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Mathématique et Informatique Fondamentale de Lyon

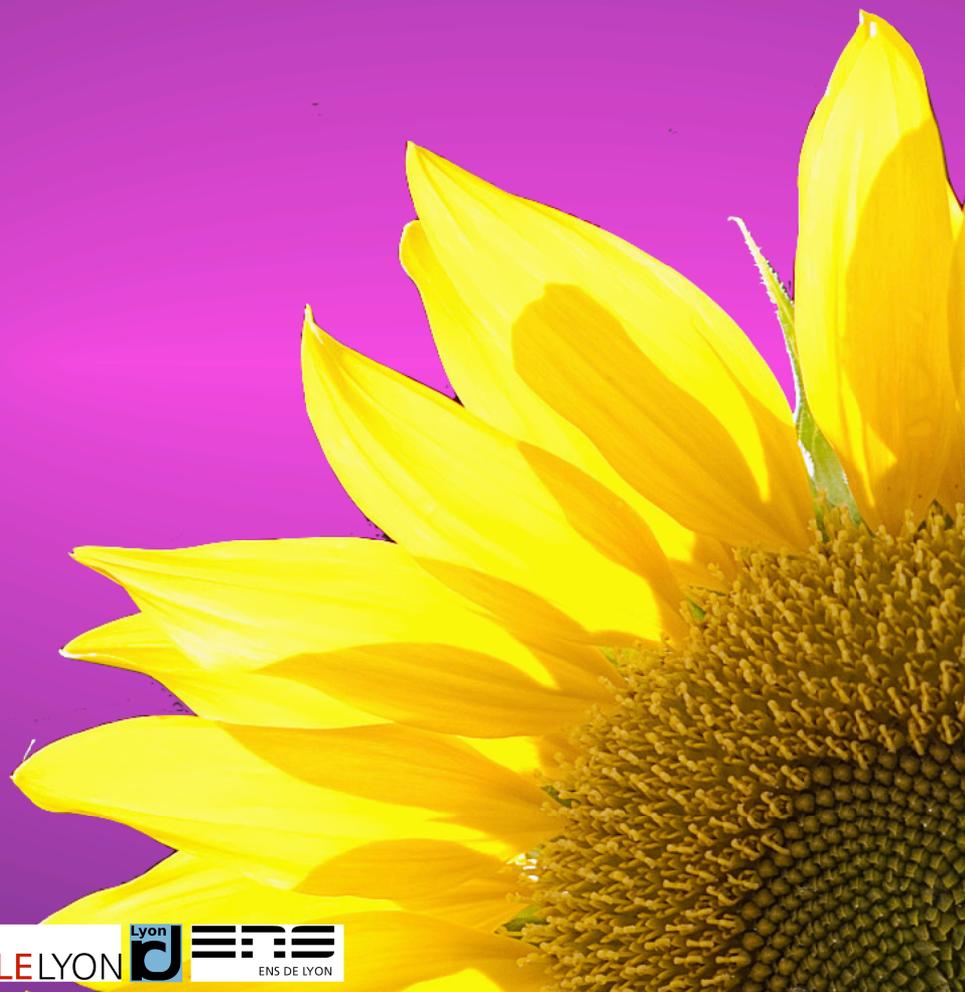
UNIVERSITÉ DE LYON

# BIOMATH SPRING LYON

## CONFERENCE

### “Biological Invasions and Evolutionary Biology Stochastic and Deterministic Models”

March 11-15, 2013



<http://mathbio2013.sciencesconf.org/>



# Supports

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Graphic design : **Laurent Pujou-Menjouet** (Université Claude Bernard, Lyon 1)

www : **Emeric Bouin** (ENS de Lyon)

	Monday	Tuesday	Wednesday	Thursday	Friday
09:00-09:45		<b>MELEARD</b>	<b>VEBER</b>	<b>DIEKMANN</b>	<b>CHALUB</b>
09:45-10:30	Welcome	<b>BEARDMORE</b>	<b>BURGER</b>	<b>MARTIN</b>	<b>ROQUES</b>
10:30-11:00	Welcome	Coffee break	Coffee break	Coffee break	Coffee break
11:00-11:45	<b>FERRIERE</b>	<b>AGUILEE</b>	<b>RONCE</b>	<b>JONES</b>	<b>PHILLIPS</b>
11:45-12:30	<b>JABIN</b>	<b>BERESTYCKI</b>	<b>NADIN</b>	<b>DEBARRE</b>	<b>DESVILLETES</b>
12:30-15:00	Lunch time	Lunch time	Lunch time	Lunch time	Lunch time
15:00-15:45	<b>GANDON</b>	<b>CHAMPAGNAT</b>	<i>Free afternoon (Guided tour in Old City)</i>		
15:45-16:30	<b>MIRRAHIMI</b>	<b>WAXMAN</b>			
16:30-17:00	Coffee break	Coffee break	Coffee break	Coffee break	
17:00-17:45	<b>MAGAL</b>	<b>MEETING ARP (17:00-19:00)</b>	<b>POSTER SESSION (17:00-19:00)</b>		

