Abstracts Plenary Talks

DSAβNS 2012 Lisbon

Ben Adams
Department of Mathematical Sciences, University of Bath, UK
b.adams@bath.ac.uk

Simple models for antigenic evolution, with an application to influenza A

In response to an infection the body produces antibodies specific to the antigenic structure of the pathogen. After the infection has been cleared these antibodies prevent repeated infection with an identical pathogen, but may offer only partial protection against pathogen strains with an altered antigenic structure. Epidemic models with many co-circulating pathogen strains indicate that partial cross-immunity can lead to three solutions: all strains stably coexisting; a subset of strains stably coexisting; all strains coexisting in complex cycles. These results are important since they underpin the evolutionary dynamics of a number of pathogens, most notably influenza.

In this talk I will consider an epidemic model in which multiple strains have immune cross-reactions determined by their relative locations in an antigenic space. I will examine how the structure of the antigenic space, and the way in which antigenic distance is related to the intensity of cross-immunity, determine exclusion or coexistence. I will then consider some implications of these results including the optimal antigenic location for an invading strain, the optimal antigenic distribution for two strains to resist invasion, the likely antigenic evolution and the resultant strain structure. Finally I will discuss the application of these ideas in a more complex model to examine how the evolutionary dynamics of influenza A virus are affected by year-round and seasonal transmission regimes.

Konstantin Blyuss
University of Sussex, UK
K.Blyuss@sussex.ac.uk

Symmetry and synchronization in models of antigenic variation

In this talk I will discuss the dynamics of interactions between antigenic variants and the human immune system during immune escape. Using the methods of equivariant bifurcation theory, I will show on the example of a model of antigenic variation in malaria the effects of symmetry on possible dynamical regimes and (de)synchronization of antigenic variants. The results of the analysis are quite generic, and I will also discuss their wider applications to the studies of multi-strain diseases.

Daniel Bontje
Central Veterinary Institute of Wageningen, The Netherlands
daniel.bontje@wur.nl

Optimal culling and vaccination strategies given limited culling and vaccination capacities during FMD epidemics

During the Food-and-mouth (FMD) epidemic of 2001 around 270 thousand animals were (pre-emptively) culled in the Netherlands. Based on the 2001 outbreak data, the Central Veterinary Institute (CVI) developed a kernel to model the spatial and temporal spread of FMD. In Backer et al (2009) the effect of different ring-culling and ring-vaccination strategies on the course of FMD epidemics were simulated to provide input for policy making: to vaccinate or not to vaccinate? For these simulations culling and vaccination capacities were assumed to be unlimited. Now we present the results of the effect of including realistic culling and vaccination capacities on epidemic duration and magnitude while considering different strategies in dealing with the order of ring culling and vaccination. This stochastic FMD model consist of the following components: a SEIR within-herd model, a kernel based between-herd transmission model, farm data with herd size and species, and parameters which can be affect by the control measures. Although the focus of this presentation will be on modelling FMD, the same generic approach to simulate epidemics was also applied on Classic Swine Fever (CSF) and Avian Influenza (AI) and some examples will be drawn from these simulation results.

Fabio Chalub
Universidade Nova de Lisboa, Portugal
chalub@fct.unl.pt

Discrete and continuous models in evolutionary dynamics

We consider finite populations and a discrete (in time) evolutionary process, e.g., the Moran and the Wright-Fisher process. We show that the state of the population can be approximated in all time scales by the solution of a given partial differential equation (PDE) of parabolic type with degenerated diffusion (i.e., the coefficient of diffusion is zero on the boundaries). We cannot impose boundary conditions to this equation, however the uniqueness of solution is guaranteed using conservation laws that can be directly obtained from the discrete process.

The PDE has naturally two different time scales, the first one represents the natural selection (and is equivalent to the replicator dynamics) and the second one the genetic drift. The adjoint equation is known as the Kimura equation.

We will also show that the replicator equation is a good approximation for a finite population of size N when N is large or when the selection is strong.

Bob W. Kooi
Department of Theoretical Biology,
VU university Amsterdam, the Netherlands
kooi@bio.vu.nl

Analysis of Arnold tongues in non-autonomous epidemiological models

The long-term dynamics of periodically forced epidemiological dynamical systems is studied. In autonomous systems the simplest long-term dynamic behaviour is the equilibrium. The equilibrium o fa dynamical system with dimension higher than one, can become unstable at a Hopf bifurcation after which a periodic solution or limit cycle emerges when a parameter is varied. For systems with dimension higher than two a limit cycle can become unstable at a Torus bifurcation. After such a bifurcation the dynamics can be restricted to a torus where periodic or quasi-periodic solutions exist. But also chaotic dynamics is possible. Equilibriums do not exist for nonautonomous systems but periodic solutions do. The simplest situation is when the period of this solution equals the period of the forcing. The stability of this periodic solution (limit cycle) is defined in the same way as that of the corresponding limit cycle of a autonomous system. Furthermore, the Hopf bifurcation of the autonomous systems becomes a Torus bifurcation in the associated periodically forced system where the amplitude of the forcing is zero. Also this limit cycle can become unstable via a torus bifurcation and so on.

An interesting phenomenon that can occur in the forced system is called frequency locking which gives regions in the parameter space where a stable periodic solution exist. These regions emanate from points on the torus bifurcation or on the zero-forcing axis where the dynamics is periodic and are called Arnol'd tongues. These Arnol'd tongues are bounded by tangent bifurcations for the limit cycles associated with the periodic solution. At these bifurcation points the stable limit cycle collides with a coexisting unstable limit cycle. With computer packages for detecting and continuing bifurcations one can calculate these bifurcation curves in a deliberately chosen parameter space. We will show results for the seasonal forced multi-strain dengue model that captures differences between primary and secondary infections which is described and analysed in [1].

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[1] Aguiar, M., Ballesteros, S., Kooi, B.W., & Stollenwerk, N. The role of seasonality and import in a minimalistic multi-strain dengue model capturing differences between primary and secondary infections: complex dynamics and its implications for data Analysis. Journal of Theoretical Biology, 289:181--196, 2011.

Jorge Pacheco
Lisbon University and Minho University, Portugal
jorgem.pacheco@gmail.com

If cells play dice, can we gamble our way of out of cancer?

Stem cells are the target of mutations that can lead to life threatening diseases. However, stem cell populations tend to be small and therefore clonal expansion of mutant cells is highly sensitive to stochastic fluctuations. The evolutionary dynamics of mutations in these cells is discussed, taking into consideration the impact of such mutations on the reproductive fitness of cells. We show how stochastic effects can explain clinical observations including extinction of acquired clonal stem cell disorders.

