spatiotemporal prey-predator models of phyto- and zooplankton with infected phytoplankton. Infection processes were modelled by contacts of susceptible and infected phytoplankton rather than by explicitely accounting for viruses.

Recently, these models were extended by an additional equation describing virus dynamics. The basis of this new approach is the Beretta-Kuang model [5]:

$$\frac{dV}{dt} = -\lambda SV - m_V V + Bm_I I, \quad (1)$$

$$\frac{dS}{dt} = -\lambda SV + rS\left(1 - \frac{S+I}{K}\right), \quad (2)$$

$$\frac{dI}{dt} = \lambda SV - m_I I. \quad (3)$$

This ODE model which describes a viral infection of phytoplankton was extended by a predator (zooplankton). The extension which includes also diffusive movement of all species and stochastic environmental variabilty revealed several interesting pattern formation phenomena [6]. One effect is the displacement of a viral infection by an invading predator (cf. Fig. 1).

In (1-3), viral replication within host cells has not yet been considered. Recent results show that it is possible to model the complete life cycle of viruses — from intrusion into host cells via replication processes in the cell body to being set free as the host cell dies — with a surprisingly simple model.

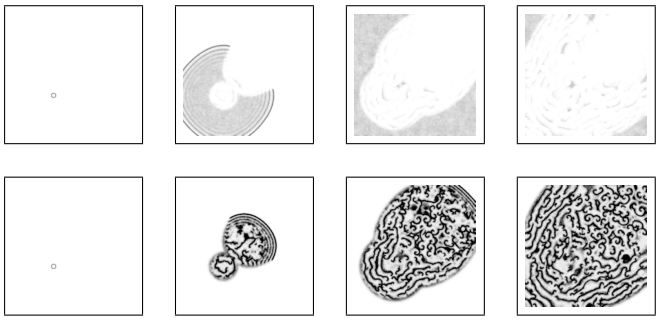

Figure 1: The infection is displaced by the expanding phytoplankton-zooplankton patch. Infected phytoplankton are displayed in upper, zooplankton in lower row. Initial conditions: One lower left patch with viruses, susceptibles and zooplankton, as well as one upper right patch with only susceptibles and zooplankton.

References

[1] Curtis A. Suttle. Viruses in the sea. *Nature*, 437:356–361, 2005.

[2] Horst Malchow, Frank M. Hilker, Sergei V. Petrovskii, and Klaus Brauer. Oscillations and waves in a virally infected plankton system. Part I: The lysogenic stage. *Ecological Complexity*, 1(3):211–223, 2004.

[3] Frank M. Hilker and Horst Malchow. Strange periodic attractors in a prey-predator system with infected prey. *Mathematical Population Studies*, 13(3):119–134, 2006.

[4] F. M. Hilker, H. Malchow, M. Langlais, and Sergei V. Petrovskii. Oscillations and waves in a virally infected plankton system. Part II: Transition from lysogeny to lysis. *Ecological Complexity*, 3(3):200–208, 2006.

[5] Edoardo Beretta and Yang Kuang. Modeling and analysis of a marine bacteriophage infection. *Mathematical Biosciences*, 149:57–76, 1998.

[6] I. Siekmann, H. Malchow, and E. Venturino. Predation may defeat spatial spread of infection. *Journal of Biological Dynamics*, submitted, 2007.

The Immune Response against HIV-1 as an Optimal Process.

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Keywords: HIV-1, Immune Response, Pontryagin Maximun Principle.

Mathematical models that describe the interaction of the HIV-1 virus with the host immune system have demonstrated to be a valuable tool of analysis of the dynamics of the infection.
These models depend on many parameters that are estimated from experimental data and/or adjusted to obtain consistent results with the clinical experience. These parameters represent, for example, the infection rate of healthy cells, the rate of production of specific cytotoxic lymphocytes, half-life of cells populations and viral copies, etc. There exists evidence that some of these parameters change their values during the typical course of the infection.

The aim of this work was to explore the variability of the parameter values that regulates the immune response against the infection as an optimal process.
We have performed the analysis using a three-state nonlinear deterministic model and The Pontryagin Maximum Principle.
The numerical simulations show that some aspects of the immune response against HIV-1 mediated by cytotoxic lymphocytes can be interpreted as an optimal process respect to some cost functional with biological meaning.

References

[1] A. Perelson, P. Nelson, Mathematical Analysis of HIV-1 Dynamics in Vivo, SIAM Review, Vol.41, No.1, pp. 3-44, 1999.

[2] M.A. Nowak, R.M. May, Virus Dynamics: Mathematical Principles of Immunology and Virology, Oxford University Press, New York, 2000.

[3] C. Hoffmann, B. Kamps, HIV Medicine 2006, Flying Publisher, 2006. Available: www.hivmedicine.com

[4] R.A. Arnaout, M.A Nowak, D. Wodarz: HIV-1 dynamics revisited: biphasic decay by cytotoxic T lymphocyte killing? Proc. Roy. Soc. Lond. B 265, 1347–1354 ,2000.

[5] R V. Culshaw , S. Ruan, R J. Spiteri, Optimal HIV treatment by maximising immune response, J. Math. Biol. 48, 545–562 ,2004.

[6] W. Fleming, R. Rishel, Deterministic and Stochastic Optimal Control, Springer-Verlag, NewYork, 1975

Modeling bovine brucellosis control by vaccination

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Keywords: bovine brucellosis, epidemiology, control, vaccination.

Bovine brucellosis is a chronic bacterial zoonosis, and its main etiological agent is *Brucella abortus*.. In cattle, brucellosis is mainly transmitted through exposure to an aborted fetus or other birth materials and fluids. Humans may also be infected with the bacteria when they consume unpasteurized milk or contaminated meat that has not been properly cooked. Bovine brucellosis presents a worldwide distribution, producing economic loss in livestock and dairy herds. Besides, it is a disease whose importance for the trade of animal products and by-products cannot be ignored.

In 2001, the Brazilian Ministry of Agriculture, Livestock and Food Supply initiated a National Program for the Control and Eradication of Brucellosis and Tuberculosis, whose aims are to decrease disease prevalence and to implement a certification process of disease-free farm premises. Vaccination of females is one of the measures that will be taken in this control program.

Females play a central role in the transmission process of brucellosis. To simulate the dynamics of brucellosis in cattle and the effects of a vaccination program, we developed a deterministic mathematical model, based on a set of compartments for the female population, namely: susceptible females, vaccinated females, latent carriers, and infectious females.

In a survey carried out in the State of São Paulo, Brazil, a seroprevalence of 3,8% was estimated for the females aged more than 24 months [1]. Taking this value as an initial prevalence, we simulated the effects of different proportions of vaccination coverage. The results are depicted in Figure 1.

To reach a prevalence level of 2%, for instance, it would take around 15 years, for a vaccination coverage of 80%.

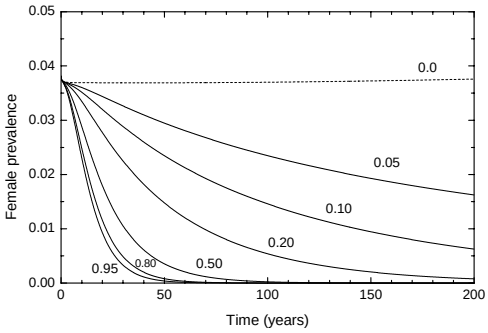

Figure 1 – Predicted female prevalence for different proportions of vaccination coverage.

We noticed in Figure 1 that the prevalence level decreases slowly over time, even for high vaccination coverages. Thus, for prevalences below a certain threshold (2%, for example), active measures of surveillance could contribute to the disease elimination.

References

[1] R. A. Dias, Caracterização espacial da brucelose bovina no Estado de São Paulo. University of São Paulo, 2004 [PhD Thesis].

Turing's instabilities and sinchronism in hierarchical models

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Keywords: metapopulation, stability, migration, transversal stability, hierarchy.

The effect of migration in population networks have been extensively studied (see [2], [3], [4]). In this context, we consider a metapopulation model of k species subjected to a density dependent migration. We investigate the transversal stability of a synchronized orbit and the Turing's instabilities associates to this system.

We have assumed that, to each generation, the local process of dynamics (reproduction and survival) precedes the migration process. As a consequence, the metapopulation model includes the composition of the migration processes with the reproduction and survival processes ($\mathbf{x}_j^{t+1} = \mathbf{f}(\mathbf{x}_j^t)$, $\mathbf{f}$ is a continuously differentiable map) and it is described by

$$\mathbf{x}_j^{t+1} = \mathbf{f}(\mathbf{x}_j^t) - \sum_{i=1}^{n} b_{ji}\Phi(\mathbf{f}(\mathbf{x}_i^t)),\ \forall j = 1, 2, ..., n, \quad (1)$$

where $\mathbf{x}_j = (x_{1j}, x_{2j}, ..., x_{kj}) \in \mathbb{R}^k$ is the population density in the patch j and x_{kj} is the amount of individuals of the species k in the patch j. The function $\Phi : \mathbb{R}^k \to \mathbb{R}^k$ is given by $\Phi(\mathbf{x_i}) = M(\mathbf{x_i})\mathbf{x_i}$, where $M(\mathbf{x}_i) = diag(\mu_1(\mathbf{x}_i), \mu_2(\mathbf{x}_i), ..., \mu_k(\mathbf{x}_i))$ is a matrix $k \times k$ and $\mu_i : \mathbb{R}^k \to \mathbb{R}$ is the migratory fraction of the species i.

The local dynamics has been given by a model with hierarchy ([1]): each species only depends on those that are above of its level, that is, f_1 depends on the specie 1, f_2 depends on species 1 and 2, and thus successively in such a way that f_i depends on i species. Some factors as the climate, the amount of resources for the survival, competitive abilities can characterize this hierarchic form. Moreover, this process of reproduction and survival is supposed stable and the migratory process μ_i depends only on the species i. In each generation, after the local dynamic process, a fraction μ_i of individuals of the species i leaves a given patch and goes to the others patches. Under these conditions we have established some analytical results on the occurrence of Turing's instabilities in the system, extending the studies of Silva et al. [3]. These results have been illustrated by means of numerical simulations.

We have also established the following analytical expression for the $(n-1)k$ transversal Liapunov numbers related to the system (1):

$$\widetilde{\Lambda}_i^j = \exp \int_{\mathbb{R}^k} \ln \left| 1 - \lambda_j \frac{\partial \phi_i}{\partial x_i}(\mathbf{x}) \right| \left| \frac{\partial f_i}{\partial x_i}(\mathbf{x}) \right| d\rho(\mathbf{x}), \quad (2)$$

for $1 \leq i \leq k$ and $1 \leq j \leq n-1$, where λ_j are the eigenvalues of the matrix $B = I - C$, and ρ is the natural probability measure of the system $\mathbf{x}_j^{t+1} = \mathbf{f}(\mathbf{x}_j^t)$ that is ergodic. As a consequence, if we define $\Lambda = \max_{i,j} \widetilde{\Lambda}_i^j$ for $1 \leq i \leq k$ and $1 \leq j \leq n-1$, an attractor of the system (1) lying on the invariant synchronized set is stable if $\Lambda < 1$. Our results of transversal stability are an extension of the works of Earn et al. [2] and Silva & Giordani [4], in the sense that both had considered a model of nets of populations connected to an only involved species.

