International Conference

Modelling Biological Evolution 2015:

Linking Mathematical Theories with Empirical Realities

University of Leicester (UK), April 28-May 1, 2015

Sponsored by the London Mathematical Society and the University of Leicester

Organizer: **Andrew Morozov** (University of Leicester, UK)

Aim and Scope

The conference will focus on various aspects of modelling evolution, adaptation and acclimation in different biological systems ranging from macromolecules and cells to processes in sociobiology, including the evolution of human culture and behaviour. Various methods and modelling techniques will be represented including (but not restricted to) general aspects of game theory, adaptive dynamics, optimization, reinforcement learning, model reduction, individual based models as well as their combinations. An important part of the conference will be comparison of the results obtained based on different mathematical techniques to stimulate further advances in modelling biological evolution. The conference is also expected to be an open forum for communication (and probably, hot debates!) between empirical evolutionary biologists and mathematicians with the main goal of enhancing interdisciplinary approaches and stimulating further advances in understanding biological evolution.

Organization & structure

Plenary Speakers:

Samuel Alizon (Montpellier, France)
Nick Britton (University of Bath, UK)
Sergey Gavrilets (University of Tennessee, Knoxville, USA)
Alexander Gorban (University of Leicester, UK)
Ivana Gudelj (University of Exeter, UK)
Rebecca Hoyle (University of Southampton, UK)
Yoh Iwasa (Kyushu University, Japan)
John McNamara (University of Bristol, UK)
Katerina Stankova (Maastricht University, the Netherlands), in memory of Professor Maurice Sabelis
Arne Traulsen (Max-Planck-Institute, Ploen, Germany)

Advisory Scientific Committee:

Alexander Gorban (Leicester, UK)
Ivana Gudelj (University of Exeter, UK)
Vincent Jansen (Royal Holloway University of London, UK)
Kalle Parvinen (Turku, Finland)
Minus van Baalen (Université Pierre et Marie Curie, France)
Andy White (Heriot-Watt University, UK)

Local organizing committee:

Matthew Adamson (University of Leicester, UK)
Scott Balchin, (University of Leicester, UK)
Oksana Gonchar, (University of Leicester, UK)
Masha Jankovic, (University of Leicester, UK)
Yadigar Sekerchi, (University of Leicester, UK)

Detailed Conference Program

Tuesday April 28th

Venue: Bennett Building, ground floor

8.20-9.00 Registration

9.00-10.50 **Introduction and plenary talks 1, 2**

Venue: Bennett Building, LT1

9.00-9.10 **Introduction and welcome address**

9.10-10.00 **Plenary talk 1**. Nick Britton (University of Bath, UK). *Interspecific kleptoparasitism.*

10.00-10.50 **Plenary talk 2**. Rebecca Hoyle (University of Southampton, UK). *Maternal effects, within-generation plasticity and environmental change.*

10.50-11.20 Coffee break: Bennett Building, ground floor

11.20-13.00 **Contributed talks** (session 1)

Venue: Bennett Building, LT1

Chair: Géza Meszéna

11.20-11.40 Nadav Shnerb (Bar-Ilan University, Israel) *Emergence of structured communities through evolutionary dynamics*.

11.40-12.00 Suzanne Sindi (University of California, Merced, USA). *A Mathematical Test for Selection in Word Frequencies*.

12.00-12.20 Anne Kandler (City University London, UK). *Inferring cultural transmission processes from frequency data*.

12.20-12.40 Olof Leimar (Stockholm University, Sweden). *Social evolution and genetic polymorphism.*

12.40-13.00. Gunnar Brandt (Leibniz Center for Tropical Marine Ecology, Germany). *Introducing human behaviour into models of resource extraction*.

13.00-14.20 Lunch break

Venue: Bennett Building, LT1

14.20-15.10 **Plenary talk 3**. Samuel Alizon (CNRS, Montpellier, France). *More than pretty figures: clinical and epidemiological applications of virus phylogenies*

15.15-16.15 **Contributed talks** (session 2)

Venue: Bennett Building, LT1

Chair: Rebecca Hoyle

15.15-15.35 Robert Beardmore (University of Exeter, UK). *Using mathematics to make sense of genomic and phenotypic datasets from rapid antibiotic resistance evolution experiments.*

15.35-15.55. Alexander Bentley (University of Bristol, UK). *Fitness landscapes among many options under social influence.*

15.55-16.15 Sebastien Lion (CNRS, Montpellier, France). *Spatial structure, host heterogeneity and parasite evolution: implications for vaccination.*

16.15-16.45 Coffee break: Bennett Building, ground floor

16.45-18.05 **Contributed talks** (session 3)

Venue: Bennett Building, LT1

Chair: Andrew Morozov

16.45-17.05 Tobias Galla (University of Manchester, UK). *Stochastic evolutionary delay dynamics in epidemiology and gene regulation.*

17.05-17.25 Michael Sieber (University of Potsdam, Germany). *Beyond trade-offs: how life cycle complexity limits parasite host ranges.*

17.25-17.45 Barbara Boldin (University of Primorska, Slovenia). *An extension of the classification of evolutionarily singular strategies in Adaptive Dynamics.*

17.45-18.05 Matthew Adamson (University of Leicester, UK). *Evaluating structural sensitivity of partially specified models in ecology and evolution.*

Time for rest and relaxation

Wednesday April 29th

9.00-10.40 **Plenary talks 3,4**

Venue: Bennett Building, LT2

9.00-9.50 **Plenary talk 4.** John McNamara (University of Bristol, UK). *Ecological rationality and environmental complexity.*

9.50-10.40 **Plenary talk 5.** Sergey Gavrilets (University of Tennessee, Knoxville, USA) *Collective action and the collaborative brain*.

10.40-11.10 Coffee break: Bennett Building, ground floor

11.10-13.10 **Contributed talks** (session 4)

Venue: Bennett Building, LT3

Chair: Arne Traulsen

11.10-11.30 Caroline Colijn (Imperial College London). *Phylogenetic trees and outbreaks of pathogens: mapping one kind of tree onto the other.*

11.30-11.50 Michelle Kendall (Imperial College London, UK). *A new metric for the comparison of phylogenetic trees.*

11.50-12.10 Andrzej Swierniak (Silesian University of Technology, Poland). *Mixed spatial evolutionary games in modelling cancer cell interactions*

12.10-12.30 Peter Ashcroft (University of Manchester, UK). *Stochastic tunnelling and metastable states during the somatic evolution of cancer.*

12.30-12.50 Antje Vollrath (TU Braunschweig, Germany). *A framework for multi-gene-loci inheritance in resistance modeling*

11.10-13.10 Mini-section '**Eco-evolution'**

Venue: Bennett Building, LT2

Chair: Andrew Morozov

11.10-11.30 Daniel Ritterskamp. (ICBM, University Oldenburg). *Evolutionary Dynamics in Food Webs: Influence of Interaction Range, Resource Distribution and Space*

11.30-11.50 Katharina Brinck (Imperial College London, UK). *The evolution of ecosystem organisation: a complexity science approach.*

11.50-12.10 Jaspreet Toor (University of Sheffield, UK). *The evolution of host resistance to disease in the presence of predators*