Sergei Petrovskii
Department of Mathematics, University of Leicester, UK
sp237@leicester.ac.uk

Computational Ecology as an Emerging Science

It has long been recognized that numerical modelling and computer simulations can be used as a powerful research tool to understand, and sometimes to predict, the tendencies and peculiarities in the dynamics of populations and ecosystems. It has been, however, much less appreciated that the context of modelling and simulations in ecology is essentially different from those that normally exist in other natural sciences. In our talk, we review the computational challenges arising in modern ecology. Concept modelling and predictive modelling are two basic streams in ecological research that employ essentially different computational techniques, and we explain where that difference comes from. We then mainly focus our attention on the concept modelling. Somewhat paradoxically, the complexity of ecological problems does not lead to the use of complex computational methods. This paradox, however, can be easily resolved if we recall that application of sophisticated computational methods usually require clear and unambiguous mathematical problem statement. At the same time, many ecological problems still do not have mathematically accurate and unambiguous description yet, and it is often hard to understand how the results of computations should be interpreted from the ecological viewpoint. In this scientific context, the computational ecology has to deal with a new paradigm: conventional issues of numerical modelling such as convergence and stability become less important than the qualitative analysis that can be provided with the help of computational techniques. We discuss this paradigm by considering several computational challenges posed by ecological applications. We also provide a few examples of ecological problems where computational methods have successfully been applied.

Jean-Christophe Poggiale
Université Aix - Marseille II
jean-christophe.poggiale@univmed.fr

Mathematical formulation of ecological processes : a problem of scale

Mathematical approach and ecological consequences

In this talk, we present some works dealing with the choice of mathematical expressions satisfying similar ecological assumptions and analyze the consequences of these choices. The ideas are illustrated on communities models. These examples raise the importance of the choice of a mathematical formulation in a model and introduce the concept of structural sensitivity (Cordoleani et al., 2011, Poggiale et al., 2010). In the perspective of ecological modeling, we propose an approach to build model on mechanistic arguments, by integrating processes occurring on different time scales. This allows us to get rather simple formulations at the community level for any given process. We illustrate this approach on the example of trophic interactions, an more precisely on the functional response in a phytoplankton - zooplankton model in the column water, based on data obtained in chemostat cultures. Other examples are envisaged.

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J.-C. Poggiale, M. Baklouti, B. Queguiner and S.A.L.M. Kooijman, (2010), How far details are important in ecosystem modelling: the case of multi-limiting nutrients in phytoplankton - zooplankton interactions, *Philosophical Transactions of the Royal Society - B*, Vol. 365, 3495-3507.

Hauke Reuter
Leibniz-Center for Tropical Marine Ecology, Germany
hauke.reuter@zmt-bremen.de

Level crossing interactions in ecological frameworks - The potential of agent-based models

Analysing complex ecological causality networks constitutes one of the main challenges for ecological science. Especially ecological modelling and ecological theory as disciplines have to develop concepts and methodologies to achieve this aim. The necessary abstraction include handling the number of components, pooling the numerous interaction types and coping with the high variability inherent to ecological units, processes and interactions.

Agent-based models constitute a powerful approach which allows to incorporate and analyse a heterogeneous knowledge and data derived from different ecological sub-disciplines and with different overall qualities, which represent ecological components, biotic interactions and dynamics. In this presentation we will give several examples for agent-based models which illustrate the potential to analyse complex ecological networks across hierarchical levels.
a) The model on coral reef dynamics allows to investigate the conditions for phase-shifts resulting from spatial interactions between corals and competing benthic organisms (e.g. macroalgae). Coral reefs are subject to numerous anthropogenic threats (e.g. overexploitation, mechanical destruction, global climate change) and the model allows to identify critical situations and helps in management.

b) A second application focuses on the analyses of trophic networks. Most of the results gained in classical food chain simulation models use averaged interaction intensity values which implicitly assume spatial homogeneity leading to potential inaccuracies if spatial processes are involved. With a spatially explicit agent-based modelling approach we developed a predator-prey system which exhibits different forms of spatial and temporal self-organisation (e.g. stationary clusters or travelling waves). The simulations demonstrate clearly that temporal population dynamics depend on different movement patterns of the represented organisms.

Both agent-based models show the potential of the approach to extend the range of ecological processes and phenomena modelling can handle.

Francisco Santos
Universidade Nova de Lisboa, Portugal
xsantos@gmail.com

An evolutionary dynamics approach to global warming

The welfare of our planet stands as a perfect example of what scientists commonly refer to as public goods—a global good from which everyone profits, whether or not they contribute to maintain it. Indeed, reducing the effects of global warming has been described as one of the greatest public goods problems humans have faced, and the one we cannot afford to lose. Unfortunately, individuals, regions or nations may opt to be "free riders", hoping to benefit from the efforts of others while choosing not to make any effort themselves. Cooperation problems faced by humans often share this setting, in which the immediate advantage of free riding drives the population into the "tragedy of the commons", the ultimate limit of widespread defection. Moreover, nations and their leaders seek a collective goal that is shadowed by the uncertainty of its achievement. Such types of uncertainties have repeatedly happened throughout human history from group hunting to voluntary adoption of public health measures and prospective choices.

In this talk, I will discuss an evolutionary dynamics approach to a broad class of cooperation problems in which attempting to minimize future losses turns the risk of failure into a central issue in individual decisions [1]. Resorting to the mathematical tools of game theory, we find that decisions within small groups under high risk and stringent requirements to success significantly raise the chances of coordinating actions and escaping the tragedy of the commons. We also offer insights on the scale at which public goods problems of cooperation are best solved. Instead of large-scale endeavors involving most of the population, which as we argue, may be counterproductive to achieve cooperation, the joint combination of local agreements within groups that are smaller than the population at risk is prone to significantly raise the probability of success. In addition, our model predicts that, if one takes into consideration that groups of different sizes are interwoven in complex networks of contacts [2], the chances for global coordination in an overall cooperating state are further enhanced.

[1] Francisco C. Santos and Jorge M. Pacheco, "Risk of collective failure provides an escape from the tragedy of the commons", *Proc. Natl. Acad. Sci. USA* 108 (26), pp. 10421-5 (2011).
[2] Francisco C. Santos, Marta D. Santos and Jorge M. Pacheco, "Social diversity promotes the emergence of cooperation in public goods games", *Nature* 454, pp.213-216 (2008).

Nico Stollenwerk
Centro de Matematica e Aplicações Fundamentais, Lisbon University, Portugal
nico@ptmat.fc.ul.pt

Dynamic noise, chaos and parameter estimation in population biology

We revisit the parameter estimation framework for population biological dynamical systems, and apply it to calibrate various models in epidemiology with empirical time series, namely influenza and dengue fever. When it comes to more complex models like multi-strain dynamics to describe the virus-host interaction in dengue fever, even most recently developed parameter estimation techniques, like maximum likelihood iterated filtering, come to their computational limits. However, the first results of parameter estimation with data on dengue fever from Thailand indicate a subtle interplay between stochasticity and deterministic skeleton. The deterministic system on its own already displays complex dynamics up to deterministic chaos and coexistence of multiple attractors.