References

[1] J. Best, C. Castillo-Chavez and A-A. Yakubu, Hierarchical Competition in Discrete Time Models with Dispersal. Fields Instituional of Communications, 36, 59-86, 2003.

[2] D. J. D. Earn, S. A. Levin and P. Rohani, Coherence and Conservation. Science, 290, 1360-1364, 2000.

[3] J. A. L. Silva, M. L. De Castro and D. A. R. Justo, Stability in a metapopulation model with density-dependent dispersal. Bull. Math. Biol., 63, 485-506, 2001.

[4] J. A. L. Silva & F. T. Giordani, Density-dependent Migration and Synchronism in Metapopulations. Bull. Math. Biol., 68, 451-465, 2006.

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A simple model for terraccumulation of antibiotics and the development of antimicrobial resistance in environmental bacterial populations

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Keywords: *antibiotics, terraccumulation, antimicrobial resistance, compartmental analysis, Caratheodory solutions*

In a recent review article, Rooklidge [2] identified diffusive pathways of antibiotics in the environment and described the effect of terraccumulation of antimicrobial contaminants on the development of antimicrobial resistance patterns in environmental bacterial populations. In the present paper we give a mathematical model for this theory that allows a dynamic study of this complex environmental-biological system.

Following Rooklidge [2], we compartmenalize the complex environmental system as follows: (i) human consumption, (ii) agriculture, (iii) aquaculture, (iv) wastewater treatment, (v) aquatic environment, (vi) landfills, (vii) water treatment, (viii) terraccumulation, cf also Fig. 1. Compartments (i) through (iii) are the source compartments. The model is formulated in terms of the dependent variables amount of antibiotics in the ith compartment c_i, unadapted (non-resistant) bacterial population x_i, and adapted (resistant) bacterial population y_i. Transport between compartments is assumed to be linear and donor controlled, while the reaction terms are nonlinear. This gives a system of 24 ordinary differential equations of the semi-linear form

$$\dot{z} = A(t)z + f(t,z) \qquad (1)$$

where A is block diagonal and the dependent variable is $z = (c_1, ..c_8, x_1, ...x_8, y_1, ..., y_8)^T$. The coefficients of A as well as f can depend directly on t in order to allow for seasonal or longterm fluctuations and variations. Temporal changes on time-scales shorter than t (e.g., abrupt changes) are admitted as long as the resulting coefficients are measurable in t (for fixed z, in the case of function f) . Thus (1) is understood as a differential equation in the sense of Caratheodory [1].

A quantitative study of model (1) is difficult, because reliable quantitative information about the occurring parameters and even input data is lacking. However, we are able to give a qualitative discussion. Under an additional simple, not too restrictive assumption, we can reduce the 24-dimensional system to a quasi-monotone 16-dimensional system. This enables us to obtain estimates of the solutions of (1) and discuss the longterm behavior. Moreover, this allows us to discuss how changes in the environment and changes in human behavior (both expressed in terms of modifications of coefficient and input functions) affect the development of antimicrobial resistance in the environmental bacterial population. Furthermore, we will give an outlook how this coarse first model can be refined.

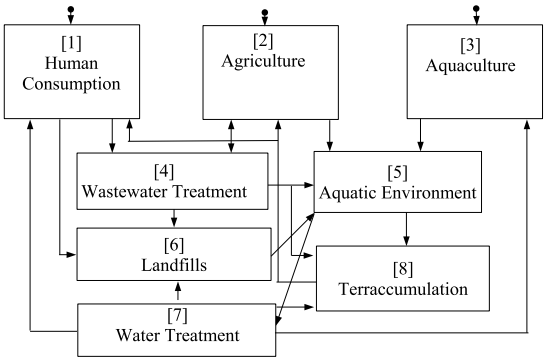

Figure 1: Pathways of environmental contamination with antibiotics, adapted from [2]. Arrows indicate the directions in which antibiotics are transported. Three source compartments are taken into account: agriculture, aquaculture and human consumption (administration of drugs, biocide usage).

References

[1] Caratheodory C. *Vorlesungen über reelle Funktionen*, Leipzig, 1918

[2] Rooklidge SJ. Environmental antimicrobial contamination from terraccumulation and diffuse pollution pathways, *Science of the Total Environment* 325:1-13, 204

An individual-based model for trophic interactions and species assembly

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Keywords: Individual-based model, Community dynamics, Species assembly.

The study of species assembly represents an important field for understanding community structure and functioning [1, 2]. Some simple mathematical models has been used for this task, producing important results about the relation between community-level features, like species richness, and community invasibility [3, 4]. Nevertheless, these models do not allow analysing the influence of some individual-level features, like body size and other life-history parameters. This last problem is very important when we are dealing with species invasions, which can represent a major threat to global biodiversity [2]. I developed an individual-based model (IBM) to simulate community dynamics and species assembly. At its present state, the model is oriented towards fish species, but it can be used to represent a variety of other heterotrophic organisms. In the simulations, every individual of a consumer species (fish) is followed along their entire life cicle. Each one has some key bionomic features that determine its reproductive strategies and the potential for interactions with other individuals and with basal resources, the last growing according to the logistic model. Among all features used, the body size is far the most important. Because it was framed as the determinant trophic niche axis, the body size drives the patterns of predation within communities, which incorporates some theoretical and empirical findings [5, 6]. By means of alometric relationships, it has also strong influence on the life history of model fish. Using the present model, invasion experiments were simulated to investigate how some individual and community-level features are related to the invader success and to its effects over the resident species. The baseline communities were set up by a standard assembly process, in which randomly selected species were introduced in the model system at regularly spaced time intervals. Later on, a factorial designed experiment of invasions was carried out, differing planned invader species by only two parameters of the predation window (the size range that caracterizes susceptible preys and resources), and differing resident communities by species richness and the number of preceding invasions during assembly. The species persistence during assembly processes was highly and negatively influenced by maximum body size. From the planed invasion experiments, the results point out that the time invaders remained in the communities until extinction depended only from their intrinsic caracteristics (those related to predation window), whereas the response of resident communities, measured as changes in species biomass and as percentage extinctions, depended only from their own features (species richness and assembly history). It is not possible yet to say whether the responses of communities were really influenced by the invaders, as none of these were able to survive the entire simulation time (50 years), and most of them went extinct in less than five years of simulation. So, their net effects over the resident species should be presumably minimal. More conclusive results require the continuity of simulation experiments.

References

[1] J. M. Diamond, Assembly of Species Communities. In: M. L. Cody, J. M. Diamond, Ecology and Evolution of Communities, Harvard Univ. Press, 1975.

[2] J. H. Brown, M. V. Lomolino, Biogeography, 2nd edition, Sinauer Associates, Inc., Sunderland, USA, 1998.

[3] J. A. Drake, The mechanics of community assembly and succession. J. of Theor. Biol. 147, 213-233, 1990.

[4] W. M. Post, S. L. Pimm, Community assembly and food web stability. Math. Biosc. 64, 169-192, 1992.

[5] R. J. Williams, N. D. Martinez, Simple rules yield complex food webs. Nature 404, 180-183, 2000.

[6] S. Jennings, J. K. Pinnegar, N. V. C. Polunin, T. W. Boon. Weak cross-species relationships between body size and trophic level belie powerful size-based trophic structuring in fish communities. J. of Animal Ecology 70, 934-944, 2001.

The stability of the Lotka Volterra system with uncertainty parameters - Monte Carlo simulation and Fuzzy sets approach

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Keywords: nonlinear dynamic systems, sensitivity analysis, system behavior, jacobian matrices.

The Lotka-Volterra predator prey model has been extensively used to describe the relationship between species, and, as a consequence, quite a variety of dynamical biological systems have been related to it. In this work, we present a formal sensitivity analysis for the Lotka-Volterra model with logistic growth of prey. The mathematical approach is designed to study the relationships between the parameters and the system behavior. The main idea is to study the effect of the random perturbations and fuzzy sets in the parameters on the stability. For this purpose, we made use of a Monte Carlo simulation - which generates random jacobian matrices and fuzzy sets techniques.

References

[1] S. H. Strogatz, Nonlinear Dynamics and Chaos. Westview Press, Cambridge Mass. 1994.
[2] Lotfi A. Zadeh Discussion: Probability Theory and Fuzzy Logic Are Complementary Rather Than Competitive Technometrics, 37-3, 271-276, 1995.
[3] R. Z. Khasminskii and F. C. Klebaner, The Annals of Applied Probability 11, 952 2001.
[4] M. F. Dimentberg, Physical Review E65, 2002.
[5] R. Z. Khaminskii, F. C. Klebaner and R. Liptser, Acta Applicandae Mathematicae 78, 201, 2003.
[6] Leah Edelstein-Keshet, Mathematical Models in Biology. SIAM, Random House, New York, 2005.

Epidemic Spread in Populations at Demographic Equilibrium

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Abstract. We introduce an integrodifference equation model to study the spatial spread of epidemics through populations with overlapping and non-overlapping epidemiological generations. Our focus is on the existence of travelling wave solutions and their minimum asymptotic speed of propagation c^*. We contrast the results here with similar work carried out in the context of ecological invasions. We illustrate the theoretical results numerically in the context of SI (susceptible-infected) and SIS (susceptible-infected-susceptible) epidemic models.

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Key words and phrases. discrete-time models, dispersal, epidemiology, integrodifference equations, travelling waves.

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Multiscale modelling for the virotherapy of avascular tumours

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Keywords: Cancer; Growth phenomena; Reaction-Diffusion model; Virotherapy.

Therapeutic oncolytic viruses (virotherapy) constitute a novel class of highly specific anticancer agents. Those viruses have been engineered to infect, replicate in, and kill malignant cells through diverse mechanisms. One of the benefits of oncolytic virus therapy is the therapeutic index, for every $10,000$ tumour cells killed by viruses, 1 normal cell is killed[1]. For comparison, the therapeutic index of chemotherapy is reported as 6:1[1]. Clinically-significant systemic antitumoural activity has now been demonstrated with oncolytic virotherapy. However, the virotherapy, as the most of the clinically used cancer therapies, have been developed empirically, and therefore mathematical models might be complementary (may be necessary) tools for the understanding of the dynamics of drug response in the organism

A multiscale model for virotherapy of cancer proposed by Ferreira et al.[2] is investigated and we present an extensive study of its complete parameter space, not reported before. This nutrient-limited model combines macroscopic reaction-diffusion equations, describing the nutrient and virus field concentrations, with microscopic stochastic rules governing the actions (division, infection, death by necrosis and by lysis) of individual tumour cells. Hence, the model involves three distinct time scales, the first one of these ($\sim$ 10 s) associated to nutrient diffusion, the second one ($\sim$ 30 min) related to virus diffusion, and the third one ($\sim$ 10 h) associated to cell dynamics. Virotherapy begins when the tumour attains N_0 cells and it consists of a single virus injection. In the intratumoral administration a uniform virus concentration over the entire tumour is supplied. The state diagrams in the parameter space were determined by specifying the dominant behaviour of the temporal evolution of the number of cancer cells after the viral injection, namely, successful therapy (tumour erradication), oscillatory response with increasing tumour cells and virus populations, oscillatory response with tumour control and unsuccessful therapy. These behaviors are shown in Fig.1. In these oscillatory behaviours, there is the coexistence of virus and tumor cells populations, and the oscillations are undamped, as those found by Wu et al.[3].