# Abstracts of the talks

- Robin Aguilée (University Paul Sabatier, Toulouse, France)

## **Title: How does pollen versus seed dispersal affect niche evolution?**

**Abstract:** In heterogeneous environments, the genetic and demographic consequences of dispersal influence the evolution of niche width. Unless pollen is limiting, pollen dispersal does not contribute directly to population growth. However, by disrupting local adaptation, it indirectly affects population dynamics. We compare the effect of pollen versus seed dispersal on the evolution of niche width in heterogeneous habitats, explicitly considering the feedback between maladaptation and demography.

We first consider two scenarios: the secondary contact of two subpopulations, in distinct, formerly isolated habitats, and the invasion of an empty habitat, with dispersal between the new and ancestral habitat. With an analytical model, we identify critical levels of genetic variance leading to niche contraction (secondary contact scenario), or expansion (new habitat scenario). We confront these predictions with simulations where the genetic variance freely evolves. Niche contraction occurs when habitats are very different. It is faster as total gene flow increases or as pollen predominates in overall gene flow. Niche expansion occurs when habitat heterogeneity is not too high. Seed dispersal accelerates it, whereas pollen dispersal tends to retard it. In both scenarios very high seed dispersal leads to extinction. Overall, the results of this model predict a wider niche for species dispersing seeds more than pollen.

We then consider a population living on a continuous environmental gradient shifting in time, mimicking e.g. climate change. We analyse how combinations of adaptation and dispersal allow the persistence of the population, and how pollen and seed dispersal differentially affect niche width (measured by range size). Preliminary results of this second model will be shown.

- Robert Beardmore (University of Exeter, UK)

## **Title: Antibiotic interactions and a multi-drug resistance mutation consisting of 10% of E.coli's genome.**

**Abstract:** Antibiotic efflux is often said not to be clinically relevant but recent evidence shows that MRSA and TB do efflux the drugs we use to treat them. It is therefore important that we understand how efflux affects important measures of antibiotic efficacy. Now, interactions between antibiotics can be hard to pin down. The idea of synergy between two drugs is a natural one, each small molecule enhances the killing efficacy of the other, but one of the standard treatments for MRSA uses a combination of rifampin and vancomycin where different strains show different antibiotic interactions for this drug pair at 24h and 48h. Why should this be? The purpose of this talk is to apply a mathematical modelling and functional genomics approach to show that we should expect antibiotic interactions to be dynamic and dependent upon, for example, the frequency of key resistance genes in the population. Using a model system of E.coli K12 with the translational inhibitors erythromycin and doxycycline, whole-genome sequencing shows that the rapid up-regulation and genetic duplication of the efflux pump *acrAB-tolC* can morph what looks like a synergism into an antagonism within 48h. We then perform experiments to show that this lab model, provided the treatment is sufficiently long, produces fewer bacterial cells when these "synergistic drugs" are never in physical contact in the environment that supports the cells. In short, it appears that our measures of antibiotic synergy are not robust to drug-resistance adaptation. This is joint work with Hinrich Schulenberg, Gunther Jansen, David Laenemann, Ayari Fuentes and Rafael Pena Miller.

- Henri Berestycki (EHESS, France)

**Title: The effect of a line with fast diffusion on biological invasions**

**Abstract:** In this talk I will present a system of equations for the effect of a "road" with a fast diffusion on biological invasions. A classical Fisher-KPP propagation is assumed outside of the road. It is found that past a certain threshold for the ratio of diffusivities, the presence of the road enhances the global propagation. Several other effects such as transport or reaction on the "road" are discussed. I will also derive the asymptotic behavior of the invasion speed depending on the various parameters. I will report on joint works with Jean-Michel Roquejoffre and Luca Rossi.