Ezio Venturino
Turin University, Italy
ezio.venturino@unito.it

The action of diffusivity on some recent ecoepidemic models

In this talk we will review some recently conceived models in ecoepidemiology and reformulate them explicitly introducing space in their description. We will investigate possible changes in the stability of their equilibria due to the diffusion processes inherent in this enlarged perspective.

Abstracts Invited Talks

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Carlos Braumann
Évora University, Portugal
braumann@uevora.pt

Population growth in random environments and consequences of an incorrect model specification

We consider stochastic differential equations to model the growth of a population in a randomly varying environment. These growth models are usually based on classical deterministic models, such as the logistic or the Gompertz model, taken as approximate models of the "true" (usually unknown) growth rate. We examine the effect of the model misspecification, more precisely the effect of the gap between the approximate and the "true" model, on model predictions, particularly on asymptotic behavior and mean and variance of the population time to extinction. This will shed some light whether we can trust, with some degree of accuracy, the predictions based on the approximate model, certainly a simpler model to deal with.

Jean Clairambault
INRIA Paris-Rocquencourt, LJLL Paris, and INSERM Villejuif, France
jean.clairambault@inria.fr

Modelling cell and tissue proliferation with applications to therapeutic optimisation in oncology

Rapid decrease of cell proliferation in fast renewing tissues is the result of the action of anticancer drugs. Such result is searched for in the case of tumours but avoided - which is seldom completely possible – in healthy tissues.

The targets, proliferating cell populations, cancerous or healthy, are described by models of population dynamics using physiologically structured partial differential equations (PDEs).

The means of action, drugs, are represented by their tissue concentrations and their effects on cells by physiologically based pharmacokinetic-pharmacodynamic (PBPKPD) ordinary differential equations (ODEs).

The continuous delivery of drugs is optimized under the constraint of limiting toxicity on healthy tissues by using numerical optimization algorithms that are technologically implementable in programmable pumps, that are portable by the patients and presently in use in the clinic.

Another constraint, linked to the limitation of emergence of drug resistance in cancer cell populations, another main pitfall of clinical treatments, harder to model, is a further challenge in oncology that has lately been faced by modeling.

References of articles on these subjects may be found on the page
http://www-roc.inria.fr/bang/JC/JCarticles.html

José Fernando Fontanari
Instituto de Física de São Carlos, São Paulo University, Brazil
fontanar@ifsc.usp.br

Analytical solution of the n-site approximation of the dynamics of the majority model for the spread of rumors in a chain

Understanding how opinions and, more generally, cultural traits disseminate through a population is crucial to fully comprehend the present organization of human society. Of particular interest here is the understanding of the mechanisms that lead to the appearance of stable domains characterized by distinct cultural traits, given that people's beliefs have a tendency to become more similar to each other's as people interact repeatedly. Here we study an extreme version of the frequency-dependent bias model in which an individual adopts the trait/opinion shared by the majority of its neighbors - the majority-vote rule model. We assume that the individuals are fixed in the sites of a chain of linear size L and that they can interact with their two nearest neighbors only. The state of a site $i=1,...,L$ is modeled by a binary variable that takes on the values $Si=0,1$ and changes only if it differs from those of its two neighbors, $i-1$ and $i+1$. Monte Carlo simulations indicate that the dynamics breaks the ergodicity by exhibiting an infinite number of absorbing configurations in the thermodynamic limit. Within a mean-field framework, we derive and solve analytically the equations of motion up to the 4-sites approximation. The breaking of ergodicity is manifested by the dependence of the steady-state solutions of the mean-field equations on the initial conditions, for which, as pointed above, we obtain exact analytic expressions. In addition, we claim that our expressions for the one site $\rho= <Si>$ and two-site $\Phi=<SiSi+1>$densities obtained using the 3 and 4 site approximations are exact since they do not change in these two approximation schemes.

Gabriela Gomes
Gulbenkian Institute of Science, Portugal
ggomes.igc@gmail.com

How host heterogeneity governs tuberculosis reinfection

Recurrent episodes of tuberculosis can be due to relapse of latent infection or exogenous reinfection, and discrimination is crucial for control planning. Molecular genotyping of Mycobacterium tuberculosis isolates offers concrete opportunities to measure the relative contribution of reinfection in recurrent disease. Here, a mathematical model of tuberculosis transmission is fitted to data from 14 molecular epidemiology studies enabling the estimation of relevant epidemiological parameters. Meta-analysis reveals that rates of reinfection after successful treatment are higher than rates of new tuberculosis, raising an important question about the underlying mechanism. We formulate two alternative mechanisms within our model framework: (1) infection increases susceptibility to reinfection; or (2) infection affects individuals differentially, thereby recruiting high-risk individuals to the group at risk for reinfection. The second mechanism is better supported by the fittings to the data suggesting that reinfection rates are inflated through a population phenomenon that occurs in the presence of heterogeneity in individual risk of infection. As a result, rates of reinfection are higher when measured at the population level even though they might be lower at the individual level. Finally, differential host recruitment is modulated by transmission intensity, being less pronounced when incidence is high.

Greg King
Lisbon University, Portugal
gkinglisboa@gmail.com

Testing turbulence theory using satellite measured ocean winds

An account of the trials and tribulations of working with satellite winds to test turbulence theory predictions of atmospheric mesoscale turbulence.

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Andrea Parisi
Lisbon University, Portugal
parisia@ptmat.fc.ul.pt

Age-dependent immune response and its consequences on antigenic drift

Recent research has shown that the human immune response is heterogeneous: young individuals have a monoclonal response, directed to specific antigen sites, while a polyclonal response developes around 4-5 years of age. We insert this heterogeneous immune response in a model for disease spread showing that this mechanism can not only explain the generation of escape mutants, but can also provide an explanation for the limited diversity observed in circulating influenza strains, their rapid drift and the regular appearence of dominant strains.

Hyun Mo Yang
Campinas University, Brazil
hyunyang@ime.unicamp.br

Incidence of dengue and temperature – Case study in Campinas City

A deterministic model is used to assess the incidence of dengue in Campinas City. The model considers temperature dependent entomological parameters [1] [2] [3], and also the transmission coefficients are allowed to depend on temperature. The model is simulated using the temperature records from 1990 to 2010 of Campinas City [4], situated in São Paulo State, Brazil.

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Abstracts Contributed Talks

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Maíra Aguiar
Lisbon University, Portugal
maira@ptmat.fc.ul.pt

How much complexity is needed to describe dengue haemorrhagic fever incidence data

Different extensions of the classical single-strain SIR model for the host population, motivated by modelling dengue fever epidemiology, have reported a rich dynamic structure including deterministic chaos which was able to describe the large fluctuations of disease incidences. A comparison between the basic two-strain dengue model, which already captures differences between primary and secondary infections, with the four-strain dengue model, that introduces the idea of competition of multiple strains in dengue epidemics shows that the difference between first and secondary infections drives the rich dynamics more than the detailed number of strains to be considered in the model structure. Chaotic dynamics were found to happen at the same parameter region of interest, for both the two and the four-strain models, able to explain the fluctuations observed in empirical data and showing a qualitatively good agreement between empirical data and model simulation. Since the law of parsimony favours the simplest of two competing models, the two-strain model would be the better candidate to be analysed, giving the expected complex behaviour to explain the fluctuations observed in empirical data, and indeed the better option for estimating all initial conditions as well as the few model parameters based on the available incidence data.