12.10-12.30 Andrew Dean (University of York, UK). *Modelling the evolution of symbiosis.*

12.30-12.50 Krzysztof Argasinski (Institute of Mathematics, Warsaw, Poland). *Selection under limited population growth. Eco evolutionary feedbacks and the replicator dynamics.*

12.50-14.10 Lunch break

14.10-16.15 **Minisyposium: 'Inferring Cancer Evolution from Genomic Data' (Part I)**

Venue: Bennett Building, LT3

Chairs: Trevor A Graham (Barts Cancer Institute, UK), Andrea Sottoriva (The Institute of Cancer Research, UK) and Ian Tomlinson (University of Oxford, UK)

14.10-14.35 Simon Tavaré (Cancer Research UK Cambridge, UK). *Some thoughts about the statistics of cancer evolution.*

14.35-15.00. Ville Mustonen (Wellcome Trust Sanger Institute, UK). *Using time-resolved genetic data to monitor evolving populations.*

15.00-15.25 Benjamin Werner (The Institute of Cancer Research, UK). *Reconstructing the in vivo dynamics of hematopoietic stem cells from telomere length distributions*

15.25-15.50. Marco Gerlinger (The Institute of Cancer Research, UK) *Copy number trees of cancer evolution.*

15.50-16.15 Robert Noble (University of Montpellier, France). *Eco-evolutionary models of tumour heterogeneity.*

14.10-16.15 **Minisyposium: 'Evolution of virulence in coupled dynamics of infectious diseases'**

Venue: Bennett Building, LT2

Chair: Zhilan Feng (Purdue University, USA)

14.10-14.35 Viggo Andreasen (Roskilde University, Denmark). *Epidemics in competition.*

14.35-15.00. Barbara Boldin (U. Primorska, Slovenia). *Linking within- and between-host dynamics to study the evolutionary dynamics of pathogens.*

15.00-15.25 Zhilan Feng (Purdue University, USA). *Coupled within –host and between-host dynamics and evolution of virulence Part I: Disease dynamics of the coupled system.*

15.25-15.50. Lorenzo Pellis (University of Warwick, UK). *Is HIV short-sighted? Insights from a multistrain nested model*

15.50-16.15 Rupert Mazzucco (IIASA, Austria). *Virulence evolution in fragmented host populations with infectivity–mobility trade-offs.*

16.15-16.45 Coffee break: Bennett Building, ground floor

16.45-18.00 I**nvited short talks**

Venue: Bennett Building, LT2

Chair: Andrew Morozov

16.45-17.15 Géza Meszéna (Eötvös University, Budapest, Hungary). *Niche theory in ecology and evolution: A mathematical exercise, or help in biology?*

17.15-17.45 Vincent Jansen (Royal Holloway University of London, UK). *Inclusive fitness models that include ecological detail: the evolution of investment in siderophore production*

17.45-18.15 Minus van Baalen (CNRS/IHES/ENS, Paris, France). *Adaptation, conflicting information and stress.*

16.45-17.35 **Minisyposium: 'Inferring Cancer Evolution from Genomic Data' (Part II)**

Venue: Bennett Building, LT3

Chairs: Trevor A Graham (Barts Cancer Institute, UK), Andrea Sottoriva (The Institute of Cancer Research, UK) and Ian Tomlinson (University of Oxford, UK)

16.45-17.10. Stephen Attwood (Sichuan University, Chengdu, China). *A practical guide to estimating phylogenies for cancer.*

17.10-17.35. Andrea Sottoriva (The Institute of Cancer Research, UK). *Neutral evolution and star-like phylogenies in next-generation sequencing data.*

18.15-20.00 **Poster session and wine reception**: Bennett Building, ground floor

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Time for rest and relaxation

Thursday April 30th

9.00-9.50 **Plenary talk 6**

Venue: Bennett Building, LT2

9.00-9.50 **Plenary talk 6.** Alexander Gorban (University of Leicester, UK). *Evolution of adaptation mechanisms: adaptation `energy', stress, and oscillating death.*

9.50-10.40 **Plenary talk 7.** Yoh Iwasa (Kyushu University, Japan). *Rate of species creation by geographic isolation and recurrent migration.*

10.40-11.10 Coffee break: Bennett Building, ground floor

11.10-12.50 **Minisyposium: 'Molecular evolution and fitness landscapes. Part I'**

Venue: Bennett Building, LT2

Chairs: Michael Stich (Aston University, Birmingham, UK), Jacobo Aguirre (CNB, Madrid, Spain)

11.10-11.35 Adam Kun (Parmenides Foundation, Pullach,Germany and Eötvös University, Budapest, Hungary). *The minimal genome and functionality of a ribo-organism.*

11.35-12.00. Nobuto Takeuchi (University of Tokyo, Japan). *Spontaneous symmetry breaking in complementary replication as a consequence of multilevel selection in a minimal model of protocells*

12.00-12.25 Tomas Alarcón (CRM, Barcelona, Spain)) *Evolutionary dynamics of systems with genotype-phenotype map*

12.25-12.50 Pablo Catalan (Universidad Carlos III de Madrid, Spain). *toyLIFE: the complexities of the genotype-phenotype map.*

11.10-12.50 **Minisyposium: 'Evolution and Adaptive Dynamics: Applications in Biological Systems'**

Venue: Bennett Building, LT5

Chair: Andy Hoyle (University of Stirling, UK).

11.10-11.35 Andy Hoyle (University of Stirling, UK). *Evolution of antibiotic resistance in aquatic bacteria – biofilms vs well-mixed models.*

11.35-12.00. Alex Best (University of Sheffield, UK). *Co-evolutionary cycles in host-parasite interactions: experiment and theory.*

12.00-12.25 Daniel Balaz (University of Glasgow, UK). Do sheep cheat themselves by mounting weak immune responses? An adaptive dynamics approach.

12.25-12.50 Christina Cobbold (University of Glasgow, UK). *Modelling the evolution of cold tolerance and adaptation to temperature changes: application to the mountain pine beetle.*

12.50-14.10 Lunch break

14.10-15.50 **Plenary talk 8**

Venue: Bennett Building, LT2

14.10-15.00 **Plenary talk 8.** Arne Traulsen (Max-Planck-Institute, Ploen, Germany). *Mathematical models of disease progression: From conceptual insights to quantitative predictions*

15.05-16.05 **Contributed talks** (session 5)

Venue: Bennett Building, LT2

Chair: Minus van Baalen

15.05-15.25 Tamas David-Barrett (University of Oxford, UK). *The evolution of constrained sociality.*

15.25-15.45 Matthijs Van Veelen (University of Amsterdam, the Netherlands). *Inclusive fitness and group selection: the regression method vs. the counterfactual method.*

15.45-16.05 Andrew Pomiankowski (University College London, UK). *The evolution of larger sexual ornaments.*

16.05-16.35 Coffee break: Bennett Building, ground floor

16.35-18.15 **Minisyposium: 'Molecular evolution and fitness landscapes. Part II'**

Venue: Bennett Building, LT2

Chair: Michael Stich (Aston University, Birmingham, UK), Jacobo Aguirre (CNB, Madrid, Spain)