- Reinhard Bürger (University of Vienna, Austria)

**Title: Consequences of genetic architecture for local adaptation under gene flow: a two-locus model**

**Abstract:** In subdivided populations, adaptation to a local environment may be hampered by maladaptive gene flow from other subpopulations. At an isolated locus, i.e., unlinked to other loci under selection, a locally beneficial mutation can be maintained only if its selective advantage exceeds the immigration rate of alternative allelic types. Deterministic modeling shows that, if a beneficial mutation arises in linkage to a locus at which a locally adapted allele is already segregating in migration-selection balance, the new mutant can be maintained under much higher immigration rates than predicted by one-locus theory. However, deterministic theory ignores stochastic effects which are especially important in the early phase during which the mutant is still rare. If the beneficial mutation is linked to a beneficial genetic background, it will profit from a hitch-hiking-like effect. If it occurs on a deleterious background, it is doomed to extinction unless it recombines away. Therefore, recombination plays an ambiguous role. Using the theory of branching processes, we obtain exact numerical and approximate analytical results for the invasion probability as a function of the migration and the recombination rate. We derive predictions for the genetic architecture of traits undergoing local adaptation, such as the degree of genomic clustering and the distribution of fitness effects, and study sojourn times of beneficial mutations. This talk is based on joint work with Simon Aeschbacher.

- Angel Calsina (UAB Barcelona, Spain)

**Title: On steady states of population dynamics models: selection-mutation in an agestructured population**

**Abstract:** Steady states for population models can often be formulated as eigenvector problems for positive operators or generators of positive semigroups plus fixed point problems. As an example we will consider a selection-mutation model for an age and age at maturity structured population, where the latter plays the role of an evolutionary trait on which the vital rates depend. We will discuss existence and uniqueness depending on the dimension of the interaction variable and the possible monotonicity of the spectral radius with respect to this interaction variable.

- Fabio Chalub (Universidade Nova de Lisboa, Portugal)

**Title: Continuous approximations of discrete evolutionary processes**

**Abstract:** Joint work with Max O. Souza (UFF, Brazil). We consider simple discrete evolutionary processes (e.g., the Moran or the Wright-Fisher process) and obtain continuous approximation to the forward and to the backward evolutions. The continuous approximation (in its most general form) consists of a degenerated partial differential equation of drift-diffusion type. The forward equation must be supplemented by a set of conservation laws, while the backward evolution must be supplemented by incomplete information on the boundaries. In both cases, these extra conditions can be obtained directly from the discrete evolutionary process. We will also show how simple expressions for the fixation probability of a given type and the time to fixation of any type, in a population of two types, can be obtained from the continuous approximation and we will compare these expressions with numerical simulations for the discrete evolution.

- Nicolas Champagnat (INRIA Nancy, France)

**Title: Adaptive dynamics in an individual-based, multi-resources chemostat model**

**Abstract:** This is joint work with Pierre-Emmanuel Jabin and Sylvie Méléard. We consider an evolutionary model of population with competition for resources through a chemostat-type model, where individuals consume several common resources that are constantly supplied. This model describes for example the adaptation of bacteria interacting with their environment composed of resources. Bacteria are characterized by a continuous traits describing their consumption ability for each resource. The population of bacteria is assumed to follow a discrete stochastic dynamics, and the dynamics of resources concentrations is governed by deterministic ODEs. We consider fast resource and birth and death dynamics and a slow mutation rate. We prove that the population behaves on the mutation time scale as a jump process describing successive fast mutant invasions between evolutionary equilibria. In the small mutation steps limit, this process converges to an ODE known as the canonical equation, and we are able to characterize the trait values where evolutionary branching, a form of diversification or speciation, may occur.

- Florence Débarre (UBC, Vancouver, Canada)

**Title: Migration-mutation-selection balance in spatially heterogeneous environments.**

**Abstract:** (part of this work has been done in collaboration with Ophélie Ronce and Sylvain Gandon) How do mutation and gene flow influence niche expansion in spatially heterogeneous environments? To address this question, I analyse an evolutionary model of adaptation to an environment containing two habitats in equal frequencies. Using a mixture of adaptive dynamics techniques and moment-based approaches, I derive analytical approximations for the distributions of traits in each habitat under a migration--selection--mutation balance, and contrast the effects of migration and mutation on the evolutionary outcome. In particular, I show that niche expansion---the ability for the population to adapt to both habitats---is ultimately prevented only when migration rates are small and mutations are rare and of small effect. Although the results are derived for clonally reproducing populations, I show the links with quantitative genetics models, and emphasize the limits of classical quantitative genetics assumptions in situations where selection varies spatially.

- Laurent Desvillettes (ENS Cachan, France)

**Title: Asymmetry of the invasion and retreat of neighbour species under the effect of a moving environment**

**Abstract:** We present a work done in collaboration with Henri Berestycki and Odo Diekmann. We are interested in a spatially structured setting in which two species are in competition. The modeling is done through a system of reaction-diffusion PDEs. Initially, each species occupies a specific part of the available space. We assume that the environment changes at a certain speed (for example because of the climate change), in such a way that one of the species will have to perform a retreat, and the other one will be able to invade the corresponding territory. We study (at the mathematical and numerical level) the possible appearance of a widening gap (that is, a growing zone in which very few individuals of the two species are present), due to an asymmetry between the invasion and the retreat.