Nicolas Bacaer
IRD, France
nicolas.bacaer@ird.fr

The basic reproduction number for epidemics with seasonality

This will be a review of the definition, properties, and applications of the basic reproduction number for epidemic models with periodic coefficients. Emphasis will be put on vector-borne diseases and influenza.

Martín López García
Complutense University of Madrid, Spain
martin_lopez@mat.ucm.es

An approximating model for a two-species competition process

Our interest is in the stochastic model of a competition process analyzed by Ridler-Rowe [2], which is related to an ecosystem of two species competing for common resources. In this talk, we present an approximating model [1] based on percentiles of the maximum number of individuals in the ecosystem. Specifically, we study extinction probabilities, extinction times, and the numbers of births and deaths during an extinction cycle. This method is also applicable to the survival of a certain individual, for which we present three different models depending on the way the individuals are selected to die. All our results are illustrated with reference to simulated data.

References:

[1] A. Gomez-Corral, M. Lopez Garca, Extinction times and size of the surviving species in a two-species competition process, Journal of Mathematical Biology 64 (2012) 255-289.
[2] C.J. Ridler-Rowe, On competition between two species, Journal of Applied Probability 15 (1978) 457-465.

David Diego Castro
Instituto de Matemática Aplicada a la Ciencia y la Ingeniería (IMACI), UCLM, Spain
david.diego@uclm.es

Evolution towards critical fluctuations in a system of accidental pathogens

We put forward a model for cancer metastasis as a migration phenomenon between tumor cell populations coexisting and evolving in two different habitats. One of them is a primary tumor and the other one is a secondary or metastatic tumor. The evolution of the different cell phenotype populations in each habitat is described by means of a simple quasispecies model allowing for a cascade of mutations between the different phenotypes in each habitat. The cell migration event is supposed to be unidirectional and take place continuously in time. The possible clinical outcomes of the model depending on the parameter space are analyzed and the effect of the resection of the primary tumor is studied.

Martín López García
Complutense University of Madrid, Spain
martin_lopez@mat.ucm.es

An approximating model for a two-species competition process

Our interest is in the stochastic model of a competition process analyzed by Ridler-Rowe [2], which is related to an ecosystem of two species competing for common resources. In this talk, we present an approximating model [1] based on percentiles of the maximum number of individuals in the ecosystem. Specifically, we study extinction probabilities, extinction times, and the numbers of births and deaths during an extinction cycle. This method is also applicable to the survival of a certain individual, for which we present three different models depending on the way the individuals are selected to die. All our results are illustrated with reference to simulated data.

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[1] A. Gomez-Corral, M. Lopez Garca, Extinction times and size of the surviving species in a two-species competition process, Journal of Mathematical Biology 64 (2012) 255-289.
[2] C.J. Ridler-Rowe, On competition between two species, Journal of Applied Probability 15 (1978) 457-465.

Antonio Gomez-Corral
Complutense University of Madrid, Spain
antonio_gomez@mat.ucm.es

Maximum population sizes in host-parasitoid models

Overpopulation in host-parasitoid models has implications for species and habitat conservation. In this talk we are concerned with the problem of characterizing the distribution of the maximum number of individuals alive during a fixed time interval [0; t0] in host-parasitoid models, which is shown to have a matrix exponential form. For practical use, a fixed value t0 might amount to the epoch of a planned inspection. In a closed community, inspections are frequently scheduled in a periodic manner, and they may mean immigration from outside and slaughtering of a certain subset of remaining individuals, among other possible decisions to guarantee the survival of parasitoids and hosts, and the habitat conservation. We present simple conditions on the rates of change of population sizes for the matrix exponential solution to be explicit or algorithmically tractable. A particularly appealing feature of our solution based on splitting methods is that it allows us to obtain global error control. Based on numerical examples, we discuss on the use of the maximum number of individuals alive in an attempt to describe how a community of parasitoids and hosts is affected by extreme values.

Peyman Gaffari
Lisbon University, Portugal
peyman@ptmat.fc.ul.pt

Evolution towards critical fluctuations in a system of accidental pathogens

We investigate the by now so called SJ model [1] not only in its simple formulation as recently used, but an extended version, the SJ model version II. In this we find the system to evolve to low pathogenicity causing large critical fluctuations without tuning the control parameter, a self-organization of criticality.

Reference:
[1] Pinto, O.A., & Mu˜noz, M.A. (2011). Quasi-neutral theory of epidemic outbreaks, PLoS ONE 6, e21946.

Sandra Lagarto
Évora University, Portugal
sdlagarto@gmail.com

ModelLing death rates with stochastic differential equations

When analyzing demographic data across time, instead of doing a cohort data analysis, we must consider random environmental fluctuations which influence mortality at all ages, on both males and females. That way, we can see that death rates had a strong decreasing trend during the last century. Considering these facts, we apply Stochastic Differential Equations (with a long tradition of application on finance and, more recently, on animal growth models) to death rates.

First, we started with very simple models, like a Geometric Brownian Motion Model or a Generalize Stochastic Gompertz Model, applied to a certain age and sex.

Then, we notice that, frequently, mortality increases and decreases occur for males and females of the same age group at the same time period. This similar death behavior between the two genders suggests that the SDE driving Wiener processes of the two genders must also be correlated.

We show, in particular, an application of a bidimensional Stochastic Gompertz Model to death rates of Portuguese population (1940 to 2009). Results show significant differences, per age, when comparing the complete model with the model without correlation effect.

Torsten Lindström
Division of Communications and Transport Systems, Department of Science and Technology, Linköping University, Sweden
torsten.axel.lindstrom@liu.se

Detecting chaos requires careful analysis of nearly periodic data

We show that models fitted to data in many cases fit unstable periodic solutions to attracting periodic solutions of the 'true model' that generated the data. An attracting solution containing the neighborhood of the fitted unstable solution in its domain of attraction may possess entirely different dynamical properties. Thus, an attracting chaotic solution with positive Lyapunov exponent may describe periodic solutions with negative Lyapunov exponents and vice versa. These problems can in principle be remedied, if the fitted models would be allowed to contain an arbitrary complexity and if an infinite amount of data would be available. We claim that we stay far from such limits in ecology, for instance. Therefore, we think our approach is essential to bear in mind when making data-based predictions concerning dynamical behavior. Our general conclusion is that less data is required in nearly periodic cases than in chaotic cases for rejecting models not allowing complex behavior.