16.35-17.00 Sebastian Ahnert (University of Cambridge, UK) *A tractable genotype-phenotype map for biological self-assembly.*

17.00-17.25 Carlos Lugo (Sainsbury Laboratory, Norwich, UK). *Genomic evolution of pathogens as a consequence of host shifts.*

17.25-17.50 Jose Jiménez (University of Surrey, UK). *Comprehensive experimental fitness landscape and evolutionary network for RNA*

17. 50-18.15 Ester Lazaro (Centro de Astrobiologia, Madrid, Spain). *Transient increases in the error rate can open new pathways for adaptation to new selective pressures.*

16.35-18.15 **Minisyposium: 'Adaptive evolution and the emergence of diversity in ecological communities'**

Venue: Bennett Building, LT5

Chair: Vincent Calcagno (INRA, Sophia Antipolis, France)

16.35-17.00 Vincent Calcagno (INRA, Sophia Antipolis, France). *The interplay of colonization and evolution in models of adaptive radiation*

17.00-17.25 Florence Debarre (Wissenschaftskolleg zu Berlin, Germany). *(Co)evolution in multiple dimensions: how does the number of traits under selection influence evolutionary and co-evolutionary processes?*

17.25-17.50 Fabien Laroche (University of Montpellier, France). *Evolution of dispersal impacts species diversity patterns in a heterogeneous metacommunity*

Contributed talk:

17.50-18.15 Judith Perez-Velazquez (Helmholtz Zentrum München, Germany). *An age-structured model to analyze the evolutionary stability of bacterial quorum sensing.*

Time for rest and relaxation

Friday May 1st

9.00-9.50 **Plenary talk**

Venue: Bennett Building, LT2

9.00-9.50 **Plenary talk 9.** Ivana Gudelj (University of Exeter, UK). *The role of trade-offs in the evolution of diversity.*

10.00-11.20 **Contributed talks** (session 6)

Venue: Bennett Building, LT2

Chairs: Andrew Morozov

10.00-10.20. Galina Kuzenkova (Lobachevsky State University, Nizhni Novgorod, Russia). *Controlled selection process of self-replicating systems.*

10.20-10.40. Oleg Kuzenkov (Lobachevsky State University, Nizhni Novgorod, Russia) *Revealing patterns of optimal zooplankton diel vertical migration on the basis of dynamics of the underlying measure.*

10.40-11.00. Max Souza (Departamento de Matemática Aplicada, UFF, Brazil). *Fixation in large populations: a continuous view of a discrete problem.*

11.00-11.20. Magnus Lindh (Umeå University, Sweden). *Early starters beat optimal reproduction strategy in evolutionary game with annual plants*

10.00-11.20 **Contributed talks** (session 7)

Venue: Bennett Building, LT5

Chairs: Nick Britton

10.00-10.20. Andrew Whalen (University of St Andrews, UK). *The Learning of Sequences of Actions through Low Fidelity Social Transmission*

10.20-10.40. Daniel van der Post (University of St Andrews, UK) *Learning mechanisms modulate the evolutionary trade-off between social learning and exploration*

10.40-11.00. Ke Yuan (University of Cambridge, UK). *Reconstructing intra-tumor phylogenies with Bayesian nonparametric models.*

11.00-11.20 Bhavin S. Khatri (National Institute for Medical Research, UK). *A simple biophysical model of protein binding DNA predicts higher rates of speciation in small populations.*

11.20-11.50 Coffee break: Bennett Building, ground floor

11.50-12.40 **Plenary talk**

Venue: Bennett Building, LT2

11.50-12.40 **Plenary talk 10.** Katerina Stankova (Maastricht University, the Netherlands). *Evolution of diapause timing in an acarine predator-prey system on apple: caused by phylogeny, ecology or both?*

12.40-14.00 Lunch break

14.00-16.00 **Contributed talks** (session 8)

Venue: Bennett Building, LT2

Chair: Max Souza

14.00-14.20 Jack Aidley (University of Leicester, UK). *Modelling the behaviour of hypermutable regions in populations of Campylobacter jejuni under selective and non-selective conditions*

14.20-14.40. Weini Huang (Max Planck Institute, Plön, Germany) *Evolutionary game dynamics under demographic fluctuations*

14.40-15.00. Juan C. Ramírez (University of Sheffield, UK) *Self-deception Can Evolve Under Appropriate Costs*

15.00-15.20 Michael Pocklington (University of Leicester, UK). *The empirical genetic interaction map, the molecular ecosystem, and the nature of mathematical and computational abstraction.*

15.20-15.40 Christopher Quickfall (University of Sheffield, UK) *Evolution of Maternally-Transmitted Symbionts*

14.00-15.40 **Contributed talks** (session 9)

Venue: Bennett Building, LT5

Chair: Géza Meszéna

14.00-14.20 Yoav Soen (Weizmann Institute of Science, Israel). *Bridging Ecology and Evolution by Symbiosis and Epigenesis*

14.20-14.40 Tat Dat Tran (Max Planck Institute, Leipzig, Germany). *The free energy method for the Wright-Fisher model*

14.40-15.00 Axel Rossberg (Cefas, UK) *Are there species smaller than 1mm?*

15.00-15.20 Gereon Kaiping (University of Southampton, UK). *Structured populations facilitate cooperation in policed Public Goods Games.*

15.20-15.40 Virgile Baudrot (Université de Franche-Comté / CNRS, France). The influence of adaptive foraging on trophically transmitted parasite.

15.50-16.00 **Closing address and end of meeting.**

Venue: Bennett Building, LT2

Plenary Talks

(in alphabetic order)

Samuel Alizon, CNRS, Montpellier, France

More than pretty figures: clinical and epidemiological applications of virus phylogenies

Abstract

Virus phylogenies have become commonplace in biomedical articles, especially to identify novel strains. However, the power of phylogenetic inference is currently only used to a fraction of its potential. For instance, several studies have shown that epidemiological parameters such as R_0 or even infection duration can be inferred from sequence data via phylogenies. Furthermore, there are also clinical applications. For instance, in absence of treatment, some individuals infected by IV-1 progress to AIDS rapidly, whereas others remain asymptomatic for more than a decade. Most of the research effort has focused on identifying host factors controlling the virulence of HIV infections but these only explain part of the variance we observe. Using virus phylogenies allows to show that a fraction of the virulence of an infection can be explained by virus factors. Overall, the increased availability of large amounts of clinical data is arguably turning phylogenies into the most important component of evolutionary medicine.

Nicholas F Britton, University of Bath, UK

Interspecific kleptoparasitism

Abstract

Kleptoparasitism is parasitism by theft. In a typical kleptoparasitic host–parasite interaction the parasite steals some resource, such as a food item, from the host. The host and parasite belong to the same species in intraspecific and to different species in interspecific kleptoparasitism. An interaction may be considered as an asymmetric game, with the parasite (or intruder) deciding whether to challenge the host (or owner) for the resource and the host deciding whether to resist the challenge. In the intraspecific but not the interspecific case a single animal may play the role of owner at one time and that of intruder at another. The problem is to determine the evolution of the behaviour of the host and the parasite under the action of natural selection. We review the intraspecific case, and then go on to analyse the interspecific case, considering both the adaptive and the replicator dynamics. We explore the possibility that, on an ecological time scale, the system does not settle to a steady state but to oscillatory behaviour in strategy space. We conclude by considering the features of kleptoparasitism that lead to this possibility, and other host–parasite systems that may behave similarly.