- Odo Diekmann (University of Utrecht, Netherlands)

**Title: Infectious disease transmission on dynamic sexual networks**

**Abstract:** First we introduce a population model that incorporates demographic turnover as well as, for each individual, a variable number of simultaneous partnerships. The resulting dynamic network next serves as a template for the transmission of an infectious disease. After introducing some general concepts underlying structured population models, in particular the notions of *i*-state (*i* for individual) and *i*-state-at-birth, I shall focus on the way to deduce generation dynamics from real time dynamics and show how to construct a next-

generation matrix from a decomposition of the relevant Jacobi matrix into a transition part and a transmission part. The aim is to characterize  $R_0$  and other severity indicators. The ultimate aim is to shed some light on the impact of concurrency on the spread of HIV. The talk is based on ongoing joint work with KaYin Leung and Mirjam Kretzschmar.

- Régis Ferrière (ENS Paris, France)

#### Opening lecture

- Sylvain Gandon (CEFE Montpellier, France)

#### **Title: The epidemiology and evolution of multi-locus drug resistance**

**Abstract:** The evolution of resistance to drugs is a major public health concern as it erodes the efficacy of our therapeutic arsenal against bacterial, viral and fungal pathogens. What is particularly worrying is the rate of acquisition of multiple drug resistance, which seems to outpace the development of new drugs. Here we develop and analyse a model that tracks both the epidemiology and the evolution of pathogens that can resist drugs at two different loci. This model bridges the gap between classical multilocus population genetics and mathematical epidemiology. It can be used to study the effects of different treatment policies on evolutionary and epidemiological outcomes and, in particular, on the likelihood of reversion to drug sensitive pathogens after the end of therapy. We show that pathogen recombination can either speed up or slow down the spread of multiple drug resistance depending of patterns of epistasis and treatment policies. This analysis also helps specify the type of data we need to acquire to predict the adaptation of infectious diseases. It is also a theoretical framework that can be used to study other evolutionary scenarios and, in particular, the evolution of virulence.

- Pierre-Emmanuel Jabin (University of Maryland, USA)

#### **Title: Small populations effect in selection-mutation models**

**Abstract:** We consider integro-differential models describing the evolution of a population structured by a quantitative trait. Individuals interact competitively, creating a strong selection pressure on the population. On the other hand, mutations are assumed to be small. This creates concentration phenomena, typically consisting in a sum of Dirac masses slowly evolving in time. However classical models do not take the effect of small populations into account, producing some abnormal behaviors: Evolution without mutations, an infinite speed at branching...

- Emily Jones (Washington State University, USA)

#### **Title: Community vulnerability to invasion: the role of evolution before and after species introductions**

**Abstract:** The success of introduced species frequently depends on the outcome of biotic interactions with resident species, which can either resist or facilitate the introduced species. Reciprocally, the consequences for the recipient community depend on the magnitude and direction of biotic interactions with the introduced species. Through a series of evolutionary and eco-evolutionary models, I explore how phenotypic evolution shapes these novel interactions. Specifically, I consider how the evolution affects: (1) the consequences of the introduction of an antagonist into a naïve community, (2) the relationship between an introduced species' phylogenetic relatedness to resident species and its probability of becoming established in the recipient community, and (3) the population growth or extinction of the introduced species during the establishment phase of invasion. These models demonstrate how understanding the evolution of introduced and resident species prior to the introduction, and to a lesser extent immediately after the introduction, can give insight into the progress and consequences of incipient invasions.

- Pierre Magal (Institut de Mathématiques de Bordeaux, France)

**Title: P-gp Transfer and Acquired Multi-drug Resistance in Tumors Cells**

**Abstract:** Multi-Drug resistance for cancer cells has been a serious issue since several decades. In the past, many models have been proposed to describe this problem. These models use a discrete structured for the cancer cell population, and they may include some class of resistant, non resistant, and acquired resistant cells. Recently, this problem has received a more detailed biological description, and it turns out that the resistance to treatments is due in 40% of cancers to a protein called P-glycoprotein (P-gp). Moreover some new biological experiments show that transfers can occur by the mean of Tunneling nanoTubes built in between cells (direct transfers). Transfers can also occur through microparticles (containing P-gp) released by over expressing cells into the liquid surrounding these cells. These microparticles can then diffuse and can be recaptured by the cells (indirect transfers).

- Guillaume Martin (University Montpellier 2, France)

**Title: The distribution of mutation effects on fitness and Random matrix Theory**

**Abstract:** The distribution of the effects of mutations on key demographic/fitness traits (growth rates, competitive index etc.) is a central input of all models of adaptation to new environments, both from standing variance or de novo mutations. Yet there is no widely accepted model to describe this distribution, or how it is affected by context (evolutionary history, harshness of the environmental challenge etc.), although those effects are well-recognized and documented empirically. Various simplified models exist for random or beneficial mutations, but all are often deemed too heuristic to be quantitatively predictive. In this talk I will try to convince you otherwise: that one such type of models, Fisher's geometrical model (which is known to account for several observed empirical patterns), may bear some striking realism in spite of its simplicity.