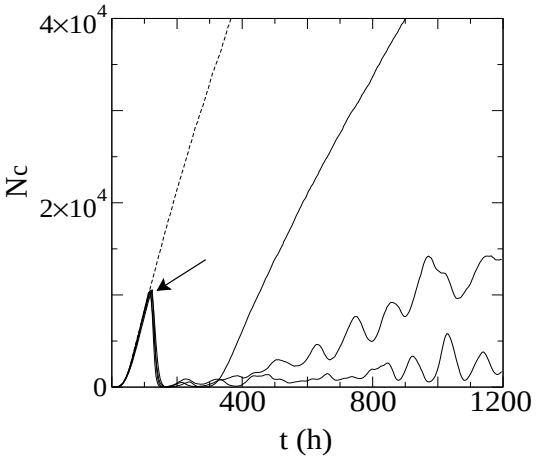

Figure 1: Temporal evolution of the number of cancer cells N_c with (solid lines) and without (dashed line) virotherapy.

These undamped oscillations are striking and novel results of this model and were identified in an ovarian cancer xenograft model by Peng et al.[4].

References

[1] J. A. M. Ring, Cytolytic viruses as potential anti-cancer agents. Journal of General Virology, 83:491-502, 2002

[2] S.C. Ferreira, Jr., M.L. Martins and M.J. Vilela. Fighting cancer with viruses. Physica A, 345:591-602, 2005

[3] J. T. Wu, H. M. Byrne, D. H. Kirn and L. M. Wein. Modeling and Analysis of a Virus that Replicates Selectively in Tumor Cells. Bulletin of Mathematical Biology, 63:731-768, 2001

[4] K.-W. Peng, E. M. Hadac, B. D. Anderson, R. Myers, M. Harvey, S. M. Greiner, D. Soeffker, M. J. Federspiel and S. J. Russel, Pharmacokinetics of oncolytic measles virotherapy: eventual equilibrium between virus and tumour in an ovarian cancer xenograft model. Cancer Gene Therapy, 13:732-738, 2006

Estimation of time lag in a reaction of self-regulative mechanisms: application of Hutchinson model for fitting of diamondback moth population dynamics

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Keywords: time series analysis, Hutchinson model.

For the description of diamondback moth (*Plutella xylostella*) population dynamics, determination of its type of fluctuations, and estimation of time lag in a reaction of self-regulative intra-population mechanisms on changing of population size, we used the well-known Hutchinson model [1]:

$$\frac{dx}{dt} = \lambda x(K - x(t-\tau)),$$

where $x(t)$ is a population size at moment t, λK is Malthusian parameter, K is carrying capacity, λ, $K = const > 0$, and τ is amount of time lag in a reaction of self-regulating mechanisms. Diamondback moth in Kenya has overlapping generations and continuous time in its development.

For considering model we assumed that initial function $\varphi(t) > 0$, $t \in [-\tau, 0]$, is a polynomial function of 6-th order, $\varphi(t) = P_n(t)$. Respectively we had 9 unknown model parameters, which must be determined at use of given sample of diamondback moth population fluctuation in some regions of Kenya.

Initially we had the sample $\{x_k = x(t_k)\}$, $t_k = 0,1,2...,N-1$, $N = 33$ (the difference between the time moments t_{k+1} and t_k is equal to two weeks) where x_k is population density at fixed time moment. For finding the values of model parameters we used the following statistical criterion (global fitting):

$$Q(\vec{\alpha}) = \sum_{k=0}^{N-1} \left(x_k - x(\vec{\alpha}, t_k)\right)^2 \to \min_{\vec{\alpha}}$$

where $x(\vec{\alpha}, t)$ is a solution of Hutchinson model for given vector $\vec{\alpha}$ of model parameters in a combination with parameters of initial function.

The use of this statistical criterion allowed us to find the best model parameters and, in particular, to determine the amount of time lag that was less than two weeks. Note that such estimation couldn't be obtained at the use of standard statistical methods (in particular, at the analysis of auto-correlation function behavior or its modifications [2,3]).

On the other hand, analysis of the behavior of residuals $\{x_k - x(\vec{\alpha}, t_k)\}$ showed that 1. the use of Kolmogorov-Smirnov test and Shapiro-Wilk's W test showed that there are no reasons for rejecting the hypothesis (with 1% confidence level) about normality of distribution of residuals, with zero average and respectively small sample variation; 2. the use of Darbin-Watson criterion showed the existence of negative serial correlation in sequence of residuals; 3. with 5% confidence level we can reject the hypotheses about equivalence of the values of auto-correlation function to zero.

Finally, results of analysis of residuals shows that we have to reject the hypothesis about possibility to use Hutchinson model for fitting the time series of diamondback moth population dynamics.

References

[1] G.E. Hutchinson, Theory of Competition Between two social species. Ecology, V. 28, 319-321, 1947

[2] P. Turchin, Rarity of density dependence or population regulation with lags? Nature, V. 344, 660-663, 1990

[3] P. Turchin, A.A. Berryman, Detecting cycles and delayed density dependence: a comment on Hunter and Price (1998). Ecological Entomology, V. 25,119-121, 2000

Modelling the Interaction between Trypanosoma cruzi and the Immune System

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Keywords: mathematical model, immune system, disease chagas

In this work, a mathematical model was developed to describe the action of the immune system taking into account the mechanisms of antibodies production by B cells, induced by the presence of the parasite in the organism, the parasite Trypanosoma cruzi and mediated by action of B cells. The Trypanosoma cruzi is a species of parasitic protozoan trypanosomes, etiologic agent of the disease of Chagas or American Tripanossomiase. This disease is common in the South America and Central America and south part of the United States. The disease of Chagas is a transmissible infection caused by a parasite that circulates in the peripheral blood and tissue, in which causes serious tecidual injuries mainly in the heart, as well as organs of the digestive system (esophagus and intestine).

The model is composed for a system of us nonlinear ordinary differential equations, which describes the interaction of B cells with the Trypanosoma.

The model analysis give us a trivial ateady state, which represents the elimination of the parasite, this means that the immune system goes to the steady state and is ready against new infection. If the infection coefficient is less than a threshold value, otherwise, it is unstable. The non-trivial steady state of the system was analysed without considering the antibodies action, and if the cloning rate is greater than threashold value, then the immune system is efficient and the disease is not establishid host organism.

Numerical simulations of the model have been carried out using package MATLAB@, using the Runge – Kutta of fourth order.

References

[1] S.Barrozo, H.M.Yang, Desenvolvimento de um Modelo para Resposta Imunológica Primária Célula-mediada. TEMA Tend Mat. Apl. Comput., 7, nº 1, (2006), 31-42.

[2] S.Barrozo, H.M. Yang, Mecanismos da Interação Antígeno-anticorpo em uma Resposta Primária Célula T-mediada. TEMA Tend Mat. Apl. Comput., 7, nº 1, (2006), 43-52.

[3] M.T. Quartieri. Estudo de modelos epidemiológicos determinísticos básicos em doenças causadas por microparasitas p 1-185, 2004.

[4] A.K. Abbas, A.H. Lichtman e J. S. Pober, "Imunologia Celular e Molecular", Revinter., 3ª Ed., Rio de Janeiro, 2000.

Semiparametric generalized additive model for ordinal response variables applied to epidemiology spatial data.

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Keywords: smoother; proportional odds model; spatial risk, surface smoothing.

The parametric models for ordinal response variables[1], like the proportional odds model (generalized linear model for ordinal response[2]), assume that the predictor is given as a linear form of covariates. In this work the parametric models were extended to a semiparametric form[4,5], where parts of covariates are modeled linearly and parts were modeled as smooth functions (generalized additive model[3]). The bivariate smooth functions were fitted to spatial variables estimating a surface smoothing. This model was applied to work accident risk in the informal worker market dataset in the urban zone of an industrialized city in Southeast Brazil. The mean idea is to examine concomitant effects of age, gender, type of occupation and years of schooling controlling for spatial risk variation in the gravity of the work accident – ordinal response variable. The design adopted is a population based case-control study focused on the spatial location of work.

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References

[1] A. Agresti, Categorical data analysis. Wiley, New York, 1990.

[2] P. McCullagh, Regression model for ordinal data (with discussion). J. Roy. Statist. Soc. Ser B 42, 109-127, 1980.

[3] T. Hastie and R. Tibshirani, Generalized additive model. Chapman and Hall, London, 1990.

[4] G. Tutz, Generalized semiparametrically structured ordinal models. Biometrics, 59, 263-273, 2003.

[5] G. Tutz, Sequential models in ordinal regression. Computational Statistics and Data Analysis, 11, 275-295, 1991.

Prey preference, food web structure and long term stability

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Keywords: Food web, prey preference, interaction strength, non-switching and switching predators, nutrient enrichment, stability.

Food webs are a central idea in ecology and an useful departure point for the development of a predictive theory of community ecology. Empirically ecological research has demonstrated that food webs may contain a vast array of species connected via multiple links of variable interaction strength [1]. Predation selection is commonly used in models to analyze the time issue of the relation among food web structure, diversity and stability [2, 3]. Within this context, this work investigates the role that the interaction strength and structure of prey preference play in the determination of some food web dynamics. Given the variety of these terms in ecological literature, this work shows the structure of prey selection may alter significantly the food web dynamics. This may serve as a caution with respect to the robustness of some results of food web theory.

References

[1] G. A. Polis and D. R. Strong, Food web complexity and community dynamics. The American Naturalist 147, 812-846, 1996.

[2] K. McCann, A. Hastings and G. Huxel, Weak trophic interactions and balance of nature. Nature 395, 794-798, 1998.

[3] M. D. Post, M.E. Conners and D.S. Goldberg, Prey preference by a top predator and the stability of linked food chains. Ecology 81, 8-14, 2000.

Dengue Epidemics: *Urbi et Orbi*

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Keywords: Dengue, *Aedes aegypti*. Dynamic Systems

Dengue is a viral disease which is transmitted by arthropods of the species *Aedes aegypti*, a mosquito found throughout the world where a hot and humid climate is predominant.Infectious individuals, either humans or mosquitoes, can start a dengue epidemic in human populations very quickly when placed in a previously *Aedes aegypti* infested region [2]. Dengue is a particularly serious public health problem in Brazil due to favourable climate and environmental conditions for the *Aedes aegypti* population expansion.

Since no vaccine for dengue is expected in the near future, any feasible strategy to control its epidemiological dynamics must concentrate efforts on the containment of *Aedes aegypti* population, especially when infection is detected [3]. So, to develop public policies for prevention and controlling strategies of this disease it is indispensable to obtain a solid and tractable knowledge about the *Aedes aegypti* population behavior and dynamics in order to find parameters appropriate to practical intervention. Mathematical models can give such knowledge since they are, by necessity, a reduced description of the reality, and if reasonably faithful, they authomatically yield the desired control parameters.

In this paper we develop a mathematical model for the Aedes aegypti spatial population dynamics coupled to dengue epidemics in a geographic network of human population clusters. The general model can be applied either to urban networks, where nodes are represented by well defined neighborhoods, or else to a spatially larger scale where nodes are represented by metropolitan areas.

Human visitation is an important factor in dengue epidemic dynamics and will be considered through a connection weight. The intensity of human visitation from residents of one node to the other will be measured by the road traffic between them. This is not a symmetric relation and a direction can be inserted toward the larger populated node, which is commonly the case, or toward nodes that have special features, like business centers, stadiums, schools, touristic spots and etc. On the other hand, the mosquito populations at each node are measured by their body presence. Since mosquito migration within the network, especially in a geographic context, depends strongly on human transportation, their flux between two nodes is scaled also according to the respective road traffic connection as in the case of human visitation.