Sergey Gavrilets, University of Tennessee, Knoxville, USA

Collective action and the collaborative brain

Abstract

Humans are unique both in their cognitive abilities and in the extent of cooperation in large groups of unrelated individuals. How our species evolved high intelligence in spite of various costs of having a large brain is perplexing. Equally puzzling is how our ancestors managed to overcome the collective action problem and evolve strong innate preferences for cooperative behaviour. Here, I theoretically study the evolution of social-cognitive competencies as driven by selection emerging from the need to produce public goods in games against nature or in direct competition with other groups. I use collaborative ability in collective actions as a proxy for social-cognitive competencies. My results suggest that collaborative ability is more likely to evolve first by between-group conflicts and then later be utilized and improved in games against nature. If collaborative abilities remain low, the species is predicted to become genetically dimorphic with a small proportion of individuals contributing to public goods and the rest free-riding. Evolution of collaborative ability creates conditions for the subsequent evolution of collaborative communication and cultural learning.

Alexander Gorban, University of Leicester, UK

Evolution of adaptation mechanisms: adaptation `energy', stress, and oscillating death

Abstract

In 1938, H. Selye proposed the notion of adaptation energy and published some "Experimental evidence supporting the conception of adaptation energy". This idea was widely criticized and its use nowadays is rather limited. Nevertheless, the response to many harmful factors has often general non-specific form and we can guess that the mechanisms of physiological adaptation admit a very general and nonspecific description.

We assume that natural selection plays a key role in the evolution of mechanisms of physiological adaptation and apply the optimality models to description of these mechanisms. In the light of the optimality models, the mechanisms of adaptation are represented as the optimal distribution of resources for neutralization of harmful factors. We study dynamics of resources redistribution and revisit the theory of the general adaptation syndrome. Adaptation energy is considered as an internal coordinate on the `dominant path' in the model of adaptation. The phenomenon of `oscillating death' is predicted on the base of the dynamical models of adaptation.

The developed theory is supported by various experimental evidences from ecological physiology and by medical data.

Ivana Gudelj, University of Exeter, UK

The role of trade-offs in the evolution of diversity

Abstract

Organisms cannot excel at all things but are obliged to be jack-of-all trades. This constraint is central to the concept of trade-offs. A bacteria, for example, cannot both grow extremely fast and be resilient to harmful substances; if it invests more into one it must invest less into the other. A seminal theoretical solution first formalized by Levins almost 50 years ago showed that, in theory, the geometry of a trade-off should determine how the species will respond over time to selection. A plethora of subsequent theories would be nullified were it be proven that Levins' seminal postulate were not true. Unfortunately, because of the technical difficulty in doing the necessary tests this theory has gone untested. Using a combination of synthetic ecology and mathematical modeling we provide the first verification, showing that the exact form of the trade-off determines, in a predictable way, the outcome of evolution. However in nature trade-off data is often too noisy to discern shape. To address this problem we discuss how we can infer geometry directly from the biophysical mechanisms that cause trade-offs.

Rebecca Hoyle, University of Southampton, UK

Maternal effects, within-generation plasticity and environmental change

Abstract

Maternal effects are the influences of the maternal phenotype on offspring phenotypes by routes other than direct genetic transmission. Potentially maternal effects provide an additional means of adaptation to changing environmental conditions over and above that afforded by within-generation phenotypic plasticity. I will present insights from a series of quantitative genetics models into the role of maternal effects during environmental change. In particular I will discuss the coevolution of maternal effects and phenotypic plasticity following an abrupt environmental change and during cyclical environmental fluctuations such as seasonal variation, and show that our models suggest finding substantial maternal influences on offspring phenotypes may be more challenging than anticipated.

Yoh Iwasa, Department of Biology, Kyushu University, Japan

Rate of species creation by geographic isolation and recurrent migration.

(joint work with Ryo Yamaguchi[1] and Sergey Gavrilets[2])

Abstract

We study the time to speciation by geographic isolation for a species living on multiple islands connected by rare migration. We assume that the incompatibility is controlled by many loci, and that individuals differing in loci more than a threshold do not mix genetically with each other. For the case of two populations, we can analyze the system by tracing their genetic distance, defined as the number of incompatibility loci differing between the populations. If each population is nearly monomorphic, the genetic distance follows stochastic processes, which can be analyzed by diffusion equation and by stochastic differential equation (SDE). There exists an intermediate optimal rate of migration that maximises the rate of species creation by recurrent invasion and diversification.

We also study the case when the strength of incompatibility gradually increases with the genetic difference. For the case of three or more populations, genetic distances do not form closed stochastic dynamics. To overcome this difficulty, for each locus we define "geographic configuration (GC)" which specifies islands with common alleles, and we trace the stochastic transitions between different geographic configurations by SDEs for the number of loci with different GC. We also discuss how the speciation rate changes with geographical structure.

[1]Department of Biology, Kyushu University, Japan.

[2]Department of Ecology and Evolutionary Biology, University of Tennessee, Knoxville, USA.

John McNamara, Department of Mathematics, University of Bristol, UK

Ecological rationality and environmental complexity

Abstract

Behavioural ecologists have built complex models of optimal behaviour in simple environments. I argue that they need to focus on simple mechanisms that perform well in complex environments. This is, however, a difficult area for a modeller in that various modelling choices are not obvious. In particular, what complexities should a model world include and what sorts of rules should be considered? In my talk I consider an approach that is more limited but which sidesteps these difficulties; I will take rules that have evolved and ask what aspects of the environment make these rules ecological rational. I argue that various psychological phenomena can only be understood in adaptive terms if the environment in which the rules evolved was sufficiently complex. For example, rules may only make sense in adaptive terms if there are specific spatial or temporal heterogeneities. I illustrate this general theme using examples that show that lack of transitivity, contrast effects, aspects of prospect theory, and other behavioural phenomena which are often regarded as irrational can all have adaptive explanations.

Katerina Stankova, Maastricht University, The Netherlands.

in memory of Professor Maurice Sabelis

Evolution of diapause timing in an acarine predator-prey system on apple: caused by phylogeny, ecology or both?