This model emerges from a much more complex model, as a result of the high degree of phenotypic integration from random genetic changes (of many possible types) into an effect on fitness. As this integration involves a potentially very complex network of interaction, some large number arguments can be used (central limit theorem and random matrix theory) to simplify a model with initially infinitely many parameters into a much simpler Fisher type of model.

Finally I will show a few examples of how this model can be used to predict rates of adaptation to new environments or the cost of beneficial mutations a function of genomic or environmental context.

- Sylvie Méléard (Ecole Polytechnique, Palaiseau, France)

**Title: Stochastic modeling of phylogenies driven by a trait under selection**

**Abstract:** This talk presents a work in progress with Sylvain Billard, Régis Ferrière and Chi Viet Tran. Our aim is to model the effect of natural selection on the genealogy process obtained from the dynamics of the neutral markers. From an individual-based stochastic model, where individuals are characterized by heritable phenotypic trait and neutral marker (and submitted to mutation), we derive some jumping Fleming-Viot process approximating the neutral marker dynamics. The drift and variance coefficients are driven by a trait under selection and jump as the support of the trait, in the limit of the adaptive dynamics. The tools are based on previous works on trait substitution sequences and a fine study of slow and fast dynamics related to the different ecological and mutation (trait and marker) time scales. This work could be the basis for sophisticated models allowing to better understand the reconstruction of phylogenies from molecular data.

- Hans Metz (University of Leiden, Netherlands)

**Title: The canonical equation of adaptive dynamics: a new role for the effective population sizes of population genetics**

**Abstract:** Deterministic population dynamical models connect to reality through their interpretation as limits for system size going to infinity of stochastic processes in which individuals are represented as discrete entities. In structured population models individuals may be born in different  $h$ (eterogeneity)-states, spanned by e.g. their  $i$ (dividual)-state and location in space, after which they proceed through their  $h$ -state space.

Given such a population model one can graft onto it (i) an adaptive dynamics, i.e. an adaptive walk in a space of heritable traits affecting the state transition and reproduction processes of the individuals, or (ii) a random genetic drift. The former is derived by assuming large populations sizes and the rare occurrence of mutations affecting said traits. From this general perspective I will derive the so-called Canonical Equation of adaptive dynamics, a differential equation for evolutionary trait change derived under the additional assumption that mutations have small effect. In the CE approximation the rate of evolution is found to correspond to the product of a parameter  $n_{\{e,A\}}$ , equal to the population size times a dimensionless product of life history parameters (including spatial movements), the probability of a mutation per birth event, the covariance matrix of the mutational steps, and the gradient of the invasion fitness of potential mutants with respect to their trait vector. I will end by heuristically deriving the pleasing and useful result that  $n_{\{e,A\}} = n_{\{e,D\}}$ , the effective population size from the theory of random genetic drift. This follows by connecting the mutant invasion probabilities calculated from a branching process approximation at small mutant numbers with a diffusion approximation for the mutant frequency.

- Sepideh Mirrahimi (Institut Mathématique de Toulouse, France)

**Title: Migration and adaptation of a structured population**

**Abstract:** We present an approach based on Hamilton-Jacobi equations to study models structured by a phenotypical trait and a space variable. This approach has been well-developed previously for population models structured only by a phenotypical trait and provides a framework that allows us to describe the dynamics of the dominant traits. In this talk we present some first attempts to generalize these results to spatial models.

- Grégoire Nadin (Université Pierre et Marie Curie, France)

**Title: On the influence of heterogeneity on the propagation speed for random stationary Fisher-KPP equations**

**Abstract:** We will discuss in this talk the dependence of the propagation speed with respect to the random stationary ergodic coefficients of a reaction-diffusion of Fisher-KPP type, characterizing the heterogeneity of the coefficients through various notions. Such equations are a common model in population dynamics and thus this question is related to conservation and invasion issues. Many dependence results have been investigated in the last decade in periodic media, but the random stationary ergodic framework is much less known due to the absence of classical eigenvalues in such frameworks. The speed could indeed be characterized through an appropriate family of generalized principal eigenvalues (Berestycki-N. 12), reducing this dependence to an optimization problem for generalized eigenvalues. This new characterization thus opens up new perspectives on this issue.