At each node we consider three human subpopulations, susceptible, infected/infectious and recovered, and two mosquito subpopulations, susceptible and infected/infectious. A susceptible human individual can be infected only through a bite from an infected mosquito, and a susceptible mosquito can be infected only by biting an infected human and those are very sure to happen in a first encounter.

The general mathematical model is then applied to a case study of annual dengue epidemics which have happened in the São Paulo State, Brazil, for the last twenty years. Numerical simulations for the approximate period of one year are performed taking into account the state road network linking the 60 largest metropolitan areas of that region. The results compare reasonably well to the observed data of some episodes, especially those which began at far east cities of the state. Some crucial nodes are detected and control parameters are changed, mainly related to road connection and the mosquito carrying capacity of some nodes, in such a way as to delay the propagation of the epidemics. It should be pointed out that if a dengue epidemic does not reach a region before winter time (approximately from June through August) it will fade away because there is no vertical transmission to sustain it.

References

[1] L. Esteva, Influence of vertical and mechanical transmission on the dynamics of dengue disease, Mathematical Biosciences 167, 51-64, 2000.

[2] D.A.T. Cummings , R.A. Irizarry, N.E. Huang, T.P. Endy, A. Nisalak, K. Ungchusak and D.S. Burke, Travelling Waves in the occurrence of dengue haemorrhagic fever in Thailand, Nature 427, 344-347, 2004.

[3] D.J. Gubler, Dengue and Dengue Hemorrhagic Fever, Clinical Microb. Rev. 11, 480-496, 1998.

Prey-predator Modeling of CO_2 Atmospheric Concentration

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Keywords: CO_2 absorption, prey-predator model, photosynthesis rate.

Starting from a modified version of the Lotka-Volterra prey-predator equations, a model for predicting CO_2 atmospheric concentration, C_a, is presented. Vegetables are considered as predators and CO_2 as the inorganic prey. The interaction mechanism (average photosynthesis rate) will be taken into account using the formula given by Kirshbaum [1]:

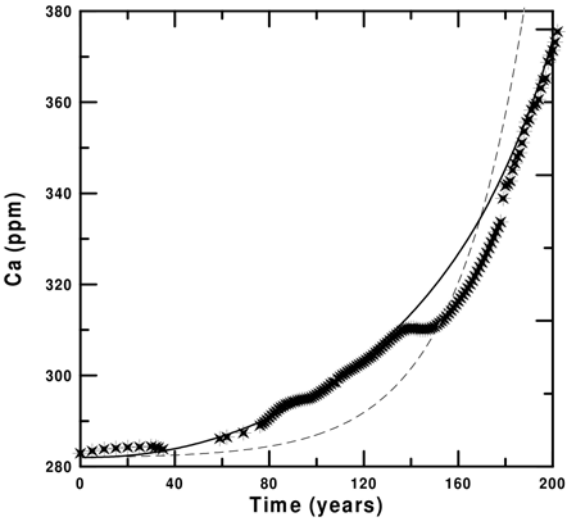
$$A(C_a,T)=\frac{C_a-1.5\,\Gamma}{C_a+3\,\Gamma} \quad ,$$

where A is the relative photosynthesis rate and Γ is a function of temeratue, T, which in the present work will be assumed equal to 25^0C in such a way that Γ=42. The time evolution of C_a and the average vegetable foliar surface participating in the process, P, will be described through the following modified prey-predator equations:

$$\frac{dC_a}{dt}=-A(C_a,T=25)\,P(t)+k\,Q(t) \quad ,$$

$$\frac{dP}{dt}=g\,A(C_a,T=25)\,P(t)-b\,P(t) \quad ,$$

where $Q(t)$ represents the total CO_2 emission (natural plus human), k measures its efficiency in increrasing C_a, b is a measure of vegetable foliar surface death rate and g is a parameter realting the efficiency with which the absorbed CO_2 is transformed into biomass. Under some specific conditions such system of equations can be in equilibrium and this will be used in estimating the various parameters before industrial revolution (using some historical data). Assuming an exponential behaviour for the growing of human emissions in $Q(t)$ and an adjustable vegetable death rate, it is possible to reasonable fit data for the growing behaviour of C_a., as it is shown in the figure. Historical data from [2], for the period 1800ac (Time=0) to 2000ac (Time=200), are represented with asterisks. The dashed line corresponds to solutions of the models assuming an exponential growing of human emissions, constant parameter g and a constant vegetable death rate. The filled line corresponds to an exponential growing of the vegetable death rate b, all the other



parameters unchanged. As it can be seen the agreement with historical data is better when an increasing vegetable death rate is considered. This is clearly related to the increase of human activities (urbanization, deflorestation etc.) and emissions (burning) and must be taken into account in any reasonable prediction model that tries to extrapolate present data to the near future.

References

[1] M.U.F. Kirshbaum, *Plant, Cell and Environment*, **17,** 747-754, 1994.
[2] H.Fischer, M.Wahlen, J.Smith., D.Mastroianni and B.Deck, *Science* , **283,** 1712-1714, 1999.

Bayesian Estimation of SEIR Models

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Keywords: Bayesian Inference; Estimation of SEIR models, Stochastic epidemic model.

Inference for epidemic models is complicated because some of the model variables may be only partially or even completely unobservable, and they can be observed at irregular intervals. Several methods can be found in the literature to estimate SEIR models. A survey of some of the proposed the methods can be found in Lekone and Finkenstädt (2006). Many of the methods use a bayesian approach in order to deal with the large number of parameters. In this work we propose the bayesian melding method proposed by Raftery *et al.* (1995) in order to estimate epidemic SEIR model. We apply the method to a model proposed by Raimundo *et al.* (2002, 2003) which describes the phenomenon of the interaction between HIV and MTB infection. The model is applied to data collected from the Female Penitentiary of São Paulo. The data were collected during the beginning of the experiment and after 6, 12 and 14 months. Raimundo *et al.* (2002, 2003) estimated the parameters and their standard error using the mean square method. They found some standard error which were remarkably low, especially considering the quality of the data. For some parameters our estimates were quite different and our estimated standard error were much larger than their estimates. The same type of difference is also found comparing the bayesian estimate of Lekone and Finkenstädt (2006) with the Chowell *et al.* (2004) minimum mean square error estimate. Chowell *et al.* and Lekone and Finkenstädt (2006) fitted a stochastic SEIR model to the 1995 Ebola outbreak in the Democratic Republic of Congo. We also proposed the use simulation in order to evaluate the likelihood.

References

[1] G. Chowell, N.W. Hengartner, C. Castillo-Chavez, P.W. Fenimore and J.M. Hyman, The basic reproductive number of Ebola and the e.ects of public health measures: The cases of Congo and Uganda. Journal of Theoretical Biology 229, 119126, 2004.

[2] P.E. Lekone and B.F. Finkenstädt, Statistical inference in a stochastic epidemic SEIR model with control intervention: Ebola as a case study. Biometrics 62, 1170-1177, 2006

[3] A.E. Raftery, G.H. Givens and J.E. Zeh, Inference from a deterministic population dynamics model for bowhead whales. Journal of the American Statistical Association 90, 402-416, 1995.

[4] S. M. RAIMUNDO, A. B. ENGEL, H. M. YANG and R. C. BASSANEZI, An approach to estimating the Transmission Coefficients for AIDS and for Tuberculosis. Systems Analysis Modelling Simulation 43, 423-442, 2003.

A fuzzy approach for a predator-prey model connected parasitism

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Keywords: fuzzy sets, fuzzy rule-based systems, parasitoid, aphids, ladybugs, Citrus Sudden Death.

This work describes a methodology based in fuzzy set theory to create a model that studies the interaction prey/host, predator and parasitoid. The aim of this investigation was to develop a simple and specific methodology to study Citrus Sudden Death (CSD). CSD is a disease that has affected sweet orange trees grafted on Rangpur lime in the south of the state of Minas Gerais and in the north of the state of São Paulo [3]. Some studies suggest that this disease has been caused by a virus transmitted by insects known as aphids (vector). Among the most known enemies of aphids in citrus in Brazil, it was selected the ladybug (predator) and a parasitoid ([1]). It was proposed the use of a fuzzy rule-based system ([4]), instead of usual differential equations which characterize the classic deterministic models. Since there is not enough information derived from the experimental data that can be related to the phenomenon, it is difficult to express the variations as functions of the states. On the other hand, qualitative information from specialists allows to propose rules that relate (at least partially), the state variables, with their own variations. The variables of the system are number of preys, number of parasitoids and potentiality of the predators (inputs), and their variations (outputs) ([2]). Simulations were performed and the graph shown prey population, potentiality of the predators, and the parasitoid population, over time. The rules are of the following form: "If the number of aphids is large and the potential of predation is very small, then the variation of aphids
is positive small and the variation of the potential of predation is positive large; If the number of aphids is small and the number of parasitoids is small, then the variation of aphids is positive small and the variation of parasitoids is negative large".

References

[1] J. R. P. Parra, V. Oliveira, A. S. Pinto, Guia Ilustrado de Pragas e Insetos benéficos dos citros, Esalq-USP, Piracicaba, 2003.

[2] M. S. Peixoto, Sistemas Dinâmicos e Controladores Fuzzy: um Estudo da Dispersão da Morte Súbita dos Citros em São Paulo, Ph. D. Thesis, IMECC-UNICAMP, Campinas/SP, Brazil, 2005.

[3] R. B. Bassanezi, A. Bergamin, L. Amorim, N. Gimenes-Fernandes, T. R. Gottwald and J. M. Bové, Spatial and temporal analyses of Citrus Sudden Death as a tool to generate hypotheses concerning its etiology. Phytopathology, 93:502-512, 2003.

[4] W. Pedrycz and F. Gomide, An Introduction to Fuzzy Sets: Analysis and Design, Massachusetts Institute of Technology, 1998.

A dynamic model to evaluate antiretroviral therapy for HIV type 1

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Keywords: Mathematical epidemiology - Infectious disease - HIV.

HIV type 1, the main responsible for AIDS pandemic, is a retrovirus with high mutation rate and great capacity of replication. Antiretroviral drugs have been described since eighty decade and the most used in Brazil are inhibitors of two important enzymes that act in viral replication known as reverse transcriptase (RT) and protease. These drugs belong to three different classes: nucleoside analogue reverse transcriptase inhibitors (NRTI), non-nucleoside analogue reverse transcriptase inhibitors (NNRTI) and protease inhibitors (PI). For the initial therapy, it is recommended the use of a combination of three drugs from two different classes: two NRTI and one NNRTI or two NRTI and one PI.

The main target of antiretroviral therapy is to delay the advance of immunological deficiency and to restore immunity by viral suppression. Two parameters are used to determine the need of therapy: viral load and CD4+ T cell count. Besides, patient clinical signals are also important for this decision.

Studies indicate that 10 to 20% of patients that begin treatment do not achieve satisfactory viremia suppression after a few months of therapy (primary virological fail) and 20 to 50% of those that present good initial response will present virological fail after one year of treatment (secondary virological fail). One of the main causes for virological fail is the incomplete adherence to treatment which may cause the selection of resistant virus strains.