Abstract

Diapause has generally been considered as a response to abiotic factors, such as day/night length and temperature. Strikingly, however, the modes of diapause induction can widely differ between species and higher taxa, even when living in exactly the same (micro-) habitat. This is the case for plant-inhabiting spider mites (Acari: Tetranychidae) and phytoseiid mites, being their most important predators (Acari: Phytoseiidae): spider mites have a distinct and irreversible response to abiotic factors, whereas predatory mites have a much more flexible response. Such differences may have evolved due to phylogenetic constraints, but we argue that this is not necessarily true. Ever since the laboratory studies by Kroon and co-workers it is known that diapause timing of two-spotted spider mites can be modified by the presence of predators. Moreover, food availability is long known to modify diapause timing by predatory and spider mites. Since diapause implies movement away from the leaf and predator-prey interaction concentrates on the leaf, predator and prey may respond to each other by opting in or out of the predator-prey interaction arena. First, we provide field evidence for density-dependent timing of diapause in a system of fruit-tree red spider mites (Panonychus ulmi) and phytoseiid mites (Typhlodromus pyri) on apple trees. Second, using Lotka-Volterra models extended to include energy dynamics we show that game theory applied to a predator-prey system predicts the diapause strategy to be reversible for the predator, yet irreversible for the prey. These predictions do not only match field observations, but also emphasize that the position in the food web matters to what is the best diapause strategy. We therefore claim that species interactions may offer a viable ground for proposing ecological hypotheses that form a (mutually exclusive or non-exclusive) alternative to phylogenetic hypotheses. In the second part of the talk I will introduce results of our current study of of tritrophic system involving cry-wolf plants, herbivorous and carnivorous mites.

Arne Traulsen, Max-Planck-Institute, Ploen, Germany

Mathematical models of disease progression: From conceptual insights to quantitative predictions

Abstract

Many tissues such as the blood system, the colon or the skin, are organized hierarchically. The somatic evolution of cancer can be strongly affected by this population structure. Mutations arising in primitive cells can lead to long lived or even persistent clones, but mutations arising in further differentiated cells are short lived and do not affect the organism. A generic mathematical model for such tissue structures can be used to model the somatic evolution in various cancers, such as certain Leukemias. While such models lead to immediate conceptual insights, it remains a challenge in these models to fix their parameters from experimental data and many models contain parameters that are inaccessible in a biological context. In particular, concrete predictions for disease dynamics within an individual typically require repeated measurements - but this is often unfeasible in a medical context. However, certain biomarkers may have properties that allow us to derive all relevant parameters from a single measurement, leading to dynamical predictions.

Contributed talks, minisymposia and short invited talks

(in alphabetic order)

Matthew Adamson, Department of mathematics, University of Leicester, UK

Evaluating structural sensitivity of partially specified models in ecology and evolution.

(joint work with Andrew Morozov[1])

Abstract

Mathematical models in ecology and evolution are highly simplified representations of a complex underlying reality. For this reason, there is always a high degree of uncertainty with regards to the model specification, not just in terms of parameters, but also in the form taken by the model equations themselves. This uncertainty becomes critical for models in which the use of two different functions fitting the same dataset can yield substantially different model predictions - a property known as structural sensitivity. In this case, the uncertainty in the model functions carries through into uncertainty in our model predictions, and new frameworks are required to deal with this. In this talk, we shall introduce such a framework by considering partially specified models, in which unknown functions are represented not by a specific functional form, but by an entire data range and constraints of biological realism. We shall show how these partially specified models can be used to rigorously detect when ecological models are structurally sensitive in their predictions concerning the character of an equilibrium point, and see that when only varying parameters of fixed model functions, we can often miss this sensitivity completely. We shall then discuss how we can introduce notions of the probability of certain model dynamics in order to quantify the uncertainty in our predictions, and to enable us to conduct bifurcation analysis of the model probabilistically. Finally, we shall discuss how our methods can be extended to investigations in adaptive dynamics by introducing hypothetical data ranges of trade-off functions into the framework of critical function analysis. In this way, we should be able to quantify the uncertainty in the characterisation of an evolutionarily singular strategy, and investigate how this depends on the level of uncertainty in the trade-off functions.

[1]Department of mathematics, University of Leicester, UK

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Jack Aidley, Department of Genetics, University of Leicester, UK

Hypermutability in *Campylobacter jejuni* populations under selective and non-selective bottlenecks

(joint work with Christopher D. Bayliss [1])

Abstract

Campylobacter jejuni is the leading cause of food poisoning in the UK, accounting for around two thirds of all cases of gastro-enteritis. *C. jejuni* is able to produce a large range of phenotypic variations rapidly within its population through highly localised, hypermutable regions of its genome. Changes in these regions produce phase variation: stochastic, high frequency, reversible change in gene expression. In the strain we work with there are 29 of these hypermutable regions, with each typically producing switching between an ON and an OFF state for a particular gene. This leads to upwards of 16 million possible phenotypes.

Our work centres on understanding the role of these changes in gene expression in a variety of biological processes and how patterns of expression change in populations. To this end we have developed a computer simulation of these hypermutable regions under cyclically changing selective conditions and through non-selective bottlenecks. The results from these simulations show that different rates of mutation are favoured by environments that change at different rates in a non-linear manner and that the impact of non-selective bottlenecks on population structure is highly dependent on both the initial population and the size of the bottleneck.

[1]Department of Genetics, University of Leicester, UK

Sebastian Ahnert, University of Cambridge, UK

A tractable genotype-phenotype map for biological self-assembly

(joint work with S. F. Greenbury[1], I. G. Johnston[2] and A. A. Louis[3])

Abstract

The mapping between biological genotypes and phenotypes is central to the study of biological evolution. We introduce a rich, intuitive and biologically realistic genotype - phenotype (GP) map that serves as a model of self-assembling biological structures, such as protein complexes, and remains computationally and analytically tractable. Our GP map arises naturally from the self-assembly of polyomino structures on a two-dimensional lattice and exhibits a number of properties: redundancy (genotypes vastly outnumber phenotypes), phenotype bias (genotypic redundancy varies greatly between phenotypes), genotype component disconnectivity (phenotypes consist of disconnected mutational networks) and shape space covering (most phenotypes can be reached in a small number of mutations). We also show that the mutational robustness of phenotypes scales roughly logarithmically with phenotype redundancy and is positively correlated with phenotypic evolvability. Although our GP map describes the assembly of disconnected objects, it shares many properties with other popular GP maps for connected units, such as models for RNA secondary structure or the hydrophobic-polar (HP) lattice model for protein tertiary structure. The remarkable fact that these important properties similarly emerge from such different models suggests the possibility that universal features underlie a much wider class of biologically realistic GP maps. A further property of the polyomino GP map is that the modularity, symmetry and structural complexity of the phenotype can be quantified rigorously. We can therefore use our GP map to study the emergence of these phenotypic properties in the course of biological evolution, and present results on the distribution of complexity in phenotypes, which are intimately linked with the properties of genotype-phenotype maps.

[1]Theory of Condensed Matter Group, Cavendish Laboratory, University of Cambridge, Cambridge, UK

[2]Department of Mathematics, Imperial College London, London, UK

[3]Rudolf Peierls Centre for Theoretical Physics, University of Oxford, Oxford, UK

Jacobo Aguirre, Centro Nacional de Biotecnología (CSIC), Madrid Spain

Weak environmental changes lead to tipping points in the genomic composition of populations.

(joint work with Susanna Manrubia[1])

Abstract

Nature has been exposed during Earth's evolutionary history to a wide variety of gradual changes in environmental and climate conditions. Recent studies hypothesize that these smooth alterations might be responsible for some of the drastic state shifts that have been reported in our planet's biosphere [1,2]. The question that ultimate faces this new line of research is whether the local impact of humans could provoke a planetary-scale tipping point in the nearby future.

