1.

Frank den Hollander

Spatial populations with seed-bank

In this lecture we consider a system of interacting Fisher-Wright diffusions with seed-bank. Individuals carry one of two types, live in colonies labelled by countable Abelian group playing the role of geographic space, and are subject to resampling and migration as long as they are active. Each colony has a seed-bank into which individuals can retreat to become dormant, suspending their resampling and migration until they become active again. Our goal is to understand in what way the seed-bank enhances genetic diversity. When individuals become dormant they adopt a random colour that determines their wake-up time. The system of continuum stochastic differential equations describing the population in the large-colony-size limit has a unique strong solution that converges to an equilibrium parametrised by the initial type densities. This equilibrium exhibits a dichotomy between two phases: coexistence(= locally multi-type equilibrium) versus clustering(= locally mono-type equilibrium). We identify the parameter regimes for which these two phases occur. We also establish the finite-systems scheme, i.e., identify how a finite truncation of the system (both in the geographic space and in the seed-bank) behaves as both the time and the truncation level tend to infinity, properly tuned together.

2.

Martin Möhle

The rate of convergence of the block counting process of exchangeable coalescents with dust

For exchangeable coalescents with dust we provide the rate of convergence of the scaled block counting process to the frequency of singleton process as the sample size tends to infinity. This rate is expressed in terms of a certain Bernstein function. Similar results are discussed for the Siegmund dual fixation line of the block counting process of exchangeable coalescents with dust. The rate of convergence is computed explicitly for beta coalescents with dust, for the Dirichlet coalescent, the Poisson-Dirichlet coalescent and a class of symmetric Xi-coalescents.

3.

Alethea Barbaro

An interacting particle model for territorial dynamics

Abstract: Many species demonstrate territoriality, with individuals or groups marking their territories either chemically or visually. In this talk, we will present an agent-based lattice model for territorial development driven by such markings. In this model, there are several groups, and agents from each group deposit that group's territorial markings as they move on the lattice. Agents move away from areas with territorial markings which do not belong to their own group. We will show that this model undergoes a phase transition between well-mixed collective dynamics and distinct territory formation as parameters are varied. We then formally derive a system of coupled convection-diffusion equations from this model. This

system exhibits cross-diffusion due to the avoidance of other groups' markings. We use linear stability analysis of the continuum system to pinpoint the critical parameter in the discrete model.

4.

Bruno Cessac

Retinal processing: Insights from mathematical modelling

The retina is the entrance of the visual system. The flux of photons coming from the external world is converted by the retina into spike trains sent via the optic nerve and deciphered by the visual cortex in a very efficient way. Although based on common biophysical principles the dynamics of retinal neurons is quite different from their cortical counterparts, raising interesting problems for modelers. In particular most of the retinal processing is done by neuron that do not spike. Only the last step of this processing, sending the visual information to the brain, requires action potentials. This raises several questions:

(1) Retina side. How does the retina structure and dynamics in response to the visual flow (especially, a moving object) shape the non stationary statistics of spike trains emitted to the brain ? What is the structure of spatio-temporal spike correlations ? What are the respective contributions of the visual flow, retinal network, non linear dynamics ?

(2) Cortical side. What could be a plausible cortical model to understand how the retinal spike trains induced by a moving object are deciphered ? The answer actually depends whether one considers that statistical indicators (like firing rates) are sufficient or if individual spikes and their timing do matter.

In this conference, I will address these questions introducing mathematically tractable models based on neuroscience findings, although, obviously, simplified compared to biology. I will show how dynamical and stochastic aspects can be combined to unravel (incomplete and non rigorous) interesting aspects of retinal coding/decoding of non stationary visual scene with moving objects.

5. Alexander van Werde

Title: *Detection and evaluation of clusters within animal movements*

Abstract: Sequential information is ubiquitous, and often the result of some underlying complex behavioral, biological, or physical process. Yet, in practice, gaining insights from such a process presents a challenging task as an underlying structure may be concealed by the high-dimensional nature of the process. Recent theoretical advancements in clustering techniques for a model called Block Markov Chains suggest that reducing the dimensionality of a process could be possible while retaining its sequential information.

We recently investigated the applicability of these techniques to real-world datasets. Our findings with animal movement data were particularly striking. There, a scatter plot of the data gives rise to a picture which is difficult to interpret. After clustering, a picture can be displayed which provides insight into the global topological structure of the animal dynamics. In this talk, I will describe the mathematical theory regarding clustering in Block Markov Chains and the application to animal movement data. If time permits, I may also describe some of our other examples which include the sequence of codons in DNA, text data, and the stock market.

6.