This work proposes a mathematical model to describe the dynamic of transmission of HIV-1 initial infection. The model considers that after the beginning of treatment the situation of the patient may evolve to success (satisfactory virus suppression) or virological fail. The infected individuals who have treatment success for a period of time may present virological fail after a year (secondary fail), and the individuals in virological fail are infected with resistant strains.

References

[1] Lopez, L.F.; Burattini, M.N.; \& Massad, E. Modelling the natural history of the HIV infection and its epidemiological implications 2001. Bulletin of Mathematical Biology 63(6): 1041-10625.

[2] Raimundo, S.M.; Yang, H.M.; Engel, A.B.and Bassanezi, R.C. An approach to estimating the Transmission Coefficients for AIDS and for Tuberculosis. Systems Analysis Modelling Simulation, vol.43, No 4, 423-442, 2003.

Assessing the effects of BCG vaccine on the transmission of Tuberculosis

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Keywords: Tuberculosis; Vaccination; Basic reproduction number; Infectious Diseases; Epidemic Model.

Tuberculosis *(TB)* is an infectious disease caused by the *Mycobacterium tuberculosis* and the second most common infectious disease worldwide. Nine million new cases of *TB* are estimated to occur around the world every year. Approximately one-third of the worlds population is estimated to be infected with *M.tuberculosis*, resulting in nearly 3 million deaths each year. The vaccine *Bacille Calmette - Guerin (BCG)* is the most widely used vaccine in the world, however, its efficacy in protecting against tuberculosis remains controversial.

In this work we developed an *SIV* mathematical model to describe the dynamics of *TB* under the assumption that the vaccine efficacy is not 100% and may wane with time. The interplay of the vaccination strategy together with the vaccine efficacy and waning is studied. Disease transmission is assumed to be of standard incidence, so that the number of infectives is produced by contacts between *TB* infectious *(I)* and S susceptible individuals. Disease immunity is induced by vaccination, but those successfully vaccinated may be only partially protected from infection, resulting in infected individuals coming from the vaccinated class. For simplicity, it is assumed that if an individual in the V class is infected, then that individual is equally as infectious as an individual infected from the S class. The model is described by nonlinear ordinary differential equations. Qualitative analysis of the model shows that it has a unique disease-free equilibrium which is locally asymptotically stable if the basic reproduction number as modified by vaccination, namely (R_{vac}) is less than one. The study of the existence and stability of endemic equilibria of the model is to addresses the question of whether vaccination could ever completely stop the spread of infection in a population. The model shows that eradication depends on vaccine efficacy as well as vaccination rate.

References

[1] Moghadas, S.M. , Modelling The Effect Of Imperfect Vaccines On Disease Epidemiology, Discrete And Continuous Dynamical Systems Series B, Vol 4, Number 4 (2004).

[2] S.M. Raimundo and H.M. Yang, Transmission of tuberculosis with exogenous re-infection and Endogenous Reativation, Mathematical Population Studies,13(4), pp 181-203, 2006.

Modeling dog population control

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Keywords: dog population, control, euthanasia, sterilization.

In many countries, a major public health problem is the increasing number of abandoned dogs. Sterilization of owned dogs and euthanasia of healthy unadopted dogs in animal shelters are examples of measures taken to solve the unwanted dogs surplus problem and the consequent abandonment of dogs to shelters or streets. Educational campaigns may also be used as strategies to avoid abandonment of dogs in the streets. It is difficult, however, to estimate the effectiveness of such strategies in reducing stray dog population, especially in short time periods.

A central question is whether sterilization and/or euthanasia or other methods may be effective to control stray dog populations. The answer is obviously not straightforward, and it depends on cultural, social, economic, and behavioral factors related to responsible pet ownership.

We elaborated mathematical models to simulate dog population dynamics and also the effects of population control measures [1]. Our intention is to analyse how effective sterilization and euthanasia of animals can be in reducing stray dog populations, studying different scenarios with abandonment of dogs in streets. We simulated the results of control strategies for different values of sterilization and euthanasia rates.

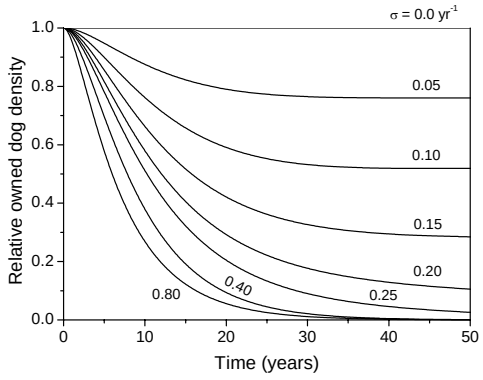

Figure 1 – Predicted relative owned dog density for different sterilization rates (σ), for a population with a rate of increase of 0.209 yr^{-1}.

In Figure 1, we show the predicted relative density (density divided by the carrying capacity) of a closed owned dog population taking into account the effect of different sterilization rates (σ). We notice that for time periods shorter than 5 years, the reduction in population density is smaller than 20%.

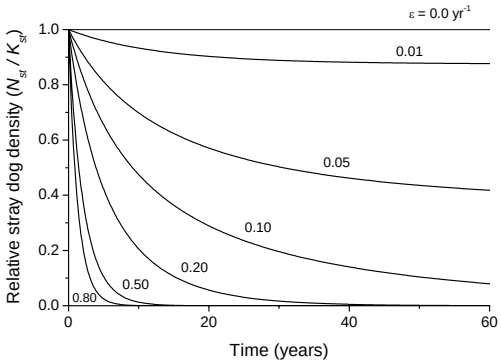

Figure 2 – Stray dog population density for different euthanasia rates, for a population with a rate of increase of 0.157 yr^{-1}.

In Figure 2, we show the relative stray dog density of a closed stray dog population for different values of euthanasia rates (ε). It is not possible to establish a direct comparison between the results of Figures 1 and 2, because they refer to populations with different rates of increase.

We intend to estimate the impact of abandonment on stray dog population density, and also to compare the effects of euthanasia and sterilization.

References

[1] M. Amaku, R. A. Dias, F. Ferreira. Modelling dog population dynamics and control. In preparation.

Approximate reduction of non-linear discrete models with two time scales.

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Keywords: Nonlinear discrete models; aggregation of variables; time scales; population dynamics.

The aim of this work is to present a general class of nonlinear discrete time models with two time scales which dynamics is susceptible of being approached by means of a reduced system. The reduction process is included in the so-called approximate aggregation of variables methods 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. For the time unit of the discrete system we use that of the slow dynamics and assume that fast dynamics acts a large number of times during it. Assuming that fast dynamics instantaneously attaints a certain equilibrium we build up the aggregated system and it is proved that certain asymptotic behaviours, hyperbolic asymptotically stable periodic solutions, to the aggregated system entail that to the original system.

A frequent 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. To illustrate the proposed reduction method we present several examples of this kind considering, in particular, host-parasitoid communities inhabiting heterogeneous environments with the migration process described by means of a matrix dependent either on the global variables or on a part of the state variables.

A Reliability Analysis of The Emergency Diesel Generators of a Four Loops PWR Nuclear Power Plant Under Aging and Perfect Repair by McKendrick Equations

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Keywords: Reliability, Numerical Analysis, Mckendrick Equations..

The aim. of this paper is to calculate the failure probability of the emergency diesel generators of a 4 loops nuclear power plant considering aging effects and perfect repair. Because of this 12 Mckendrick equations for modeling this problem has appeared. In this paper discontinuous repair rates were considered and because of this a new powerfull numerical method was created : Euler Iterative + Characteristic, using a part of the Analytical solution [1].

References

[1] I. M. S. Oliveira, Confiabilidade dos Geradores Diesel de Emergncia de Uma Central Nuclear PWR de Quatro Loops sob Envelhaecimento e com Reparo Perfeito. Dissertao de Mestrado PEN/Coppe/UFRJ, 2007.

A stochastic simulation of a yellow fever outbreak (Buenos Aires, 1871)

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Keywords: yellow fever, mathematical model, eco-epidemiology

Yellow fever is one of the most important arthropod-borne viral diseases. It has caused major epidemics in the Americas in the 17th, 18th, 19th and the 20th century. The yellow fever virus is mainly transmitted by the highly domesticated *Aedes aegypti* mosquito, which is distributed in the Americas from the south of North America to the Buenos Aires province and the north of the La Pampa province in Argentina. Today, the Word Health Organization informs that nine South America countries and several Caribbean islands are endemic for this disease, and estimates that there are globally 200.000 cases with 30.000 deaths per year. *Aedes aegypti* is currently a permanent inhabitant of Buenos Aires city. In the past, the presence of this mosquito, in Buenos Aires city, was evidenced by several epidemic outbreaks of yellow fever initiated in the past centuries, however, the most important one happened in 1871. Mortality began by the end of January and the last cases were reported in June of the same year. The outbreak of 1871 killed as many as 13.700 people in Buenos Aires city where the population was almost 190.000.

As part of our ongoing research on mathematical eco-epidemiology we are developing a model for a yellow fever outbreak. The validation of the model will be performed against data reported from the outbreak in Buenos Aires, 1871. We will show the design of the model and the biological processes involved, based on previous models for *Aedes Aegypti* population dynamics [1,2]. We will also present the spatio-temporal data corresponding to the population of Buenos Aires in 1871 as well as weather data (input data) and the reporting of the epidemic outbreak (our target for the simulations). We will analyze the epidemics spreading, considering the events that could also contribute to the outbreak, such as the absence of drinking water distribution systems and the density of the breeding sites. Early results will be shown.

References

[1] M. Otero, N. Schweigmann, H. Solari. A stochastic spatial dynamical model for *Aedes aegypti*. Submitted to the Bulletin of Mathematical Biology.

[2] M. Otero, H. G. Solari, and N. Schweigmann. A stochastic population dynamics model for Aedes aegypti: formulation and application to a city with temperate climate. Bulletin of Mathematical Biology 68 (8), 1945-1974, 2006.

Fuzzy Clustering and Allometry to Freshwater Stingrays of the Rio Negro Basin

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Keywords: Fuzzy Clustering, Fuzzy Relations, Stingrays, polychromatism,potamotrygonidae

Abstract

Elasmobranchs completely restricted to freshwater environments are represented by myliobatoid stingrays, of which two-thirds are potamotrygonids endemic to neotropical region of South America [1]. Freshwater stingray exhibit a high polycromatism, which results in misidentifications [2]. The develpoment of conservation program for these species is frustrated by unresolved taxonomic problems. Improved assessment of ptamotrygonids stocks requires a much better understanding of these taxonomic problems.

"One of the main characteristics of the human thought is the development with factors as vacant ambiguity, uncertainty and information in the resolution of problems of difficult solution"[3].