José Lourenço
University of Oxford, UK & PDBC-IGC, Portugal
lourenco.jml@gmail.com

Host-population structure and dengue fever

The recent history of dengue has been marked by increasing epidemic activity and the emergence of the severe forms of the disease. Failure to control dengue virus (DENV) in endemic regions and to prevent globalized distribution of the vector-species and pathogen variants (serotypes 1-4) underline the urgency for reassessment of previous research methods, hypothesis and empirical observations.

Despite significant observations on the importance of the spatio-demographic context in which transmission is achieved, past theoretical studies have generally neglected these factors and instead focused on possible within-host biological drivers for the observed incidence and serotype patterns.

On the light of preliminary results from an individual-based model of dengue transmission, I intend to demonstrate how host-population structure might drive some of the general patterns of the disease.

Luis Mateus
Lisbon University, Portugal
luisgam1@yahoo.com

Stochastic Models for Population Biology: From Dynamic Noise to Bayesian Description

The purpose of the seminar is the application of methods from stochastic analysis to two models from population biology. We give analytical solutions of the models, construct the likelihood function from the model solution, estimate the maximum likelihood and give the Bayesian description of the problem. We test numerically the analytical results on simulated data. The present models and concepts can be applied to real world epidemiological data such as influenza.

Miguel Nogueira
Lisbon University, Portugal & Duke University, USA
miguelvnogueira@gmail.com

Stochastic downscaling of numerically simulated spatial rain and cloud fields using a transient multifractal approach

Atmospheric fields can be extremely variable over wide ranges of spatial scales, with a scale ratio of 109-1010 between largest (planetary) and smallest (viscous dissipation) scale. Furthermore atmospheric fields with strong variability over wide ranges in scale most likely should not be artificially split apart into large and small scales, as in reality there is no scale separation between resolved and unresolved motions. Usually the effects of the unresolved scales are modeled by a deterministic bulk formula representing an ensemble of incoherent sub-grid processes on the resolved flow. This is a pragmatic approach to the problem and not the complete solution to it. These models are expected to under-represent the small-scale spatial variability of both dynamical and scalar fields due to implicit and explicit numerical diffusion as well as physically based sub-grid scale turbulent mixing, resulting in smoother and less intermittent fields as compared to observations. Thus, a fundamental change in the way we formulate our models is required. Stochastic approaches equipped with a possible realization of sub-grid processes and potentially coupled to the resolved scales over the range of significant scale interactions range provide one alternative to address the problem. Stochastic multifractal models based on the cascade phenomenology of the atmosphere and its governing equations in particular are the focus of this research. Previous results have shown that rain and cloud fields resulting from both idealized and realistic numerical simulations display multifractal behavior in the resolved scales. This result is observed even in the absence of scaling in the initial conditions or terrain forcing, suggesting that multiscaling is a general property of the nonlinear solutions of the Navier-Stokes equations governing atmospheric dynamics. Our results also show that the corresponding multiscaling parameters for rain and cloud fields exhibit complex nonlinear behavior depending on large scale parameters such as terrain forcing and mean atmospheric conditions at each location, particularly mean wind speed and moist stability. A particularly robust behavior found is the transition of the multiscaling parameters between stable and unstable cases, which has a clear physical correspondence to the transition from stratiform to organized (banded) convective regime. Thus multifractal diagnostics of moist processes are fundamentally transient and should provide a physically robust basis for the downscaling and sub-grid scale parameterizations of moist processes. Here, we investigate the possibility of using a simplified computationally efficient multifractal downscaling methodology based on turbulent cascades to produce statistically consistent fields at scales higher than the ones resolved by the model. Specifically, we are interested in producing rainfall and cloud fields at spatial resolutions necessary for effective flash flood and earth flows forecasting.

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Flávio L. Pinheiro
Lisbon University, Portugal
flavio.lpp@gmail.com

From Local to Global Dilemmas in Social Networks

Social networks affect in such a fundamental way the dynamics of the population they support that the global, population-wide behavior that one observes often bears no relation to the individual processes it stems from. Up to now, linking the global networked dynamics to such individual mechanisms has remained elusive. Here we study the evolution of cooperation in networked populations and let individuals interact via a 2-person Prisoners Dilemma – a characteristic defection dominance social dilemma of cooperation. We show how homogeneous networks transform a Prisoner's Dilemma into a population-wide evolutionary dynamics that promotes the coexistence between cooperators and defectors, while heterogeneous networks promote their coordination. To this end, we define a dynamic variable that allows us to track the self-organization of cooperators when co-evolving with defectors in networked populations. Using the same variable, we show how the global dynamics — and effective dilemma — co-evolves with the motifs of cooperators in the population, the overall emergence of cooperation depending sensitively on this co-evolution.

Eugene Postnikov
Kursk State University, Russia
postnicov@gmail.com

Synchronization of reaction rate and epidemic oscillations and 1:1 resonance within SIRS model

The development of models, which allow to reproduce seasonality of respiratory diseases (common cold, influenza, RSV, etc) is still an actual problem of mathematical epidemiology. The common basic model for this kind of diseases is SIRS model describing population with temporal immunity and variable reaction rate, which can originate from meteorological (air temperature, humidity, illumination) as well as social (e.g. schooltime year schedule, which is important for a study of epidemics among children) conditions. But the general observation is a close coincidence of this period with epidemic seasonality and their phase synchronization.

In the present work it has been shown that there exists the change of variables, which explicitly represents SIRS equations with a time-varying reaction rate as the second-order non-homogeneous ODE, i.e. this oscillating parameter actually leads to an emergence leading terms playing a role of an outer excitation. This explains analytically 1:1 resonance in the case of weak harmonic variation of the reaction rate that was reported recently by various authors as a result of Markov chains simulations and processing of real medical and climate data.

Nadia Raissi
University Mohamed V Rabat Agdal, Marroco
nadiaraissi11@gmail.com

Modelling sardine fishery along the Atlantic coast of Morocco

We propose a dynamic model governing the evolution with respect to time of the sardine (Sardina pilchardus) stock in the Atlantic Moroccan coast, which is harvested by two different fishing fleets. We assume that the sardine stock is distributed on two closed zones and targeted by a composite fleet: The Moroccan purse seiners which can move between the fishing regions and a fleet of deepsea trawlers composed mainly of foreign vessels and operating in the southern zone. The model is a set of Ordinary Differential Equations (ODE) describing the sardine evolutions in its 2 zones of distribution, and the dynamics of the fishing efforts. We present an aggregation method which permits to reduce the system to a set of 4 ODE, and then we study analytically the existence and the asymptotic behaviors of the equilibrium points. The parameters of the stock dynamic and harvest are estimated using models fitting techniques based on a set of available data. The conditions leading to sustainable fisheries are then simulated. Interpretations of our results can be used by the authority responsible of fishery management, that is the Moroccan government, as general recommendations for a better management of Moroccan fisheries.