Our work [3] deals with the possibility of observing such drastic changes in the composition of populations at the genotypic level (e.g. RNA populations) that evolves in a changing environment. In our study, a graph represents the space of genotypic sequences of length *N*, where every locus (or nucleotide in the case of RNA) is a state taken from a genetic alphabet of *A* letters: each node stands for a different sequence and two nodes are connected by an undirected link if they differ in the state of only one locus [4]. We use a modification of the widely known *NK* model to map a rugged fitness landscape to such sequences. Finally, we model the evolution of the environment with time via perturbations in the fitness landscape.

Our work shows analytically and numerically that not only gradual and monotonic changes in environmental conditions, but also totally random fluctuations of very small amplitude, generically give rise to critical transitions in the genetic composition of populations for a wide range of evolutionary situations. In summary, our results extend the studies on planetary tipping points to the genotypic scale, where it had not been studied to the date.

[1]Centro Nacional de Biotecnología (CSIC), Madrid Spain

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Tomas Alarcon, Centre de Recerca Matematica, Barcelona, Spain

Evolutionary dynamics of systems with genotype-phenotype map

Abstract

We study the problem of evolutionary escape and survival of cell populations with a genotype-phenotype structure. We refer to evolutionary escape as the process where a cell of a given ill-adapted population to reach a well-adapted phenotype. Similarly, survival refers to the dynamics of the population once the escape phenotype has been reached. The aim of this paper is to analyze the influence of topological properties associated to robustness and evolvability on the probability of escape and on the probability of survival. In order to explore these issues, we formulate a population dynamics model, consisting of a multi-type time-continuous branching process, where types are associated to genotypes and their birth and death probabilities depend on the associated phenotype (non-escape or escape). We exploit the separation of time scales introduced by the difference in reproductive ratios between the ill-adapted phenotypes and the escape phenotype. Two dynamical regimes emerge: a fast-decaying regime associated to the escape process itself, and a slow regime which corresponds to the survival dynamics of the population once the escape phenotype has been reached. We exploit this separation of time scales to analyze the topological factors which determine escape and survival probabilities. We show that, while the escape probability depends on the degree of escape phenotype, the probability of survival is essentially determined by its robustness, measured in terms of a weighted clustering coefficient.

Viggo Andreasen, Dept of Science, Roskilde University, Denmark

Epidemics in competition

Abstract

The competition between two pathogen strains during the course of a single epidemic describes the early adaptation of emerging human diseases. In addition it represents the short time scale in the natural selection of microparasties in seasonally forced systems such as insects and baculovirus.

The mathematical description of the process, known as "epidemics in competition," takes the form of a structurally unstable dynamical system excluding most standard approaches to non-linear differential equations. Alternative methods show that the underlying map has discontinuities associated with sequential epidemics. For systems with partial cross-immunity the sequential epidemics represents an early step towards pathogen speciation.

Krzysztof Argasinski, Institute of Mathematics (PAS), Warsaw, Poland

Selection under limited population growth. Eco evolutionary feedbacks and the replicator dynamics.

(jointed work with Mark Broom[1])

Abstract

In this talk we discuss a new approach to the derivation of population dynamic models called "event based modelling," which relies on the assumption that the trajectory of the process is the aggregated outcome of individual interactions (i.e. "atomic" events) occurring with respective rates. Thus, the methodology resembles that of chemical kinetics where the interaction rate is the analogue of the reaction rate.

An important aspect of the presented framework is the explicit incorporation of growth limitations. The regulation of the population size acts through feedback driven by density dependent juvenile mortality. It was shown that at the population size equilibrium, newborns form a pool of candidates from which survivors who will replace dead adults at their nest sites will be drawn. Thus fertility payoffs can be interpreted as the entries of a nest site lottery mechanism. The new approach emphasizes the role of the turnover of individuals. In this case the stable population size is a dynamic equilibrium between different mortality and fecundity factors instead of an arbitrary fixed carrying capacity. This mechanism can be regarded as an example of eco-evolutionary feedback. This seriously alters the predictions of game-theoretic models in comparison to models with unlimited growth.

In this case there can be for example two stable manifolds: one for the frequency dynamics and a second for the population size. The global stationary points are intersections of those manifolds. For example in the Hawk-Dove Game, a pure Hawk population can become evolutionarily stable in addition to the stable mixed equilibrium known from the classical theory. This is caused by the fact that the payoff structure is not constant. The most intriguing result is that under the impact of eco-evolutionary feedback, an apparently unstable invasion barrier between two pure-strategy stable equilibria can become stable at the intersection with the stable density manifold.

[1]City University London, UK

Peter Ashcroft, School of Physics and Astronomy, University of Manchester, UK

Stochastic tunnelling and metastable states during the somatic

(joint work with Franziska Michor[1] and Tobias Galla[2])

Abstract

We study the accumulation of two mutations in a finite population of cells proliferating according to the Moran process. This corresponds to the inactivation of tumour-suppressor genes in a tissue. Analysing the deterministic dynamics of large populations we systematically identify the parameter regimes captured by existing approaches. Our analysis also reveals fitness landscapes and mutation rates for which finite populations are found in long-lived metastable states. These are landscapes in which the final mutant is not the most advantageous in the sequence, and resulting metastable states are a consequence of a mutation-selection balance. The escape from these states is driven by intrinsic noise, and their location affects the probability of stochastic tunnelling. Existing methods no longer apply. In these regimes, it is the escape from the metastable states that is the key bottleneck; fixation is no longer limited by the emergence of a successful mutant lineage. We used the WKB method to compute escape times in these parameter regimes, successfully validated by stochastic simulations. Our work fills a gap left by previous approaches and provides a more comprehensive description of the acquisition of multiple mutations in populations of somatic cells.

[1]Department of Biostatistics and Computational Biology, Dana-Farber Cancer Institute, and Department of Biostatistics, Harvard School of Public Health, Boston, USA.

[2]Theoretical Physics, School of Physics and Astronomy, University of Manchester, Manchester, UK.

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Daniel Balaz, University of Glasgow, UK

Do sheep cheat themselves by mounting weak immune responses? An adaptive dynamics approach

(joint work with Christina A. Cobbold[2], Michael J. Stear[1], Joaquín Prada Jiménez de Cisneros[1], Rodney Beard[1], and Louise Matthews[1])

Abstract

There is substantial heterogeneity in immune response to parasitic infection. For example, in gastro-intestinal nematode infections of sheep, the parasite-specific IgA response is skewed with many animals producing relatively weak responses. As these responses are also highly heritable, one might naively assume that evolution would optimise immune responses for maximal fitness, so why many sheep produce weak responses is unclear. Traditional explanations implicate trade-offs between immunity and growth or immunity to different diseases but these are not supported by the data, which suggest that the sheep with strong responses and low parasite loads have the highest growth rates.

One alternative explanation is that it may be evolutionarily advantageous for sheep to 'cheat' by mounting reduced immune responses and allow other sheep to control infection. We used an adaptive dynamics approach to explore the benefit of mounting a lower immune response as a function of the strategies of other individuals. Our model, parameterised using data on the sheep - *Teladorsagia circumcinta* system, encompasses dynamics on epidemiological and evolutionary timescales. The epidemiological dynamics determine the infection levels and growth rates of the flock for a given set of immune responses, whilst the evolutionary dynamics allow mutations to the strategies that determine the host densities and evolutionarily stable immune strategies.

