

Parallel Session

Evolutionary Dynamics VI

DETERMINISTIC APPROXIMATIONS OF STOCHASTIC DYNAMICS IN EVOLUTIONARY GRAPH THEORY

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Joint work with Mark Broom (City, University of London), Christoforos Hadjichrysanthou (Imperial College London) and Kieran J. Sharkey (University of Liverpool).

Keywords: Evolutionary graph theory, Networks, Markov process, Population structure, Moment closure approximations.

Evolutionary graph theory aims to investigate the dynamics of evolution in a context where there is spatial population structure which is represented by a graph. Previous studies in this area have mainly focused on the outcome of evolutionary processes on highly idealised graphs. On such structures, quantities of interest, such as the fixation probability of an invading mutant, can be calculated analytically.

Investigations of the evolution of populations represented by complex heterogeneous graphs require computationally intensive methods. In this presentation we discuss methods of obtaining approximate descriptions of the underlying stochastic dynamics within a deterministic framework. In particular, we derive approximations directly from the master equation. Although these approximations are commonly used in the modelling of epidemics on networks, their application in evolutionary dynamics is particularly challenging. The purpose of this work is to develop deterministic approximation methods with low computational complexity to describe stochastic evolutionary dynamics on arbitrary graphs.

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THE EFFECT OF GRAPH STRUCTURE ON FIXATION PROBABILITY AND TIME

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Keywords: Moran process, Evolutionary graph theory, Fixation probability, Amplifier of selection, Spatial population structure.

In evolutionary graph theory, a graph can represent a population structure by placing individuals/cells on the nodes of the graph and letting them interact or replace each other via the links. We are interested in the probability that a mutant placed randomly on the graph takes over the whole population of wild-type individuals. Compared to a well-mixed population, a graph is called an amplifier of selection, if it increases the fixation probability of advantageous mutants and decreases it for disadvantageous mutants. The reverse is called a suppressor of selection. We have shown in earlier work that most small undirected graphs are amplifiers of selection for Birth-death updating [1]. Beyond the fixation probability, it is also of interest to study the time this process takes until absorbing into the all-mutant state, i.e. the conditional fixation time. Finding the effect of certain graph properties on fixation probability and time is of growing interest. Employing a genetic algorithm we find graphs with either high or low fixation probability and time and study their structure. Our work unravels certain structural properties that maximise or minimise fixation probability and time. In this way we pinpoint classes of graphs that make up interesting sets within the probability-time-plane.

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REPRODUCTION COSTS CAN DRIVE THE EVOLUTION OF GROUPS

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Keywords: Life cycles evolution, Reproduction cost, Staying-together groups.

A fascinating wealth of life cycles can be observed in biology, from unicellular ones to the concerted fragmentation of multi-cellular units. However, we still have a limited understanding of the factors driving the evolution of life cycles. How does the reproduction cost influence the evolution of life cycles? We consider a basic model of a group structured population of undifferentiated cells, in which larger groups reproduce by fragmentation into smaller groups. Each fragmentation event in our model is associated with a cost expressed by either a fragmentation delay, a fragmentation risk, or a fragmentation loss. The introduction of fragmentation costs vastly increases the set of life cycles which can be optimal. Based on these findings, we suggest that the evolution of life cycles and the splitting into multiple offspring can be directly associated with the fragmentation cost. Moreover, the impact of the fragmentation cost alone is strong enough to drive the emergence of multi-cellular groups, even under scenarios that strongly disfavour groups compared to solitary individuals.

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**A SPATIALLY EXPLICIT INDIVIDUAL BASED MODEL
TO STUDY THE MOTILE-PHYTOPLANKTON
AGGREGATION PROCESS**

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Keywords: Individual-based model, Phytoplankton cellular aggregation, Density-dependent mortality model, Chemosensory ability, Nearest-Neighbor indexes.