Paula Rodrigues
Departamento de Matemática and Centro de Matemática e
Aplicações, Universidade Nova de Lisboa, Portugal
pcpr@fct.unl.pt

Synchronization of reaction rate and epidemic oscillations and 1:1 resonance within SIRS model

Tuberculosis (TB) control programs recommend a treatment strategy that consists on a 6- to 8-month regimen. The long duration of treatment seems to be related to a high treatment default rate, which ranges from 6% to 30%. Treatment default contributes to treatment failure and increases the risk of multidrug-resistant TB. Some efforts based on new drugs have been done to reduce the length of TB treatment. Here we use a stylized model structure to explore, systematically, the effects of varying treatment duration and default rate. We find that shortening treatment alone may not reduce TB prevalence, indicating the necessity of complementation with increased adherence to treatment (lower default rate). Even then, efficacy of the intervention decays for higher transmission intensities. Results depend mainly on the assumptions made for the class of individuals that are under treatment. TB literature is not consistent on which should be these assumptions and this is not discussed in prior models. We hope that the use of simple mechanistic mathematical models can help to clarify important biological questions.

Filipe Rocha
Lisbon University, Portugal
filipe.pinho.rocha@gmail.com

TBA

Ganna Rozhnova
University of Lisbon, Portugal & University of Manchester, UK
a_rozhnova@cii.fc.ul.pt

Stochastic oscillations in models of epidemics on a network of cities

In this talk we outline a framework for investigating the nature of stochastic oscillations in a susceptible-infected-recovered model of disease spread on a network of n cities. In the model a fraction f_{jk} of individuals from city k commute to city j, where they may infect, or be infected by, others. Starting from a continuous time Markov description of the model the deterministic equations, which are valid in the limit when the population of each city is infinite, are recovered. The stochastic fluctuations about the fixed point of these equations are derived by use of the van Kampen system-size expansion. We show that the fixed point structure of the deterministic equations is remarkably simple: a unique non-trivial fixed point always exists and has the feature that the fraction of susceptible, infected and recovered individuals is the same for each city irrespective of its size. We find that the stochastic fluctuations have an analogously simple dynamics: all oscillations have a single frequency, equal to that found in the one city case. We interpret this phenomenon in terms of the properties of the spectrum of the matrix of the linear approximation of the deterministic equations at the fixed point.

Cristrana J. Silva
Department of Mathematics, University of Aveiro, Portugal
cjoaosilva@ua.pt

Optimal control strategies for tuberculosis

We apply optimal control theory to a tuberculosis (TB) model where reinfection and post-exposure interventions are considered. We propose optimal control strategies for the minimization of the number of active TB infectious and persistent latent individuals, taking into account the cost of the measures for the treatment of these individuals. We show that optimal control strategies reduce effectively both the fraction of active infectious and persistent latent individuals.

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Max Souza
Universidade Federal Fluminense, Brazil
max.souza@gmail.com

Modelling urban arboviruses epidemics

Data from Dengue epidemics suggest that the transport of population can be very important in the infection dynamics. We shall describe a multi-group model that seems to be able to capture the qualitative dynamics of the data. The model was calibrated to epidemics time series in Rio de Janeiro, and used to predict the outcome of an initial infection by DEN4. Also we shall show some results on multi scaling vector-host dynamics.

Daniel Sánchez-Taltavull
Centre de Recerca Matemàtica, Spain
Dsanchez@crm.cat

Stability of Stochastic Models of hierarchical cell populations

Tissues in higher organisms exhibit a hierarchical structure where only a small number of stem cells have the potential for indefinite division. The descendants of the stem cell population undergo a process of maturation where, after a series of intermediate differentiation steps, a fully matured cellular type emerges. In general, mature cells have no replicative potential whereas cells belonging to the intermediate stages gradually loose their stem cell-like properties. Acquiring a proper understanding of this process is important for many reasons but one of the more pressing issues is related to the fact that disregulation of the maturation process leads to cancer. However, many questions related to the regulation of this process remain unclear. Our aim is to examine some of these open questions. Specifically, it has been suggested that the number of differentiation stages between self-renewing stem cells and fully-differentiated cells plays a major role in the evolutionary stability of cell populations. We will formulate a stochastic model of stem cell maturation with resource limitation to account for homeostatic constraints. Here we examine how the introduction of symmetric stem cell division affects the stability of the whole population.

Vítor V. Vasconcelos
Lisbon University, Portugal
vvlvasconcelos@gmail.com

Co-evolution of sanctioning and cooperation under collective dilemmas

Preventing global warming is a public good requiring overall cooperation. Inspired by the subject of world climate agreements, we resort to the tools of evolutionary game theory to understand the impact of punishment in global climate policy. We investigate behavioral dynamics in finite populations and show how institutions provide a significant increase in cooperation. We argue that the creation of local supervision institutions, instead of global ones, may further enhance cooperation in public goods dilemmas.

Jeromos Vukov
Lisbon University, Portugal
jvukov@gmail.com

Incipient Cognition Solves the Spatial Reciprocity Conundrum of Cooperation

From the simplest living organisms to human societies, cooperation among individuals emerges as a paradox difficult to explain and describe mathematically, although very often observed in reality. Evolutionary game theory offers an excellent toolbar to investigate this issue. Spatial structure has been one of the first mechanisms promoting cooperation; however, alone it only opens a narrow window of viability. Here we equip individuals with incipient cognitive abilities, and investigate the evolution of cooperation in a spatial world where retaliation, forgiveness, treason and mutualism may coexist, as individuals engage in Prisoner's Dilemma games. In the model, individuals are able to distinguish their partners and act towards them based on previous interactions. In this talk, I show how the simplest level of cognition, alone, can lead to the emergence of cooperation. Despite the incipient nature of the individuals' cognitive abilities, cooperation emerges for unprecedented values of the temptation to cheat, being also robust to invasion by cheaters, errors in decision making and inaccuracy of imitation, features akin to many species, including humans.

Stefan Wieland
Lisbon University, Portugal
swieland@cii.fc.ul.pt

Disease Awareness in Adaptive Networks

Complex networks - multiple agents ("nodes") engaging in structured interactions ("links" connecting nodes) - have become a prominent paradigm in tackling complex systems. Adaptive networks, allowing for the coevolution of network structure and node-state dynamics, are suitable frameworks for modeling the interplay of disease awareness and epidemic spreading in a population.

Several adaptive epidemiological models with a cyclic sequence of states display an active phase with perpetual node-state and link dynamics. Simulations show that in this active phase, the interplay of node-state dynamics and link rewiring often prompts the convergence towards constant node-state densities and well-defined degree distributions, irrespective of the initial network topology.

Taking the adaptive SIS model of Gross et al. as an example, we present an analytic method to detect and describe these dynamic equilibria, accurately predicting characteristic steady-state degree distributions and other network measures. We find that slight modifications of the standard rewiring rule can result in either minuscule or drastic changes of steady-state network topologies. The method applies to a broad class of systems and can be used to determine the steady-state topology of many other adaptive networks.