Roeland Merks, Universiteit Leiden

Hybrid cellular Potts modeling of cell-extracellular matrix interactions driving cell shape, cell migration and collective cell behavior

To form the patterns and behaviors that we observe in multicellular development, cells must carefully coordinate their behavior through biophysical and biochemical cues. Numerical modeling and theory are essential for analyzing the mechanism of such coordinated, collective cell behavior. To do so, single-cell models must be sufficiently detailed so they correctly capture essential aspects of individual cells and do not oversimplify. At the same time, the models must be sufficiently simple and computationally efficient so general principles can be understood and the models can be upscaled to multicellular systems. My team analyzes single cell behavior and multicellular development using a combination of mathematical, computational and experimental approaches. Our central tool is the cellular Potts model (CPM), a widely-used, lattice-based framework for modeling cell behavior. We typically couple the CPM with simulation models of the cellular microenvironment and relevant intracellular dynamics, a technique known as hybrid CPMs. I will present a series of our recent hybrid CPMs for modeling individual cell behavior, and show how these can be used to study the coordinated cell behavior that is seen in biological development. I will first discuss a series of models used to analyze observations such as anomalous cell migration patterns of immune cells, the effect of extracellular matrix stiffness on cell shape, cellular force transduction in fibrous ECMs, and models of anisotropic force generation. I will then discuss how insights from single cell models translate to understanding of multicellular development. In our ongoing work, we are developing strategies for experimental falsification and iterative correction of multicellular models of angiogenesis. Recent versions of our cell-ECM interaction models focus on how our descriptions of focal adhesions, the mechanosensitive 'feet' of cells by which they hold on the extracellular matrix, must be improved to analyze mechanical cell-ECM interactions. Also we invest in computational improvements to advance towards more detailed multicellular models. Altogether, I will present the use of cell-based modeling in analyzing how local cell-microenvironment interactions coordinate cell behavior during multicellular patterning.

7. ONLINE

Sophie Pénisson

Stochastic model of tumor evolution for cancer etiology and risk

We present a mathematical model of tumor evolution that includes all phases in the life of a tissue, from tissue development to cancer occurrence. The effects of a carrying capacity, different types of cell division, and different types of driver mutations are also accounted for. New analytical closed-form expressions are obtained from this mechanistic model, showing that the time to cancer can be approximated by a Weibull distribution, providing a simple probability distribution for the timing of a highly complex evolutionary process. The results ofthe mathematical model are then used to provide key insights into cancer etiology, by assessing the role played by normal endogenous mutational processes.

8.

Loren Coquille

Stochastic individual-based models with power law mutation rate on a general finite trait space...towards evolutionary rescue

We consider a stochastic individual-based model for the evolution of a population, whose space of possible traits is given by the vertices of a finite graph. The dynamics is driven by births, deaths, competition, and mutations along the edges of the graph.

We are interested in the large population limit under a mutation rate given by a negative power of the carrying capacity K of the system. This results in several mutant traits being present at the same time and competing for invading the resident population.

We describe the time evolution of the orders of magnitude of each sub-population on the logK time scale, as K tends to infinity.

Using techniques developed in [Champagnat, Méléard, Tran, 2019], we show that these are piecewise affine continuous functions, whose slopes are given by an algorithm describing the changes in the fitness landscape due to the succession of new resident or emergent types.

I will illustrate the theorem by examples describing surprising phenomena arising from the geometry of the graph and/or the rate of mutations.

If time permits I will finish with an application to the phenomenon of evolutionary rescue.

Based on a work in collaboration with Anna Kraut (University of Minnesota) and Charline Smadi (INRAE Grenoble), and an ongoing work with Raphael Forien (INRAE Avignon) and Charline Smadi.

9.

Nadia Belmabrouk

Title : Neural Multivariate Hawkes Process with Long-Range Interactions.

Abstract :Stochastic models have long been a key in simplifying the complexity of neuronal processing. In this context, we introduce a new findings related to Hawkes process on infinite networks, which generalize the previous results of Delattre et al. (2016) from nearest neighbours framework to long-range setting. Our analysis focuses on two regimes: the subcritical regime, where jump propagation is linear and the supercritical regime, characterized by exponential jump propagation.

10. Chitaranjan Mahapatra

Modeling the stochastic behavior of autonomic nervous system activities in detrusor smooth muscle cells, aiming to understand the mechanisms behind urinary bladder overactivity.

Chitaranjan Mahapatra, CNRS/Paris Saclay Institute of Neuroscience, Saclay, France

Introduction:

The urinary incontinence (UI) is defined as the involuntary loss of urine and associated with the enhanced spontaneous contractions of the detrusor smooth muscle (DSM). The spontaneously evoked action potentials (sAPs) in DSM cells initiate and modulate these contractions. The DSM is strongly innervated, connecting approximately 16000 afferent and efferent axons from ganglion neurons [1]. The aim of this current study is to understand the putative relationship between the fluctuating ion channel conductances and stochastically release of ATP in generating sAPs.