The diagnosis here presented is an application of the Method of Classification and recognition of Padrões Fuzzy based on Fuzzy Relations. For this application we based on the works of L.A. Zadeh [4] e Shinichi Tamura [5]. The Fuzzy Clustering is described for evaluation of the association degree among species of rays of the Família Potamotrygonidae. For analysis of the data we still used the software HCE3 (Hierarchical Clustering Explorer 3.5) developed by Jinwook Seo of the University of Maryland. The objective is to identify the existence of homogeneous classrooms among individuals of the Potamotrygonidae family. The Fuzzy Clustering is a used method for the construction of groups of objects on the basis of the similarities and differences between the same ones of form that the gotten groups are most homogeneous and well separate possible. The partition of resultant data improves the agreement and discloses its structure internal. The method used for the application is based on Fuzzy Relations. In the first step we determine the vector standard that expresses the characteristic variable of the species. We do not work here with all the measures that are established for the classification. Our choice happens in the main measures for the characterization of the species in practical of field during the activity of the capture of the rays. We determine a fuzzy compatibility relation in terms of a function distance of the classroom of Minkowisky applied to the data.

$$R(a_i, a_k) = 1 - \lambda \left(\sum_{j=1}^{p} \left| a_{ij} - a_{kj} \right|^q \right)^{\frac{1}{q}}$$

Then a similarity between two any standards are calculated through the composition of the fuzzy compatibility relation defined. Of this form we group the population of patterns of the species in classrooms for the equivalence relation or similarity. We note correlations among the selected variables main: disk width (cm), total length (cm) and two denominated measures of LI (width of the disk taken in the point of insert of the pelvic) (cm) and LS (width of the disk taken in the orbital area) (cm). The study was made with the species: Paratrygon aiereba, Potamotrygon schroederi, P. motoro, P. orbignyi, P. cf histrix. The obtained results demonstrate that the classes of larger similarity are outstanding starting from the choice certain proposed variables. Some clusters are very well outstanding as the one of the species P. cf histrix and of larger similarity between P. orbignyi and P. schroederi. The new measures used like LI and LS were shown efficient in the identification of the potamotrygonídeos. The result obtained through Clustering still detaches a larger cluster number, in other words, better separation when the data are referring exclusively to females among the mentioned species. It was inserted in all the variations tested in the search of the best correlation for the standard vector.

References

[1] Martin, R. A. Conservation of freshwater and euryhaline elasmobranchs: a review . J. Mar. Biol. Ass. U.K. 2005, 85, 1049 -1073.

[2] Araújo, M. L. G, Charvet-Almeida, P, Almeida M. P, Pereira, H. 2004. Freshwater Stingrays (Potamotrygonidae): status, conservation and management challenges. Information document AC 20 info 08:1-6. .http://www.cites.org/common/cttee/animals/20/E20-inf-08.pdf

[3] Mukaidano, M. Fuzzy Logic for beginners. Singapore. Ed. Word Scientific,2001.

[3] L.A. Zadeh, Similary Relations and Fuzzy Orderings. Inform. Sci.,vol.3,1971. pp.177-200.

[4] Tamura, S.,S.Higuchi and K. Tanaka, Pattern classification based on fuzzy relations. IEEE Trans. On Systems, Man, and Cybernetics,1(1),1971. pp.61-66.

A Bayesian Approach for the Stochastic Fowler's population growth model

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Keywords: Fowler's Model, Bayesian Inference, MCMC Simulation, Population model, Stochastic process, Growth models.

In this work, we are proposing one stochastic version of the Fowler's model [1] by adding to the rate of the growth model a stochastic process that represents the random environmental effects. The proposed model is given by:

$$N_{t+1} = N_t \left[1 + \frac{\rho}{q}\left(1 - \frac{N_t^q}{K^q}\right) + \epsilon_t\right] \qquad (1)$$

where, N_t and N_{t+1} are the numbers of individuals of the population in the instants t and $t+1$ respectively, ρ is the rate the population intrinsic growth, K is the carrying capacity and q is a parameter of form of the growth curve, and $\epsilon_t, t = 1, 2, \ldots$ is a white noise with $E(\epsilon_t) = 0$, $E(\epsilon_t^2) = \sigma^2$ and $E(\epsilon_t \epsilon_{t+k}) = 0$ for $k = 1, 2, 3 \ldots$

The q parameter estimate is very important because it is through it that we figured out the survival strategy adopted by a certain population, which is fundamental when concerning several ecological and economic aspects. This parameter makes the model flexible, facilitating the use of the same model in populations that have a distinct growth rate, that is, in small-sized populations such as insects, this strategy will result in a fast growth rate, and in big-sized populations such as mammals, it will result in a slow growth rate.

When concerning the problem of the population growth, the size of the sample is usually very small, therefore the methods of classic inference (the likelihood methods) become very imprecise. On the other hand, the Bayesian approach, besides outlining this difficulty, it allows us to incorporate prior informative densities to the model parameters, that way, it incorporates the specialists' specific information on a given population. By using that approach, the model application becomes more realistic as well as its estimates improve.

To compare the results obtained with the Bayesian inference we used the classic inference method, profile likelihood, which, in most of the cases, it has been generating "poor estimates", with confidence intervals having large amplitude. The Bayesian estimates, considering a quadratic loss function, were calculated by using the Markov Chain Monte Carlo (MCMC) simulation method through a mixed algorithm of Gibbs sampling and Metropolis-Hasting. We considered a 50% "burn-in-sample" of the sample size, then, we simulated $50,000$ mixed Metropolis-Hastings and Gibbs samples by taking every $10th$ sample, to get approximate uncorrelated samples. The mixed algorithms' convergence was monitored using graphical methods as well as standard existing indexes (see, for example, Geweke [2]).

The proposed method was applied to a simulated data set to evaluate the vices in the estimate of the parameters in function of the sample size (n) and three real data sets (e.g. population of the gray moth (*Acronicta megacehala*) [3], North American black bear (*Ursus Americanus*) [3] and Capivara (*Hydrochaeris hydrocharis*) in order to highlight the advantages of the Bayesian approach.

The results for the *Ursus Americanus* are shows in Table 1.

Table 1: Bayesian estimate of the model fitted to *Ursus Americanus* population.

	Estimated	IC
ρ	0.2985	0.10; 0.52
K	388	282; 758
q	1.505	1.324; 1.708
σ^2	0.3466	0.277; 0.4336

References

[1] C. W. Fowler, Density dependence as related to life history strategy. Ecology, 62(3), pp. 602-610, 1981.

[2] GEWEKE, J. Evaluating the accuracy of sampling-based approaches to calculating posterior moments. In **Bayesian Statistics 4**, (ed. J. M. Bernardo, J. O. Berger, A. P. Dawid, and A. F. M. Smith). Clarendon Press, Oxford, UK. 1992.

[3] Site onde os dados foram obtidos: **http://cpbnts1.bio.ic.ac.uk/gpdd/**.

An EDP model with fuzzy dispersion parameter applied on foot-and-mouth disease in bovines

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Keywords: Foot-and-mouth disease, diffusion equation, fuzzy systems.

In this work we propose a compartmental SIR model to study the temporal and spatial evolution of the foot-and-mouth disease in bovines, assuming the estate variables are: density of susceptibles animals (S), density of infected animals (I) and density of recovered animals (R).

This model, based on [3], is represented by the following systems of equations:

$$\begin{cases} \frac{\partial S}{\partial t} - \operatorname{div}(D_S(C,P).\nabla S) = -S(\beta I + \theta) \\ \\ \frac{\partial I}{\partial t} - \operatorname{div}(D_I(C,P,FD).\nabla I) = S(\beta I + \theta) - \mu I \\ \\ \frac{\partial R}{\partial t} - \operatorname{div}(\alpha D_S(C,P).\nabla R) = \mu I, \end{cases}$$

where β is the rate of transmition of the foot-and-mouth disease, which is proportional to the rate of encounter of susceptible and infected animals, θ represents the ratio of susceptible animals that turn to be infected by the foot-and-mouth disease by indirect contact and μ represents the rate of recovery.

The initial conditions are: $S(x,y;0) = S_0$, $I(x,y;0) = I_0(x,y)$ e $R(x,y;0) = 0$. The bounded conditions are: Dirichlet at Γ_0 and Von Neumann at Γ_1.

The dispersion of susceptibles is given by $D_S(C,P)$, whereas the dispersion of infected is given by $D_I(C,P,FD)$. These parameters were determined from a rule-based fuzzy system (RBFS) depending on linguistics variables: characteristics of the soil (C), size of the population (P) and stage of the disease (FD).

The simulations were obtained from the finite elements method allied to the Mandani's inference method [1, 2].

References

[1] C. G. Almeida, R. S. M. Jafelice and J. F. C. A. Meyer, Modelagem fuzzy em dinâmica populacional. XXIX CNMAC. Campinas, SP, 2006.

[2] L. C. Barros and R. C. Bassanezi, Tópicos de loógica fuzzy e biomatemática. Coleo IMECC-Textos didticos, IMECC/UNICAMP, Campinas, SP, 2006.

[3] F. Ferreira, Dinâmica espacial da febre aftosa em bovinos: um modelo matemático. Tese Doutorado, Faculdade de Saúde Pública, USP, São Paulo, SP, 2000.

Computer model for stem-cell action in the maintenance and regeneration of living tissues

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Keywords: stem-cell, computer models, population dynamics

Notwithstanding growing scientific interest attracted by the issue, still not much is known about the detailed mechanisms for stem-cell action. In this work a computer model is presented and used to study the action of stem-cells in the maintenance and regeneration of living tissues.

In each cell division a piece of the telomers, chromosome ends responsible for the genetic integrity of the cell, is lost, what can lead to a fast accumulation of mutations. The organism protects itself by imposing the proliferative Hayflick limit [1] to its cells, 50 to 70 divisions, after which they enter senescence. On the other hand non-differentiated stem-cells are able to restore their telomers ant thus have unlimited proliferative capacity. They generate all human tissues and also work for the maintenance of organs and tissues [2, 3].

In this work a computer model is presented and used to compare the population dynamics of cells in a living tissue with and without the action of stem-cells from the points of view of size regulation and genetic quality stabilization. In the stem-cell scenario new cells can be generated without any telomer shortening, together with those resulting from the mitosis of specialized tissue-specific cells that already carry some telomer loss in their lineages.

Various combinations of cell mortality and mitosis rates are investigated. We show that the model with some stem-cell activity is considerably more capable of maintaining population size for a long time. We also verified that stem-cell action can keep average genetic quality, while tissues that lack this ingredient present continuous genetic degeneration over time. This feature suggests that stem-cell activity may also be relevant for tumor prevention [4, 5].

References

[1] M. I. N. Cano, A vida nas pontas dos cromossomos, Ciência Hoje, 39, n. 223, 16-23, 2006.

[2] C. Soares, Manutenção interna, Scientific American Brasil, ed. Especial, 64-68, 2005.

[3] C. Cookson, Mãe de todas as células, Scientific American Brasil, ed. Especial, 64-68, 2005.

[4] R. A. De Pinho, The age of cancer, Nature, 408, 248-254, 2000.

[5] R. A. Weinberg, Uma célula renegada: como o câncer começa, Rocco, Rio de Janeiro, 2000.

Methodology of Rehabilitation of the muscle cuadriceps for individuals with knee artrosis using logical Fuzzy

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Keywords: Artrosis, Fuzzy, Takagi-Sugeno,
Crondomalacia rotuliana.

The *artrosis* pathology is a recurrent illness in people of advanced age, however in young individuals for congenital or sport circumstances, they generate previous squares to the same *artrosis*, as *crondomalacia rotuliana* [1], which are already restrictive for the ideally healthy individual's activities.

This pathology is generally treated, depending the advance degree by means of surgical interventions or of treatment [1]. This last one is the first option for not being invasion and doesn't have the inherent risks of all operation.

This treatment is determined by a series of exercises that you/they pursue the growth of muscular mass. This is a parameter to consider since in the clinical evaluation of the state of the knee the stability it is determined mainly by the performance of this muscle [2].

In these exercises two big types settle down, isotonic and isometric [3]. These differ for that the one finishes it presents a contraction of the muscle in a fixed position articulation, in this case it involves the contraction in a fixed angle of flexion of the knee. The one used in this study is the isotonic, with a small degree of flexion, around 15º at 30º.