Abstracts Posters

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Maira Aguiar
Lisbon University, Portugal
maira@ptmat.fc.ul.pt

The role of seasonality and import in a minimalistic multi-strain dengue model capturing differences between primary and secondary infections

Our study focuses on a seasonally forced model with temporary cross immunity and possible secondary infection, motivated by dengue fever epidemiology. The notion of at least two different strains is needed in a minimalistic model to describe differences between primary infections, often asymptomatic, and secondary infection, associated with the severe form of the disease. We extend the previously studied non-seasonal model by adding seasonal forcing, mimicking the vectorial dynamics, and a low import of infected individuals, which is realistic in the dynamics of dengue fever epidemics. A comparative study between three different scenarios (non-seasonal, low seasonal and high seasonal with a low import of infected individuals) is performed.

Nuno M. Brites
Centro de Investigação em Matemática e Aplicações, Évora University, Portugal
d7488@alunos.uevora.pt

gSDE: a new software for stochastic growth models

Models for the growth of individual organisms living in random environments were developed by Filipe and Braumann (2008). The general model considered admits the form $dY(t) = b(a-Y(t))dt + dW(t)$, with $Y(t) = g(X(t))$, where g is a strictly increasing continuously didifferentiable function (which we assume known) and $X(t)$ is the individual size at age t; bis the rate of approach to maturity, $a = g(A)$ with a the average size at maturity, is the effect of random environmental fluctuations and $W(t)$ is the standard Wiener process. The function g can be chosen leading to a variety of classical stochastic models: $g(x) = xc$; $c > 0$, for the Bertalany-Richards model; $g(x) = \ln(x)$, for the Gompertz model and $g(x) = x^{-1}$, for the logistic model, among many others. The properties of the general model, with fixed parameters a and b are known. We have extended the model to the case of different growth coefficients along the evolution of the individual growth (Filipe et al, 2010) and also considered the case where a is a random variable varying among individuals.

For these stochastic deferential equation (SDE) models, we are interested in the first passage time through a high threshold value $Q > y0$ for animal size $X(t)$. This can be a desirable market sale size in livestock production, for instance. This is equivalent to determine the first passage time of $Y(t) = g(X(t))$ by $Q = g(Q^*)$. We denote this time by TQ $=\inf\{t>0: Y(t) = Q\}$. For these models, explicit expressions for the mean and variance of TQ can be found in Braumann et al (2009).

In this work, we will present a friendly software that includes and manages all these (and other) models. The gSDE software allows any person (even with low computer knowledge) to apply these models, as well as new models proposed by users, to their data by a very simply user interface. Some of its capabilities are maximum likelihood parameter estimation, non-parametric estimation, data fitting and data prediction. Other capabilities are under development. The user can save all the results to a spreadsheet file, plot and save the most relevant graphics and use their own R algorithm. It has been developed in Microsoft Visual Studio 2010 with the Microsoft Visual C# programming language and can be easily installed in any regular personal computer with a CD-ROM or a setup file available in the project website. All the algorithms were created with the R programming language.

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Peyman Gaffari
Lisbon University, Portugal
peyman@ptmat.fc.ul.pt

Evolution towards critical fluctuations in a system of accidental pathogens

We investigate the by now so called SJ model [1] not only in its simple formulation as recently used, but an extended version, the SJ model version II. In this we find the system to evolve to low pathogenicity causing large critical fluctuations without tuning the control parameter, a self-organization of criticality.

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Andrzej Jarynowski
Jagiellonian University, Poland
andrzej.jarynowski@uj.edu.pl

The dynamics of divorces: socio-economic models of different societies

In particular, population dynamic of getting divorced depends on many global factors such as: social norms, economy, law or demographics; and individual as: level of interpersonal or problem-solving skills of the spouses. We try to find such a relationship by taking into account only quantitative variables, test theoretical models considering phase transition between coupling (married) and free (single) preferential states as a function of social and economic pressure. The analyzed data has been collected by UN across almost all the countries since 1948. Our first approach is followed by Bouchaud's model of collective behavior*, which works well with dynamics of fertility rates in postwar Europe. Unfortunately, we postulate that this pure sociological approach fails in general. Thus, we did some observation about why it went wrong and where economy (e. g. Poland) or law (e. g. Portugal) has bigger impact on getting divorce than social pressure.

José Lourenço
University of Oxford, UK & PDBC-IGC, Portugal
lourenco.jml@gmail.com

The invasion dynamics of novel advantageous dengue genotyqes

Dengue fever (DF) and tpe more severe dengue paemorrpagic fever (DHF) and dengue spock syndrome (DSS) are mosquito borne viral infections wpicp pave seen a major increase in terms of global distribution and total case numbers over tpe last few decades. There are currently four antigenically distinct and qotentially co-circulating dengue serotUqes and eacp serotyqe spows substantial genetic diversity, organised into qhylogenetically distinct lineages (clades). While there is some evidence for positive selection, the evolutionary dynamics of dengue virus (DENV) is supposed to be mostly dominated by purifying selection due to the constraints imposed by its two-host life-cycle. Motivated by a recent genotype replacement event whereby the resident American/Asian lineage of dengue virus serotype 2 (DENV2) had been displaced by the fitter Asian-1 lineage, we investigated some of the epidemiological factors that might determine the success and invasion dynamics of a novel advantageous dengue genotype. We have shown that even advantageous DENV genotypes can undergo periods of several years prior reaching sufficiently large population sizes to escape the risk of extinction. Our results thus indicate that in addition to purifying selection, the epidemiological landscape and stochastic effects might be equally important determinants in shaping the viral evolutionary ecology [1,2].

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Telmo Peixe
Lisbon University, Portugal
telmopeixe@gmail.com

Rank of Stably Dissipative Graphs

For the class of stably dissipative Lotka-Volterra systems we prove that the rank of its defining matrix is completely determined by the system's graph.

Silvia Martorano Raimundo
School of Medicine, University of São Paulo, Brazil
silviamr@dim.fm.usp.br

Modelling the emergence of HVI-1 drug resistance resulting from antiretroviral therapy: insights from theoretical and numerical studies

The use of antiretroviral therapy has proven to be remarkably effective in controlling the progression of human immunodeficiency virus (HIV) infection and prolonging patient's survival. Therapy however may fail and therefore these benefits can be compromised by the emergence of HIV strains that are resistant to the therapy. In view of these facts, the question of finding the reason for which drug-resistant strains emerge during therapy has become a worldwide problem of great interest. We present a deterministic HIV-1 model to examine the mechanisms underlying the emergence of drug-resistance during therapy. The aim of this study is to determine whether, and how fast, antiretroviral therapy may determine the emergence of drug resistance by calculating the basic reproductive numbers. The existence, feasibility and local stability of the equilibriums are also analyzed. By performing numerical simulations we show that Hopf bifurcation may occur. The model suggests that the individuals with drug-resistant infection may play an important role in the epidemic of HIV.

