Jochen Blath
Probabilistic structures emerging from dormancy

Dormancy is a complex trait that has independently evolved many times across the tree of life. 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 long-term 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

Raphael Cerf
The quasispecies equations

We introduce the quasispecies equations with the help of a very simple model of evolution. These equations turn out to be relevant in the study of classical population models. We explain the error threshold phenomenon and we describe the structure of a quasispecies.

Bruno Cessac
Etinal 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.

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.

Eva Löcherbach
Mean field limits and metastability for probabilistic systems of interacting spiking neurons

I will use the first part of the talk to introduce and discuss a probabilistic model for large systems of interacting neurons represented by their spike trains, that is, the point process of successive spiking times. The stochastic intensity of each of these point processes is a deterministic function of the membrane potential of the corresponding neuron. The membrane potential of a given neuron integrates the inputs of the presynaptic neurons since the last spiking time, up to some leakage effect, leading to a
system of interacting piecewise deterministic Markov processes.

In the second part of the talk I will discuss recent results on large population limits for such processes and then show how the structure of the associated limit process can help us understanding phenomena like metastability. In particular I will discuss how the sojourn time of the system close to the metastable state can be interpreted as an expression of ``short term memory''.

Hanz 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.)

Martin Moehle

On some scaling limits for branching processes and regular exchangeable coalescents

The first part of the talk provides scaling limits for continuous-time branching processes with discrete state space as the initial state tends to infinity. Depending on the finiteness or non-finiteness of the mean and/or variance of the offspring distribution, the limits are in general time-inhomogeneous Gaussian processes, time-inhomogeneous generalized Ornstein-Uhlenbeck type processes or continuous-state branching processes. We also provide transfer results showing how specific asymptotic relations for the probability generating function of the offspring distribution carry over to those of the one-dimensional distributions of the branching process.

In the second part of the talk we provide scaling limits for the (properly transformed) block counting process of coalescent processes with multiple collisions (Λ-coalescents) restricted to a sample of size n as the initial state n tends to infinity under the assumption that the characterizing measure Λ satisfies $\Lambda([0,x])/x\to\kappa$ as $x\to 0$ for some constant κ. The arising limiting processes

belong to the class of Ornstein-Uhlenbeck type processes. Via Siegmund duality a similar convergence result is obtained for the fixation line of the coalescent. The results are extended to a class of regular exchangeable coalescents allowing for simultaneous multiple collisions (Ξ-coalescents). Typical examples are provided.

The talk is based on joint works with Benedict Vetter.

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 of the mathematical model are then used to provide key insights into cancer etiology, by assessing the role played by normal endogenous mutational processes.

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".

Patricia Reynaud-Bouret

Neural Coding as a Statistical Testing Problem

We take the testing perspective to understand what the minimal discrimination time between two stimuli is for different types of rate coding neurons. Our main goal is to describe the testing abilities of two different encoding systems: place cells and grid cells. In particular, we show, through the notion of adaptation, that a fixed place cell system can have a shorter minimum discrimination time when the stimuli are further away. This could be a considerable advantage for the place cell system that could complement the grid cell system, which is able to discriminate stimuli that are much closer than place cells.