- Ben Phillips (James Cook University, Townsville, Australia)

**Title: The evolutionary ecology of range shift**

**Abstract:** The spread of a population through space is driven by the dispersal and reproduction of individuals in that population. Classical models of spread largely ignore individual variation. This is a problem, because the process of range expansion has three unavoidable consequences that mean that individual variation matters. First, individuals on the expanding edge of the population are sorted by dispersal ability, leading to the accumulation of highly dispersive individuals on the expanding range edge. Second, individuals on the expanding range edge experience low conspecific density relative to individuals in the range core leading to strong natural selection on reproductive rates. Finally, individuals on the expanding range edge are always a small sample of the population that constituted an earlier range edge, so serial founder events complicate matters. In this talk I will demonstrate how these unavoidable consequences appear to have driven rapid shifts in phenotypes, and led to altered spread dynamics. I will focus primarily on empirical examples.

- Ophélie Ronce (University Montpellier 2, France)

**Title: Evolution of range expansion in age-structured populations**

**Abstract:** Using a quantitative genetics model, we study the joint dynamics of adaptation and colonization in a new environment. We in particular consider how such dynamics vary depending on the age at which adaptive traits are expressed, and how adaptation affects the evolution of aging. We consider the consequences of transient disequilibrium in age-structure as well as the effect of continued gene flow from the ancestral environment. Adaptation is generally slower for late-expressed traits, which results in different patterns of aging in newly colonized populations as a function of dispersal and time since introduction.

- Lionel Roques (INRA Avignon, France)

**Title: The dynamics of the genetic structure of range-expanding populations**

**Abstract:** Scalar reaction-dispersion equations, such as reaction-diffusion or integro-differential equations, are widely used for the description of the spatio-temporal dynamics of a population. It is often useful to be able to distinguish between different subclasses in the population. These subclasses can correspond, for instance, to genetic fractions. A usual way to introduce diversity in these models, avoiding the use of complex systems with several equations, is to introduce a variable corresponding to the position in the set of subclasses, e.g. a quantitative phenotypic trait. Here, we propose a totally different approach, which aims at being easily adaptable to a large class of models, from theoretical and computational viewpoints. The idea is to decompose the solution of the global model which describes the dynamics of the particles without subclass distinction, as the sum of subclasses. As an application, we describe the dynamics of the genetic structure in traveling waves of colonization.

- Amandine Véber (CMAP, Ecole Polytechnique, France)

**Title: The effects of a weak selection pressure in a spatially structured population**

**Abstract:** One of the motivations for the introduction of the Fisher-KPP equation was to model the wave of advance of a favourable (genetic) type in a population spread over some continuous space. This model relies on the fact that reproductions occur very locally in space, so that if we assume that individuals can be of two types only, the drift term modelling the competition between the types is of the form  $s p_{t,x}(1-p_{t,x})$ . Here,  $s$  is the strength of the selection pressure and  $p_{t,x}$  is the frequency of the favoured type at location  $x$  and time  $t$ . However, large-scale extinction-recolonisation events may happen at some nonnegligible frequency, potentially disturbing the wave of advance. In this talk, we shall address and compare the effect of weak selection in the presence or absence of occasional large-scale events, based on a model of evolution in a spatial continuum called the spatial Lambda-Fleming-Viot process. This is a joint work with Alison Etheridge and Feng Yu.

- David Waxman (Fudan University, Shanghai, China)

**Title: Revisiting Random Genetic Drift**

**Abstract:** Random genetic drift is a key process in population genetics and evolution. The single most important approach to random genetic drift is the diffusion approximation. In this talk I introduce random genetic drift and the diffusion approximation and present solutions that lead to the standard results, but in a way that is theoretically preferable and in greater accord with intuition. The methods I use lead very naturally to generalisations of existing results, for the probability a gene becomes fixed in a population. I shall briefly discuss applications including the effect of changing population size and changing selection on fixation.

# Abstracts of the posters

x Helen Alexander (ETH Zurich, Switzerland)

**Title : "Evolutionary rescue of a viral population via drug resistance"**

**Abstract :** "Evolutionary rescue" is a term coined in the context of conservation biology, to describe the situation in which genetic adaptation prevents extinction of a population in a novel or changing environment. The emergence of drug resistance in pathogens has been recognized as an example of evolutionary rescue, but research in conservation and medical applications has been largely disconnected. The relationships between mathematical models developed on each side have thus hardly been explored. Here, we briefly compare theoretical approaches to scenarios in which extinction or rescue may occur. We go into a more detailed description of a model of viral dynamics within a host undergoing drug treatment (Alexander & Bonhoeffer 2012, *Epidemics* 4:187-202). The probability of emergence of resistance (i.e. rescue), from either standing genetic variation (existing before treatment begins) or de novo mutation (occurring after treatment begins), is derived. We compare these results to those obtained under a population genetics model more closely connected to the conservation literature (Martin et al. 2012, *Phil. Trans. R. Soc. B* 368:20120088). The key results turn out to fit within a common mathematical formulation, highlighting the similarities between models derived with different applications in mind. We suggest that this case is indicative of the potential for fruitful exchange between conservation biological and medical communities tackling similar, challenging theoretical problems.