Filipe Rocha
Lisbon University, Portugal
filipe.pinho.rocha@gmail.com

The influence of the vector Aedes aegypti in dynamics of the epidemiology of dengue fever

In this work, we investigate a first epidemiological model (SIRUV) for dengue fever spreading including the transmission via a vector.

Nico Stollenwerk
Lisbon University, Portugal
nico@ptmat.fc.ul.pt

Parameter estimation in epidemiology: from simple to complex dynamics

We revisit the parameter estimation framework for population biological dynamical systems, and apply it to calibrate various models in epidemiology with empirical time series, namely influenza and dengue fever. When it comes to more complex models like multi-strain dynamics to describe the virus-host interaction in dengue fever, even most recently developed parameter estimation techniques, like maximum likelihood iterated filtering, come to their computational limits. However, the first results of parameter estimation with data on dengue fever from Thailand indicate a subtle interplay between stochasticity and deterministic skeleton. The deterministic system on its own already displays complex dynamics up to deterministic chaos and coexistence of multiple attractors.

Abstracts Invited Talks

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Carlos Braumann
Évora University, Portugal
braumann@uevora.pt

Population growth in random environments and consequences of an incorrect model specification

We consider stochastic differential equations to model the growth of a population in a randomly varying environment. These growth models are usually based on classical deterministic models, such as the logistic or the Gompertz model, taken as approximate models of the "true" (usually unknown) growth rate. We examine the effect of the model misspecification, more precisely the effect of the gap between the approximate and the "true" model, on model predictions, particularly on asymptotic behavior and mean and variance of the population time to extinction. This will shed some light whether we can trust, with some degree of accuracy, the predictions based on the approximate model, certainly a simpler model to deal with.

Jean Clairambault
INRIA Paris-Rocquencourt, LJLL Paris, and INSERM Villejuif, France
jean.clairambault@inria.fr

Modelling cell and tissue proliferation with applications to therapeutic optimisation in oncology

Rapid decrease of cell proliferation in fast renewing tissues is the result of the action of anticancer drugs. Such result is searched for in the case of tumours but avoided - which is seldom completely possible – in healthy tissues.

The targets, proliferating cell populations, cancerous or healthy, are described by models of population dynamics using physiologically structured partial differential equations (PDEs).

The means of action, drugs, are represented by their tissue concentrations and their effects on cells by physiologically based pharmacokinetic-pharmacodynamic (PBPKPD) ordinary differential equations (ODEs).

The continuous delivery of drugs is optimized under the constraint of limiting toxicity on healthy tissues by using numerical optimization algorithms that are technologically implementable in programmable pumps, that are portable by the patients and presently in use in the clinic.

Another constraint, linked to the limitation of emergence of drug resistance in cancer cell populations, another main pitfall of clinical treatments, harder to model, is a further challenge in oncology that has lately been faced by modeling.

References of articles on these subjects may be found on the page http://www-roc.inria.fr/bang/JC/JCarticles.html

José Fernando Fontanari
Instituto de Física de São Carlos, São Paulo University, Brazil
fontanar@ifsc.usp.br

Analytical solution of the n-site approximation of the dynamics of the majority model for the spread of rumors in a chain

Understanding how opinions and, more generally, cultural traits disseminate through a population is crucial to fully comprehend the present organization of human society. Of particular interest here is the understanding of the mechanisms that lead to the appearance of stable domains characterized by distinct cultural traits, given that people's beliefs have a tendency to become more similar to each other's as people interact repeatedly. Here we study an extreme version of the frequency-dependent bias model in which an individual adopts the trait/opinion shared by the majority of its neighbors - the majority-vote rule model. We assume that the individuals are fixed in the sites of a chain of linear size L and that they can interact with their two nearest neighbors only. The state of a site $i=1,...,L$ is modeled by a binary variable that takes on the values $Si=0,1$ and changes only if it differs from those of its two neighbors, $i-1$ and $i+1$. Monte Carlo simulations indicate that the dynamics breaks the ergodicity by exhibiting an infinite number of absorbing configurations in the thermodynamic limit. Within a mean-field framework, we derive and solve analytically the equations of motion up to the 4-sites approximation. The breaking of ergodicity is manifested by the dependence of the steady-state solutions of the mean-field equations on the initial conditions, for which, as pointed above, we obtain exact analytic expressions. In addition, we claim that our expressions for the one site $\rho= <Si>$ and two-site $\Phi=<SiSi+1>$densities obtained using the 3 and 4 site approximations are exact since they do not change in these two approximation schemes.

Gabriela Gomes
Gulbenkian Institute of Science, Portugal
ggomes.igc@gmail.com

How host heterogeneity governs tuberculosis reinfection

Recurrent episodes of tuberculosis can be due to relapse of latent infection or exogenous reinfection, and discrimination is crucial for control planning. Molecular genotyping of Mycobacterium tuberculosis isolates offers concrete opportunities to measure the relative contribution of reinfection in recurrent disease. Here, a mathematical model of tuberculosis transmission is fitted to data from 14 molecular epidemiology studies enabling the estimation of relevant epidemiological parameters. Meta-analysis reveals that rates of reinfection after successful treatment are higher than rates of new tuberculosis, raising an important question about the underlying mechanism. We formulate two alternative mechanisms within our model framework: (1) infection increases susceptibility to reinfection; or (2) infection affects individuals differentially, thereby recruiting high-risk individuals to the group at risk for reinfection. The second mechanism is better supported by the fittings to the data suggesting that reinfection rates are inflated through a population phenomenon that occurs in the presence of heterogeneity in individual risk of infection. As a result, rates of reinfection are higher when measured at the population level even though they might be lower at the individual level. Finally, differential host recruitment is modulated by transmission intensity, being less pronounced when incidence is high.

Greg King
Lisbon University, Portugal
gkinglisboa@gmail.com

Testing turbulence theory using satellite measured ocean winds

An account of the trials and tribulations of working with satellite winds to test turbulence theory predictions of atmospheric mesoscale turbulence.

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Andrea Parisi
Lisbon University, Portugal
parisia@ptmat.fc.ul.pt

Age-dependent immune response and its consequences on antigenic drift

Recent research has shown that the human immune response is heterogeneous: young individuals have a monoclonal response, directed to specific antigen sites, while a polyclonal response developes around 4-5 years of age. We insert this heterogeneous immune response in a model for disease spread showing that this mechanism can not only explain the generation of escape mutants, but can also provide an explanation for the limited diversity observed in circulating influenza strains, their rapid drift and the regular appearence of dominant strains.

Hyun Mo Yang
Campinas University, Brazil
hyunyang@ime.unicamp.br

Incidence of dengue and temperature – Case study in Campinas City

A deterministic model is used to assess the incidence of dengue in Campinas City. The model considers temperature dependent entomological parameters [1] [2] [3], and also the transmission coefficients are allowed to depend on temperature. The model is simulated using the temperature records from 1990 to 2010 of Campinas City [4], situated in São Paulo State, Brazil.

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