A model is developed based on logical Fuzzy that represents like the flexion degrees act when carrying out the exercise and the fatigue of the muscle under stable conditions (extended knee and elevation of this). A model *Takagi-Sugeno* is carried out.This way where for different flexion degrees (X1) and the degree of fatigue of the muscle (X2) are considered the growth of the muscle (Y). For the degree of fatigue is considered a being based on the number of possible repetitions with extended leg, of that which you take the values of *Acceptable* and *Maximum.*

They are not considered the variables of characteristic of each individual like their metabolism, because these cases the pathology is advanced and the maximum yield of the exercise is not possible. For they are generated it rules in the way :

$$R^i: \quad IF \quad x_{is} B^i(x)$$
$$THEN: \quad y^i = f^i(x)$$

Where x = [x1, x2] they are the entrances of the pattern, (angle of inclination and level of fatigue) of the pattern Fuzzy regression and Bi(x) it is the combined antecedent Fuzzy of the i-èsima it rules. A kindred lineal function of the entrance variables is generated:

$$F^i(x) = a_o + a^T x$$

With this an in agreement procedure settles down to each individual's characteristics that being able to be a classic methodology of exercises, however for individuals with artrosis the continuity of the routine of exercises the knowledge of the pain degree to articulate and muscular it is very valuable. It is pursued this way a support model to the decision from the therapist to the appropriate dosage of exercises it stops the patient.

References

[1] Quintela , Ana ; De Oliveira, Lorena, Condromalácia Patelar por desequilibrio do quadriceps femoral, Cadernos Brasileiros de Medicina , Janeiro do 2003, Volumen XVI.

[2] Cailliet, R. Tecidos Moles, Dor e Instabilidade. Manole, São Paulo, SP, 1998.

A Fast nEMD Algorithm and its applications on Population Dynamics

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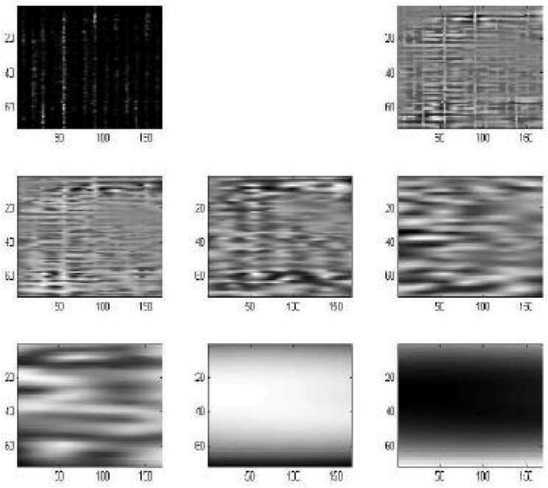
Keywords: EMD, nEMD, population dynamics, signal decomposition

We had developed an extension[2,3,4] of the Empirical Mode Decomposition1 (EMD) to signals whose domain is R^n using Radial Basis Functions (RBF)[5] as interpolants. A 2-d domain signal decomposition using this method will look like on the sample below:



Since it takes the huge $O(n^3)$ amount of disk space and RAM memory to do RBF interpolation, we have moved to Inverse Distance Weighted Interpolation, which is a much more fast and less memory consumming method – and also embarrassingly parallel.

With the speed and memory enhancements, it is now possible to perform nEMD on a much more wider set of population dynamics problems.

References

[1] N.E. Huang, Z. Shen, S.R. Long, S.R., M.C. Wu and Shih, H. H., et al. "The Empirical Mode Decomposition and Hilbert Spectrum for Nonlinear and Nonstationary Time Series Analysis". Proc. Royal Society of London Series 1998, A 454, 903-995.

[2] Raphael Vilamiu & Wilson C. Ferreira Jr. "nEMD: An extension for Empirical Mode Decomposition for Signals on R^n and Hypersurfaces", XXIX CNMAC - Congresso Nacional de Matematica Aplicada e Computacional, 2006 Sept 21th, Campinas, SP, Brazil

[3] Raphael Vilamiu & Wilson C. Ferreira Jr. "nEMD on Spatio-Temporal Data: A Case Study of Geographical Dengue Epidemics", XXIX CNMAC - Congresso Nacional de Matematica Aplicada e Computacional, 2006 Sept 21th, Campinas, SP, Brazil

[4] Raphael Vilamiu & Wilson C. Ferreira Jr. "Decomposition of Multi-Scaled Chemical Data using nEMD", CAC2006 - 10th Internation Conference on Chemometrics in Analytical Chemistry, 2006 Sept 14th, Aguas de Lindoia-SP, Brazil

[5] M. D. Buhmann. "Radial Basis Functions". Acta Numerica 2000, 1-38

A mathhematical model for *Aedes aegypti* life cycle and epidemies control

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Keywords: *Aedes aegypti*, chemical control, mechanical control

Epidemies of dengue, a disease transmitted by a vector, the *Aedes aegypti* mosquito, have become a major public health problem in tropical countries such as Brazil since controlling them is a very hard task . The usual chemical control has not been an efficient method which makes the quest for new ones a rewarding enterprise. In this work we analise the efficiency of different methods of control by means of a mathematical model of the *Aedes aegypti* life cycle. The main ingredient of this model is a set of four coupled differential equations that describe the temporal evolution of populations of the four stages of the mosquito life cycle: eggs, larvas, pulps and adults. In this model, by using metamorphosis rates that depend on temperature and solving numerically the equations with real data we can reproduce the well known fact that epidemies are associated to hot seasons. Besides this, based on this dependence on temperature we are able to devise efficient strategies for mechanical and chemical control.

References

[1] DONALÍSIO, M.R. e GLASSER, C.M., Epidemiological monitoring and Control of Dengue Vector, Rev. Bras. Epidemiol., vol. 5, n° 3, 2002.

[2] FERREIRA, G.S., It analyzes secular space of the dengue cases in Rio de Janeiro city between 1986 and 2002, Dissertação de mestrado, Rio de Janeiro: UFRJ/IM, 2004.

[3] TAUIL, P.L., Critical aspects of dengue control in Brazil, Public Health Book, Rio de Janeiro, pág 867-871, may-june de 2002

[4] YANG, H.M., FERREIRA, C.P. e TERNES, Populational dynamic of Dengue's transmitter vector, TEMA Tend. Mat. Apl. Comput., 4, n° 2, pág 287-296, 2003.

Dynamics Population of Microorganisms applied to the Food Conservation

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Keywords: mathematical model, food conservation, dynamics population.

The food contamination by *Listeria monocytogenes* represents a serious problem for the public health, which is due, mainly, to the large distribution of this phatogen in the nature and, especially, for its capacity to survive at low temperature (of refrigeration). Listeriosis, a serious infection caused by *L. monocytogenes*, affects primarily pregnant women, newborns and adults with weakened immune systems. The lactic acid bacteria (LAB) can be used for the food conservation by means of competitive exclusion with other microorganisms and, also, through the production of inhibitory substance, the lactic acid and the bacteriocins. Bacteriocins produced by the lactic acid bacteria are originated from the extracellular production or byproducts of bacterial ribosomal synthesis. Based on the dynamics population theory, a mathematical model was developed in order to describe the interactions between LAB producing bacteriocin and the *Listeria* in the food. The steady states and dynamical trajectories analysis of the model, which was structured as a system of nonlinear ordinary differential equations, provided us to assess the possible use of LAB to reduce and/or to inhibit the development of *Listeria* in the food. The loss of stability of both lactic acid and bacteriocin during the time was also considered.

The model exhibits three equilibria: the first equilibrium point corresponds to the state where the two species (LAB and *Listeria*) are absent, the second is regarded to the state where only lactic acid bacteria are present, and the third one is the coexistence equilibrium. When action of bacteriocins and acid lactic occur on the Listeria, it is possible to inhibit the growth or even to diminish the contamination up to the extinguishing of the Listeria. The existence of the non-trivial break-point to eliminate Listeria depends essentially on the values of the parameters of the model. Numerical simulations of the model had been carried out using package MATLAB®, using the Runge-Kutta of fourth order. The analysis of the model furnished information on the behavior of the solutions, and also to assess the effects of the lactic acid and the bacteriocin, produced by lactic acid bacteria, on the control of the Listeria in the food.

References

[1] W. Boyce, R. DiPrima. Equações diferenciais elementares e problemas de valores de contorno. 7ªed. Rio de Janeiro: LTC, 2002. 416p.

[2] R. C. Bassanezi, W. C. Ferreira Jr. Equações Diferenciais com aplicações. São Paulo: Harbra, 1988. 572p.

[3] P. M. Muriana. Bacteriocins for control of *Listeria* spp in food. Journal of Food Protection, Ames, Supplement, p.54-63, 1996.

Modeling species competition with intermingled basins

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Keywords: Intermingled basins, unstable dimension variability, periodic orbits, species competition.

Similar species with limited resources supply cannot, in general, coexist. The main goal in studying population dynamics is to construct a model that represents the interaction between these species and to predict which one will be extinct, which can be a very intensive task. As example there are experiments of competition between two species of flour beetle: *Tribolium castaneum* and *Tribolium confusum*. These beetles interact each other by indiscriminate cannibalism and one of them always dies out. However, under specific laboratory conditions, seemingly identical experiments exhibit a different survivor, characterizing an indeterminate competition [1].

Although it seems to be a random process, it is possible to construct a capable deterministic model to reproduce, at least qualitatively, this phenomenon [2]. Such system can be obtained from a paradigmatic equation of species competition, $\dot{x}_i = x_i\varphi_i(x_1, x_2)$, $(i = 1, 2)$, under the assumption that the species are biologically identical, their generation do not overlap, and each species has, in absence of another, a chaotic evolution. Here, x_i stands for the density of i-th population, $\varphi_i(x_1, x_2)$ is called interaction function and satisfies $\partial\varphi_i/\partial x_j < 0$, $(j \neq i)$.

Mathematically, all possible experimental arrangements forms the full phase space of the system where exist two chaotic attractors, and each one represents the survival of one species. Since the extinction is the rule, the basins of attraction fulfill all the phase space. If these basins are intermingled, or everywhere dense, the system can reproduce the indeterminate competition. It means that seemingly identical experiments, represented by nearby points in phase space, can exhibit different survivor, corresponding to different attractors.

One of the key requirements for the intermingled is that the neighborhood of one attractor contains a non-null fraction of the basin of another attractor. It is guaranteed by the existence of transversally unstable periodic orbits (UPOs) embedded in both attractors. But the attractor contains, by definition, transversally stable periodic orbits. Therefore there are UPOs with different numbers of unstable directions characterizing a strong form of nonhyperbolicity, called unstable dimension variability (UDV) [3]. The UDV causes severe difficulties in modeling, since the shadowing times of numerical solutions for these systems can be very short.

In this work, we made a dynamical approach of a system proposed in Ref.[2], focusing our attention on the UPOs embedded in the attractors. We quantified the intensity of UDV by means of finite-time transversal Lyapunov exponents, which measure the contraction or expansion rate of the space phase of the system, and by the fraction of transversally unstable periodic orbits embedded in the attractors. We found that statistical properties of the system and of the basins of attraction, as the average time to extinction and the uncertain fraction, respectively, can be estimated by means of periodic orbit analysis.