The spatial aggregation process is a very important problem which opens to many ecological and biological questions. One of the most important questions is the conducting mechanism that makes the cells aggregate and pass from unicellularity to multicellularity. In this talk, we develop a spatially explicit 3D-individual-based model (IBM) to understand the effect of various mechanisms observed at the scale of phytoplankton cells, on the cellular aggregation process. These mechanisms are: a chemotaxis due to a cell-cell local attraction, a molecular diffusion and a local competition on resources. The IBM is built from the Lagrangian description of a finite system of phytoplankton cells. The cell's movement is modeled through an Itô's diffusion in which the drift term describes the spatial interactions with the surrounding cells and the dispersal term represents the cell diffusion. For the demographical process, we used a density-dependent branching process that takes into account the local competition on the resources through a density-dependent death rate. We implement the IBM using Acceptance-Rejection stochastic algorithm and simulate several scenarios to quantify the effects of the different processes quoted above.

Our simulation study highlights the role of the branching process with a weak-to-medium competition in reinforcing the aggregating structure that forms from attraction mechanisms (under suitable conditions for diffusion and attraction forces), and shows by contrast that aggregations cannot form when competition is high.

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**MODELING A TWO-TYPE, ASYMMETRIC,
TRAIT-DEPENDENT DIVERSIFICATION PROCESS, ON
A RANDOM SPECIES TREE**

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Keywords: Mathematical modeling, Branching processes, Binary traits, Diversity dependence.

One of the main challenges in modern evolutionary biology entails recognizing how different traits evolve on a phylogenetic tree, and how they can affect the evolution of other species characteristics. In this work, we present a probabilistic modeling framework for binary traits (0 and 1), random species trees, in which the number of species and their traits are represented by a two-type, continuous time Markov branching process [1]. Our special focus is on an irreversible model where type-0 units produce offspring of both types, 0 and 1, whereas type-1 units are unable to generate type-0 offspring. In this study, we investigate this model and some of its variations under super-critical assumptions. Models in this category have appeared in various applications, such as mimicking the evolution of tumor progression [2], and branching process models of cancer [3]. The model involves a number of different parameters describing character evolution on the so-called ‘reduced’ tree, consisting of only extant species at the time of observation. We further develop our model by considering the impact of binary traits on dN/dS , the ratio of non-synonymous to synonymous substitutions. Our technique enables us to determine substitutions on a phylogenetic tree with regards to the observed traits. For the asymmetric setting in this work, we also compare our branching model with another branching model that incorporates diversity-dependent diversification [4]. Diversity-dependent diversification describes the effect of competition on speciation and extinction rates. This version of the model is introduced by replacing the branching rates with suitable state-dependent functions. The intention is typically to reduce birth rates and/or increase death rates to balance supercritical net growth. The properties obtained from our theoretical study are finally illustrated with a phylogeny of outcrossing and selfing plant species [5].

References

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EVOLUTION OF TOLERANCE UNDER SELECTIVE PREDATION

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Keywords: Host-parasite systems, Tolerance evolution, Adaptive dynamics.

Whether a host defends itself from parasitism through resistance strategies (e.g. lowered transmission, increased recovery) or tolerance strategies (e.g. reduced mortality during infection) changes drastically the possible evolutionary outcomes of defence evolution. Previous work has unravelled how these two different categories of defence induce different ecological feedbacks to parasite prevalence, showing that while resistance decreases parasite prevalence and reduces its own selective pressure, tolerance strategies boost parasite growth and increase selection for tolerance. Thus, theoretical models traditionally predict fixation as the most likely outcome for tolerance evolution. Here I will present work that identifies further important features of tolerance evolution. Firstly, I will show how the evolution of a costly tolerance mechanism can lead to host-driven parasite extinction, even against parasite counter-adaptation. Secondly, I will examine how community interactions impact tolerance evolution, specifically by including a predator of the host. I will show how the additional ecological feedback induced by the predator allows tolerance branching or non-monotonous trends in the optimal evolutionary strategy. As the interest for tolerance strategies is increasing in different research the aim of this theoretical study is to identify potential new avenues for further research with closer integration between modellers and empiricists.