Although it has been theoretically shown using adaptive dynamics that evolution may not optimise, this has not to our knowledge been identified and utilised in a real system. We show that across a range of costs of infection burden and immune response, the evolutionary dynamics converge on an equilibrium with suboptimal growth rates. This has important consequences for domesticated sheep flocks in which breeding is managed. Our results suggest that it may be possible to achieve higher growth rates in managed sheep flocks by deliberately selecting for enhanced immune responses. This is an important step forward which offers an alternative theoretical principle for selective breeding to the commonly held view that trade-offs may render selection detrimental.

[1]Institute of Biodiversity, Animal Health and Comparative Medicine, University of Glasgow, UK

[2]School of Mathematics and Statistics, University of Glasgow, UK

Robert Beardmore, University of Exeter, UK

Using mathematics to make sense of genomic and phenotypic datasets from rapid antibiotic resistance evolution experiments.

Abstract

Due to the large population densities supported by microbes, microbial evolution is rapid. Evolution, meaning inherited changes in the genome, can now be tracked in near real-time and its consequences dissected using modern wet lab and computational techniques. This approach to is perhaps best illustrated with Mike Mwangi's study [1] of adaptation in the important, often deadly, human pathogen MRSA in a clinical case study that shows about three nucleotide polymorphisms passing to fixation with each week of antibiotic treatment! His study enumerates both resistance to the drugs used, vancomycin and rifampicin, but also cross-resistance to drugs not used in the treatment of the patient in question. This is a troubling finding.

However, while omics techniques continue apace to shed new light on the rapidity of microbial evolution, elucidating something of the incredible genetic diversity found among microbes, much of the mathematical modelling literature lags (far) behind modern experimental developments. Whether one uses equations that support the competitive exclusion principle, or game theory, adaptive dynamics, or many other techniques besides, they are doomed to be poor descriptors of microbial evolution, with inherently limited predictive capacity. I therefore argue that there is a real need to bring omics datasets into modelling frameworks whereby the physical function of observed genomic changes can be implemented in a manner that provides mechanistic insight into why those changes arise, sweep, fix or else coexist stably with other genomes in the population.

This is not an easy task. Although sequencing costs are reducing and so the degree of available genetic detail is ever-increasing, whole-genome (and other omics) datasets provide only a limited snapshot of what the microbes might be doing as they evolve. Many of the bacterial variants our alignment algorithms report can be mystifying for modellers and what seem to be minor changes in the evolutionary protocol one implements can impinge on important cellular function in seemingly unpredictable ways.

I will seek to illustrate some of these difficulties using mathematical models and our own laboratory data [2,3] of one attempt to understand an antibiotic phenotype called 'non-reciprocated collateral sensitivity' (PLoS Biology, in press). This study was motivated by a simple question: how low can antibiotic dosage be and yet pathogen clearance still occur, even given rapid (within 12h) drug-resistance evolution? We broach this question using drug treatments motivated by bang-bang controls in a laboratory model in which the resistance to two antibiotic drugs evolves at high mutation rates due to the duplication, and even triplication, of a genomic region amounting to 10% of the entire chromosome that contains a key drug efflux operon.

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R. Alexander Bentley, Dept. of Archaeology and Anthropology, University of Bristol, UK

Fitness landscapes among many options under social influence

(joint work with William A. Brock[1,2], Michael J. O'Brien[4], and Alberto Acerbi[3])

Abstract

We identify three important factors among organisms that learn socially: (1) social influence, (2) transparency, or intensity, of choice, and (3) change through time. Here we explore this model in terms of the fitness-landscape function in the spirit of Sewell Wright, which is a novel problem because the optimal decision depends not only on intrinsic utility of the decision/ behavior but also on transparency and social learning as well as the relative popularity of each of the possible choices in a population. This recursive relationship means that multiple equilibria can exist, and to search for these we employ a hill-climbing algorithm that leads to the expected values of the optimal decisions, which we define as peaks on the fitness landscape.

[1]Department of Economics, University of Missouri, Columbia, MO 65211, USA

[2]Department of Economics, University of Wisconsin, Madison, WI 53706, USA

[3]Department of Archaeology & Anthropology, Bristol University, BS8 1UU, U.K.

[4]Department of Anthropology, University of Missouri, Columbia, MO 6521, USA

Alex Best, School of Mathematics and Statistics, University of Sheffield, UK

Co-evolutionary cycles in host-parasite interactions: experiment and theory

Abstract

Understanding how the environment impacts the evolution and spread of infectious disease is a major interdisciplinary challenge. Recent experimental work has found that altering the environmental conditions can shift antagonistic co-evolution between hosts and parasites from a directional 'arms-race' to a fluctuating selection regime. This has important implications for management strategies as such cycles mean the parasite is a 'moving target'. It is therefore crucial that we develop theory that can reveal the environmental conditions that promote co-evolutionary cycles. In this talk, I shall first present experimental data from a bacteria-phage system demonstrating the role of resource availability in the promotion of fluctuating selection. I shall then introduce an epidemiological model that reflects the infection process of the bacteria-phage system, where bacteria are able to evolve the 'range' of phage that they can successfully resist and phage the 'range' of bacteria that they can successfully infect. Working within the framework of adaptive dynamics, I will then take a bifurcation approach to explore whether our theoretical model predicts the same. I shall show when cycles, arms races, intermediate CSSs and evolutionary branching may be expected, as well as exploring the pattern for other environmental gradients.

Virgile Baudrot, Laboratoire Chrono-environnement, Université de Franche-Comté, France

The influence of adaptive foraging on trophically transmitted parasite

(joint work with Clémentine Friscth[1], Antoine Perasso[1] and Francis Raoul[1])

Abstract

Small mammal populations display fluctuating patterns of densities in many ecosystems worldwide, and often act as keystone species. The ability for a generalist consumer to adapt its foraging strategy (the multi-species functional response, MSFR) is a milestone in ecology as it contributes to the strength and the stability of food webs. The predator-preys relationship is the route of transmission of a large number of zoonotic parasites with a complex life cycle (infecting different hosts during their life). Understanding parasite persistence in food web is of great interest, especially as two-third of human infectious diseases are zoonoses and the number of emerging zoonoses is increasing since the mid last century.

In this study, we explored the importance of MSFR and intermediate host diversity on transmission patterns of trophically transmitted parasites. We developed a general approach to model MSFR including accessibility to resources and switching of prey preference by predator. The red fox *Vulpes vulpes* is a typical definitive host (DH) of the cestode *Echinococcus multilocularis* (*Em*), the causative agent of the emerging zoonosis alveolar echinococcosis in humans. In eastern France, the rodents *Microtus arvalis* and *Arvicola scherman* are intermediate hosts of *Em* and the main preys of *V. vulpes*. Based on specific MSFR, we investigated the dynamical behavior of an ODEs system by coupling a compartmental model SIS (Susceptible-Infectious-Susceptible) for the DH and compartmental models SI (Susceptible-Infectious) for the intermediate hosts (IHs).