References

[1] R. F. Costantino, R. A. Desharnais, Population Dynamics and the Tribolium Model: Genetics and Demography, Springer-Verlag, New York, 1991.

[2] F. Hofbauer, et. al., Intermingled basins in a two species system. J. Math. Biol. 49, 293-309, 2004.

[3] E. J. Kostelich, et. al., Unstable dimension variability: A source of nonhyperbolicity in chaotic systems. Physica D 109, 81-90, 1997.

Modeling the impact of sexual health education and behavior of young women on vertical HIV transmission: the demographic question

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Keywords: Vertical transmission; HIV; education programs; core groups. We develop a model for the transmission of HIV in a population of varying size with basically vertical transmission; that is mother to child transmission. We start developing a general deterministic mathematical model to represent the natural course of HIV infection in the sexually active population by coupling transmission of HIV through the core groups. This model is then simplified by way of limiting it to only to vertical transmission of HIV. A study of this simplified mathematical system is provided to analyze the role of the HIV/AIDS education programs and drug treatment as control mechanisms to prevent vertical transmission of HIV. From this study we derive conditions under which the burden of HIV/AIDS can be reduced in the population in the absence of horizontal transmission, assuming that new infections only occur from mother to child. Pregnant women migrate to the HIV^+ pregnant women compartment only after tested seropositive. We assume that this happens at a constant rate, and that the anti-HIV treatment is only administered to seropositive pregnant women and their newborns.

The study of just pregnant women and their newborns as the core-group, simplifies the general nonlinear model to a linear one that describes the dynamics of vertical transmission of HIV. The stability of the endemic equilibrium is studied and conditions that predict the control of disease are found. The model predicts that strategies of prevention like drug treatment and sexual education can be a powerful tool to reduce the risk of vertical transmission of HIV

References

[1] S. M. Raimundo; H.M. Yang; A. B. Engel Modelling the effects of temporary immune protection and vaccination against infectious diseases. Appl. Math. Comput. doi:10.1016/j.amc.2006.12.051, 2007

[2] M. G. Gomes; A. O. Franco; M. C. Gomes; G.F. Medley. The reinfection threshold promotes variability in tuberculosis epidemiology and vaccine efficacy. Proc. Biol. Sci. Mar 22;271(1539):617-23, 2004.

Modelling the regional dynamics of annual plants

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Keywords: seed bank, dispersal kernel, integrodifference equations, annual plants, agent-based model

We develop an integrodifference equation model to describe the large-scale spatial dynamics of an annual plant with a long lived seed bank. Our model is parameterised with data taken from our experiments on natural populations of *Brassica nigra* on the cliffs of Southern England. Our experimentation has suggested that anthropogenic dispersal is a potential dispersal vector in addition to the primary dispersal vector (wind) for this species. Informed by these experimental results we investigate mathematically the effect of a variety of dispersal kernels to describe the distribution of dispersal distances of seeds about their parent plant. We calibrate our model by comparison with data from the natural plant populations. Moreover, we compare our model with an agent-based simulation model, as part of the on-going challenge of investigating the strengths and weaknesses of different modelling approaches.

Intraguild predation and foraging dynamics in blowflies

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Keywords: Calliphoridae, Chrysomya albiceps, blowflies, intraguild predation, optimal foraging, paradox of enrichment .

Flies of Calliphoridae family are part of an interesting biological system with development in discrete time, because of their ability to consume different food items. This kind of behavior can make some species change their trophic levels from competitors to intraguild predators. The intraguild predation in blowflies have been studied systematically to evaluate the extension of the predatory behavior in the context of exotic species introduction and optimal foraging. Among the blowfly species, Chrysomya albiceps and C. rufifacies are known for making part of the exotic species introduced into the Amricas 40 years ago. They exhibit during the larval phase interactions such as competition, facultative predation and cannibalism. The optimal foraging theory is based on the maximization of net energy obtained by the predator, with the nutrient acquisition for some period of time, or in the minimization of the foraging time to obtain a minimum diet necessary. The Paradox of Enrichment [1] comes from nutrient enrichment of prey populations in stable predator-prey systems, resulting in the predator biomass increase. The enrichment produces destabilizing effect upon the system, leading populations that previously have exhibited one point stable equilibrium to show limit-cycles. Few studies have investigated effects of prey choice on the dynamics of prey and predators, with analysis focusing on multi-trophic interaction in order to obtain alternative results with respect to enrichment classic theory. This study proposes a mathematical model to explain the effect of the optimal foraging strategy on the intraguild predation dynamics, from the combination among competition and foraging models. To perform this work three discrete time mathematical models were combined: optimal foraging [2], competition and intraguild predation [3]. The combination among the three mathematical models resulted in a coupling of equations producing a model that incorporate competitive process, intraguild predation and food optimization, expressed by the optimal foraging dynamics. The model was produced in time discrete to best describe the system in which make part the blowflies. The results founded in the simulations suggest that the presence of alternative prey produces limit cycle for the non-preferred prey and exclusion for the other prey. These results are interesting, because in the simulations focused on the prey palatability [2], the consumption of non-palatable prey trends to stabilize the system contesting the classic theory of the enrichment. Our results suggest that the intraguild predation may be an interactive process capable to give support to the paradox of enrichment. Most of trophic systems exhibits higher complexity level than the founded in the investigated interactions. However, the conditions set to the simulations here performed have permitted to know what behavior dynamic patterns can arise under specific conditions, in which, an intraguild predator adopts different strategies in response to alternative prey. In addition, the mathematical structure proposed to analyse the interaction effects in the context of intraguild predation, made possible to evaluate the association between individual and population behavior, considering laboratory data.

References

[1] M. L. Rosenzweig, Paradox of enrichment: destabilization of exploitation ecosystems in ecological time. Science 171: 385-387, 1971.

[2] M. Genkai-Kato & N. Yamamura, Unpalatable prey resolves the paradox of enrichment. Proceedings of the Royal London 266: 1215-1219, 1999.

[3] N. J. Gotelli, A primer of ecology, 3th edition. Sinauer Associates, Inc., Sunderland, MA. 265p, 2001.

Investigation of population dynamics of seabob shrimp *Xiphopenaeus kroyeri* in Ubatuba Bay, Brazil by Artificial Neural Networks

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Keywords: *Xiphopenaeus kroyeri*, Artificial Neural Networks, Ubatuba

Abstract: Recent studies have been developed in order to demonstrate that indiscriminate fishing may cause in near future extinction of many marine species, like the seabob shrimp *Xiphopenaeus kroyeri* [1]. Based on data collected during January 1998 and December 1999 in Ubatuba bay region [1], we provide here a way of predicting fishing population in following months, using artificial neural networks. Each collection was carried out by a fishing boat, trawling during 30 min every first 3 days of full moon of each month, in 6 transects of length 1km previously chosen by GPS system. Features of all individuals collected were measured, such as weight, height, sex and stage of sex gonads, and these data were used as input for a class of neural networks [2] called Multilayer Perceptron (MLP), created using component NNTool, which is part of Matlab software [3]. Different parameters were tested, such as learning rate, momentum, number of training cycles and learning function in order to find out which are the best parameters for simulating this species' dynamics.

Tests showed that using function Logsig transfer function, learning rate 0.9, momentum 0.5 and 5000 training cycles the best results are reached. Mean Square Error for these parameters in training set achieved 0.0131 and 0.0119 in test set, using as input attributes the population from 3 previous months, water temperature during fishing time, salinity and transect where the shrimp were taken. Output attribute was the population for the following month, normalized between 0 and 1 (Figure 1).

In conclusion, we find that Artificial Neural Networks is a very useful tool to simulate *X. kroyeri* population dynamics. Results were not as accurate as desirable, but data from a higher period of time may make it sufficient for a good approximation of the real curve of this species.

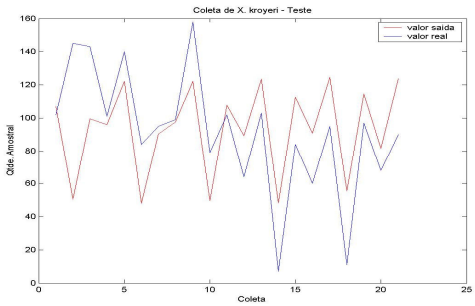

Figure 1. Real data x predicted data using parameters previously described

References

[1] A. Fransozo; R. C. Costa; M. A. A. Pinheiro; S. Santos & F. L. M. Mantelatto. (2000). "Juvenile recruitment of the seabob shrimp Xiphopenaeus kroyeri (Heller, 1862) (Crustacea: Penaeoidea) in the Fortaleza Bay, Ubatuba, SP, Brazil", Nauplius, v. 8, n. 2, p. 179-184.

[2] R. H. Castro, R. C. Costa, A. Fransozo & F. L. M. Mantelatto (2005). "Population structure of the seabob shrimp Xiphopenaeus kroyeri (Heller, 1862) (Crustacea: Penaeoidea) in the littoral of São Paulo, Brazil", Scientia Marina, v. 69, n. 1, p. 105-112.

[3] S. Haykin. "Redes neurais: princípios e práticas". 2 ed. Bookman, Porto Alegre, 2001.

[4] Matlab, User's Guide, The MathWorks, Inc., Natick, MA 01760, 1992.

Modeling the Interaction between Immune System and bacterium Streptococcus and Staphylococcus

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Abstract

When bacteria overcome physical barrier (skin or mucus of digestive and intestinal tracts), they induce local inflammatory reaction due to the action of innate immune system. We developed a mathematical model to describe the bacterial infection taking into account the invectiveness. The results are applied to assess the invasion of two species of bacteria (staphylococcus and streptococcus) through the human skin.

The bacterium streptococcus and staphylococcus are bacterial pathogens that cause the skin and soft tissue infections. They present distinct characteristics. The staphylococcus invades the tissues releasing lethal toxins. While the streptococcus doesn't cause intense local destruction. However, we observe that streptococcus has a greater disposition in spreading into the host body and cause harms, although the staphylococcus is much more destructive to the tissues.

The explanation for this phenomenon resides in the bacterium's capacity in provoke immune reactions. According to the developed mathematical model, the bacteria which managed invade through the physical barrier of an healthy and immune-competent individual, generally, are eliminated. When the greater is the toxin destruction capability released by bacteria during multiplication process, more energetic is the immune response. However, when the bacterium acts "silently", i.e., the toxins are less cytotoxic, the bacterium can evade easier to the blood vessels and spread in the host organism causing infections in the multiple organs. This is due to the few signalizing to attract macrophages to the infections site. The streptococcus cause relatively less destruction to the tissue cells, which induce few macrophage migration, as consequence, the low rate per-capita of mortality of bacteria caused by macrophages.

References

[1] L. Edelstein-Keshet, *Mathematical Models in Biology*, Random House, New York, 1987.

[2] Z. Feng, C. Castilho-Chavez and C.A.F. Capurso, *A Model for Tuberculosis with Exogenous Reinfection*, Theorical Population Biology, 57(2000), 235-247.

[3] J.D. Murray, *Mathematical Biology I: An Introduction*, Ed. Springer, 1993.

[4] Pei-Jane Tsai, Ying-Huei Chen, Chieh-Hsing Hsueh, Hsiao-Chun Hsieh, Ya-Hui Liu, Jiunn-Jong Wu, Chih-Cheng Tsou, *Streptococcus pyogenes induces epithelial inflammatory responses through NF-κ/MAPK signaling pathways*, Microbes and Infection 8 (2006) 1440-1449.

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