Methods:

The neurotransmitter current was considered as an independent excitatory conductance in the model where $g_{ex}(t)$ and E_{ex} are the one-variable stochastic process conductance and the reversal potential respectively. In addition, D_{ex} and $\lambda_1(t)$ are known as the diffusion coefficients and Gaussian white noise. The point-conductance is incorporated into a single DSM cell model based on single cylindrical compartment [2].

Results:

The elicited AP consists an after depolarization and after hyperpolarization phase. The AP peak amplitude and duration are about 5 mV and 40 ms respectively. Then, the random injection of point process model is conducted to elicit a series of sAPs and depolarization for 5 second. The membrane resting potential is held at ─ 50 mV with a 3 mV of fluctuation. The stochastically depolarization up to 20 mV activates the T-type Ca^{2+} channel first and then the L-type Ca^{2+} channel to generate action potential.

*Conclusions***:**

The T-type Ca^{2+} channel blocker can be used as a new phrmacologicl target for UI. In addition, a extended multidimensional models will aid our understanding of DSM electrical and contractile function, providing windows of insight into the factors that govern excitability and contraction in both normal and unstable bladder, in turn shedding light on such phenomena as bladder overactivity and its underlying mechanisms.

1. Young, John S., En Meng, Tom C. Cunnane, and Keith L. Brain. "Spontaneous purinergic neurotransmission in the mouse urinary bladder." The Journal of physiology 586, no. 23 (2008): 5743-5755.

2. Mahapatra, Chitaranjan, Keith L. Brain, and Rohit Manchanda. "A biophysically constrained computational model of the action potential of mouse urinary bladder smooth muscle." PloS one 13, no. 7 (2018): e0200712.

11.

Jochen Blath

Probabilistic structures emerging from dormancy

Dormancy is a complex trait that has independently evolved many times across the tree oflife. In particular, many micro-organisms can enter a reversible state of vanishing metabolic activity. The corresponding dormancy periods can range from a few hours to potentially thousands of years. Also the dormancy transitioning mechanisms are highly diverse, including spontaneous dormancy initiation and resuscitation, responsive switching due to environmental cues, and competition-induced dormancy initiation. In general, dormancy allows a population to maintain a reservoir of genotypic and phenotypic diversity (that is, a seed bank) that can contribute to its longterm survival and coexistence. In this talk, we review recent progress and future perspectives for research on stochastic individual based models incorporating dormancy in several frameworks

12.

Hans Metz

Effective population sizes and the canonical equation of adaptive dynamics

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 rare mutations affecting said traits, and asking oneself how the trait values evolve over evolutionary time. The latter by attaching to each individual one of two neutral labels and asking oneself how for largish population sizes the probability distribution of the relative frequencies of these labels develops over population dynamical time. From this general perspective I will consider 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 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 frequency of mutants with small fitness. (Residents, have fitness zero by the construction of invasion

fitnesses from population dynamical considerations.)

(For a heuristic derivation of the CE for general Mendelian populations see [Metz, J.A.J. & de Kovel, C.G.F. (2013) The canonical equation of adaptive dynamics for Mendelian diploids and haplo-diploids. Interface Focus 3: 20130025]; the results about the effective population sizes, developed together with Vincent Jansen, are still unpublished.)

13.

Christophe Pouzat

What to do with extracellular recording? A proposal

Neurophysiologists are nowadays able to record from a large number of extracellular electrodes and to extract, from the raw data, the sequences of action potentials generated by many neurons. Unfortunately these "many neurons" still represent only a tiny fraction of the neuronal population which constitutes the network. Using association statistics such as the estimation of the cross-correlation functions, they try and infer the structure of the network formed by the recorded neurons. But this inference is compromised by the tremendous under-sampling of the neuronal population and by the errors made during the sequences reconstruction. This yields a "network picture" usually called a "functional network" whose features depend strongly on the recording conditions (such as the presence/absence of a stimulation).
We consider that reconstructing the network formed by the recorded neurons is an ill-posed problem. We propose to focus instead on the "generative probability distribution" of the network: what is the probability to have a connection from a type A neuron to a type B neuron? Is the probability to have a connection from neuron Y of type B to neuron X of type A dependent on the presence of a connection from X to Y? We propose to simulate first the whole network using a simplified neuronal dynamics and different (parametrized) generative probability distributions. We will then compare the association statistics between the simulated and the experimentally observed cases. This type of approach is now commonly used in several fields under different names like "Approximate Bayesian Computation" or "Simulation based Inference". We will then be able to asses if there is an "over representation of reciprocal connections".