x Juliette Bouhours (University Pierre and Marie Curie, France)

**Title : "Front blocking and propagation in cylinders with varying cross section"**

**Abstract :** We consider a bistable reaction diffusion equation in an infinite cylinder with varying cross section and we investigate the existence of propagation phenomena (possibly partial propagation) or on the contrary of blocking phenomena depending on the geometry of the domain. These problems are of interest in different fields of study such as population dynamics and medicine, to model for instance the existence of Cortical Spreading Depression in the brain.

We consider here three different cases, one when the diameter of the cross section decreases, another when the diameter increases and lastly the case of a progressive change in the geometry and prove in each case the existence of a propagation or blocking phenomenon.

x Emeric Bouin (Ecole Normale Supérieure de Lyon, France)

**Title : "A kinetic Fisher-KPP equation : traveling waves and front acceleration"**

**Abstract :** We analyze a kinetic model which describes the same situation as the Fisher-KPP equation. Thus, this model describes particles moving according to a velocity-jump process, and proliferating thanks to a kinetic reaction term of monostable type. We study existence and stability of traveling wave solutions. It turns out that we exhibit a critical speed for this existence, when the velocity space is bounded. Moreover, we recover the standard Fisher-KPP speed in the parabolic limit. The constructed fronts of minimal speed are linearly stable in suitable weighted  $L^2$  spaces. We also investigate the case of an unbounded velocity space and we are able to conclude that not only traveling waves can not exist, but also the equation induces an accelerated propagation behavior according to a scaling law, which depends on the stationary Maxwellian.

x Ramsès Djidjou Demasse (University of Yaounde 1)

**Title : "An age-structured within-host model for multi-strain malaria infections"**

**Abstract :** In this paper we propose an age-structured malaria within-host model taking into account multi-strains interaction. We provide a global analysis of the model depending upon some threshold  $T_0$ . When  $T_0 \leq 1$ , then the disease free equilibrium is globally asymptotically stable and the parasites are cleared. On the contrary if  $T_0 > 1$ , the model exhibits the competition exclusion principle. Roughly speaking, only the strongest strain, according to a suitable order, survives while the other strains go to extinct. Under some additional parameter conditions we prove that the endemic equilibrium corresponding to the strongest strain is globally asymptotically stable.

x Caetano Souto-Maior(1), Erida Gjini(1), Joao S. Lopes(1),  
Delphine Pessoa(1), Claudio J. Struchiner (2), Luis Teixeira(1),  
M. Gabriela M. Gomes (1,2)

(1)Instituto Gulbenkian de Ciência, Oeiras, Portugal

(2)Programa de Computação Científica, Fundação Oswaldo Cruz, Rio de Janeiro, Brazil.

**Title : "Consequences of heterogeneity for Wolbachia invasion"**

**Abstract :** The maternally transmitted bacterial symbiont Wolbachia is known to modulate many life- history traits of its hosts, such as lifespan, fecundity, cytoplasmic incompatibility (CI) and resistance to pathogens. Wolbachia invasion of a resident population without Wolbachia requires a threshold initial frequency, determined by fitness costs and the advantages of CI. When Wolbachia-mediated effects include resistance to pathogens, this threshold is reduced, making invasion easier. In this study we model invasion by Wolbachia when symbiont- mediated effects are heterogeneously distributed. We link invasion thresholds derived from deterministic models with stochastic simulations of invasion. We explore systematically consequences of heterogeneity for the threshold of invasion, its speed and the equilibrium population size.

x Emmanuel Hanert (Earth and Life Institute, Université  
catholique de Louvain, Belgium)

**Title : "Front dynamics in a two-species competition model driven by Lévy flights"**

**Abstract :** A number of recent studies suggest that many biological species follow a Lévy random walk in their search for food. Such a strategy has been shown to be more efficient than classical Brownian motion when resources are scarce. However, current diffusion-reaction models used to describe many ecological systems do not account for the superdiffusive spread of populations due to Lévy flights. We have developed a model to simulate the spatial spread of two species competing for the same resources and driven by Lévy flights. The model is based on the Lotka-Volterra equations and has been obtained by replacing the second-order diffusion operator by a fractional-order one. Theoretical developments and numerical simulations show that fractional-order diffusion leads to an exponential acceleration of the population fronts and a power-law decay of the fronts' leading tail. Depending on the skewness of the fractional derivative, we derive catch-up conditions for different types of fronts. Our results indicate that second-order diffusion-reaction models are not well-suited to simulate the spatial spread of biological species that follow a Lévy random walk as they are inclined to underestimate the speed at which these species propagate.

References:

- Hanert E. (2012) Front dynamics in a two-species competition model driven by Lévy flights, Journal of Theoretical Biology, 300, 134-142.
- Hanert E., E. Schumacher and E. Deleersnijder (2011) Front dynamics in fractional- order epidemic models. Journal of Theoretical Biology 279, 9-16.

x Matthew Hartfield and Samuel Alizon (Laboratoire MIVEGEC, CNRS, IRD, Montpellier, France)

**Title : "Quantifying outbreak thresholds, and effect of susceptible depletion on pathogen emergence"**

**Abstract :** The evolutionary emergence and invasion of new pathogens causes a considerable health risk to human populations, and is a major focus of current research. We first outline criteria to determine how many infected individuals are needed to guarantee emergence in a fully susceptible population (what we call the outbreak threshold). We find that, if the pathogen has a reproductive ratio  $R_0$ , on the order of  $1/\text{Log}(R_0)$  infected individuals are needed to prevent stochastic fade-out. Secondly, we investigate the case where an initial strain spreading, with  $R_0$  close to one, can mutate into a faster-spreading strain. We show how to formulate results for the probability of emergence of the mutated strain, which take into account the ongoing depletion of the susceptible population by the first strain. Even if the first strain's  $R_0$  is close to one, ongoing susceptible depletion has a drastic effect on subsequent pathogen emergence. We highlight this fact by applying our model to the re-emergence of Chikungunya virus on La Réunion island.

x Thomas Stiehl (Interdisciplinary Center for Scientific Computing, University of Heidelberg, Germany)

**Title : "Clonal evolution in the hematopoietic system"**

joint work with Anthony D. Ho (Medical Clinic V, University of Heidelberg) and Anna Marciniak-Czochra (Interdisciplinary Center for Scientific Computing and Bioquant Center, University of Heidelberg)

**Abstract :** The hematopoietic (blood forming) system is maintained by a small population of hematopoietic stem cells (HSCs) giving rise to all types of mature blood cells. Over the last decade evidence has accumulated that the HSC compartment is heterogeneous and that its composition might change over time. Similar phenomena hold for some clonal hematological malignancies.

Based on models of blood formation and leukemias we propose a cell population model of the hematopoietic system that is structured by crucial cell traits and regulated by nonlinear negative feedback. This model allows to investigate the impact of cellular properties such as proliferation and differentiation behavior on dynamics of clonal evolution and selection.

Since the model is built based on biologically motivated parameters, modeling results can be easily interpreted in the biomedical context.

x Yury Zablotski (Helmholtz Centre for Ocean Research, Kiel, Germany)

**Title : "Sympatric speciation by optimal specialisation".**

**Abstract :** Sympatric speciation is one of the most controversial and intriguing concepts in evolutionary biology, because in contrast to allopatric speciation it happens without geographic isolation (Via:2001, Bolnick:2007, Bird:2012). Sympatric speciation can reduce resource competition (Bolnick:2007) via niche partitioning. It stimulates disruptive selection and assortative mating, leading to reproductive isolation, e.g., by divergent mate timing or habitat choice (Bolnick:2007). However, a concrete mechanism which allows sympatric speciation has been difficult to pin down. Here we show that specialisation can lead to sympatric speciation, provided that sufficient ability is retained to eat the less-preferred prey. We define a specialisation trade-off between the improved ability to eat the preferred prey (gain) and reduced ability to eat the less-

preferred prey (cost). We find that the degree of specialisation, and hence interaction strength (Emmerson:2004), strongly depends on specialisation cost and that high costs can prevent speciation. Low and intermediate specialisation costs show disruptive selection and create two niches via resource partitioning. Our model can generate a wide range of specialisation strengths, from generalists to specialists, and associated interaction strengths (Emmerson:2004).

x Paweł Zwoleński and Radosław Wieczorek (Institute of Mathematics, Polish Academy of Sciences and University of Silesia, Poland)

**Title : "Phenotypic evolution of hermaphrodites"**

**Abstract :** We consider an individual based model of phenotypic evolution in hermaphroditic populations which includes mating process of individuals with or without self-fertilization phenomena. By increasing the number of individuals to infinity we obtain a non-linear transport equation, which describes the evolution of distribution densities of phenotype. Existence of one-dimensional attractor is proved and the formula for the density of phenotype in the limiting (asymptotic) population is derived in some particular case.

# Participants

## A

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Zwolenski	Pawel	Institute of Mathematics, Polish Academy of Sciences



# Map of the neighbourhood

**Parc de la Tête d'Or**

**Conference**