Based on this model, we computed the basic reproductive number R_0, i.e. the number of secondary infections caused by a single primary infection into an otherwise susceptible population. In view of disease control options, we expressed R_0 as a function of predator control (i.e. hunting) and showed a non-linear relation. Then, we explored the influence of different birth rates in IHs community (a driver of prey steady states) on this measure of disease risk. With the use of specific MSFR, we investigated how diversity of IHs may support the parasite dynamics. We showed that switching of prey preference is a critical property for the computation of R_0. Few works have coupled adaptive predator-prey interactions with parasite transmissions, and the inclusion of switching behavior is a first attempt to feed hot topics linking adaptive foraging behavior and IHs diversity. We showed that switching of prey preference may have a dilution effect, i.e. an increase of IHs diversity reduces the disease risk.

[1]Laboratoire Chrono-environnement, Université de Franche-Comté, France

Barbara Boldin, University of Primorska, Slovenia

Linking within- and between-host dynamics to study the evolutionary dynamics of pathogens

(joint work with Odo Diekmann[1])

Abstract

Pathogens reproduce and are subject to natural selection at several different, but intertwined, levels. We present a nested model in which pathogen transmission at the host-population level is mechanistically linked to within-host dynamics. We discuss possible evolutionary outcomes and focus in particular on how re-infection shapes the course of pathogen evolution. We also show that models mechanistically linking the two levels of pathogen dynamics are interesting from a theoretical point of view as they reveal a new type of evolutionary singularity (a so called one-sided ESS) and inspire a natural extension of the existing framework of Adaptive dynamics.

[1]Utrecht University, the Netherlands.

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[2] Boldin, B. Diekmann, O.: *An extension of the classification of evolutionarily singular strategies in Adaptive Dynamics.* Journal of mathematical biology, 69 (4), pp. 905-940 (2014).

Barbara Boldin, University of Primorska, Slovenia

An extension of the classification of evolutionarily singular strategies in Adaptive Dynamics

(joint work with Odo Diekmann[1])

Abstract

The existing classification of evolutionarily singular strategies in Adaptive Dynamics [2,3] assumes an invasion fitness that is differentiable twice as a function of both the resident and the invading trait. Motivated by nested models for studying the evolution of infectious diseases [1], we consider an extended framework in which the selection gradient exists (so the definition of evolutionary singularities extends *verbatim*), but where the invasion fitness may lack the smoothness necessary for the classification `a la Geritz et al. We present the classification of singular strategies with respect to convergence stability and invadability and determine the condition for the existence of nearby dimorphisms. The extended setting allows for a new type of evolutionary singularity: a so called one-sided ESS that is invadable by mutant strategies on one side of the singularity but uninvadable by mutants on the other side. We discuss possible evolutionary scenarios nearby one-sided ESSs and conclude by applying the extended framework to nested models of infectious disease dynamics. The talk is based on joint work with Odo Diekmann [4].

[1]Utrecht University, the Netherlands.

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Gunnar Brandt, Systems Ecology, Leibniz Center for Marine Tropical Ecology, Bremen, Germany

Virulence evolution in fragmented host populations with infectivity–mobility trade-offs

(joint work with Merico, A.[1,2] and Kulesz, M.M.[3])

Abstract

The overexploitation of marine resources is a long-standing and ubiquitous problem mainly driven by the increasing global population. Current mathematical models describing the exploitation of fish stocks usually consider a multitude of factors to understand the complex dynamics of biological systems. However, management strategies based on such modelling studies, typically consist in alleviating overfishing by implementing an upper acceptable limit for resource extraction. While successful when monitoring and enforcement is efficient, this approach may completely fail in less-restricted, open-access systems, in which the compliance with imposed regulations is often poor.

We present here an adaptive dynamics model, in which harvest behaviour is explicitly simulated as a dynamic trait. When deciding on their actual harvest, resource users are subject to an apparent trade-off between immediate returns and the long-term sustainability of the resource. Our results reveal the intimate relationship between the users' believes about future returns and their ability to sustainably exploit their resource.

To better understand the co-evolving dynamics of resource and user behaviour, we develop a time-continuous common pool resource game that is implemented as an application on connected mobile devices. The combination of our adaptive model with results from the app-based behavioural experiments will allow us to better comprehend typical phenomena observed in real resource systems and presents a promising approach to substantially advance our understanding of human behaviour driving overexploitation.

[1]Systems Ecology, Leibniz Center for Marine Tropical Ecology, Bremen, Germany.

[2]Jacobs University Bremen, Germany.

[3]Institutional and Behavioural Economics, Leibniz Center for Marine Tropical Ecology, Bremen, Germany.

Katharina Brinck, Centre for Complexity Science, Imperial College London, UK

The evolution of ecosystem organisation: a complexity science approach

(joint work with Henrik Jeldtoft Jensen[1])

Abstract

The dynamics and organisation of ecological systems take place at a range of different scales and are generally approached either from a reductionist perspective, constructing ecosystems applying a bottom-up approach, or from the macroecological perspective, analysing ecological patterns from a top-down perspective. In recent years, the need for integrative and process oriented approaches to capture ecosystem growth, development and organisation, as well as the scope of information theory as a descriptive tool has been addressed from various sides. However data collection of ecological network flows is difficult and tedious and comprehensive models are lacking. To understand how the organisation of ecosystems, evolves over time, it is necessary to bridge the gap between the reductionist and holistic approaches with a complex systems' perspective, which aims to explain the emergence of the macroscopic properties from the relationship between the microscopic parts. We present a hierarchical version of the Tangled Nature Model of evolutionary ecology [Laird et al., 2008], an individual based stochastic model, in which species are emergent structures arising from the interactions between individual organisms, forming quasi-stable communities, interspersed with periods of rapid change. This model of entangled food web evolution allows for insights in the relations between structure, flow and organisation in model ecosystems, their development over evolutionary time scales and their relation to ecosystem stability. Those theoretical insights are valuable to assess the role of bottom-up and top-down controlling forces in shaping the organisation of ecosystems, can shed light on general selection principles from a ecosystems perspective and can eventually help to forecast and potentially prevent periods rapid change accompanied by mass extinctions in real-world ecosystems.

[1]Centre for Complexity Science, Imperial College London, UK

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Vincent Calcagno, INRA, Sophia Antipolis, France

The interplay of colonization and evolution in models of adaptive radiation

(joint work with P. Jarne[1], M. Loreau[2], N. Mouquet[3] and P. David[1])

Abstract

The buildup of diversity in ecological communities (community assembly) has classically been modelled as an immigration/invasion process, where species from the regional pool colonize at a certain rate, and ecological dynamics determine their fate on and impact on the resident community. More recently, models of evolutionary community assembly have studied the local buildup of diversity (adaptive radiation) as a consequence of mutation and selection from an ancestor genotype, in an isolated community. However, it is well-known that natural communities are often assembled by a combination of the two above processes. Islands are good examples of this, with adaptive radiation and the production of endemic species dominating in very remote archipelagos, and immigration of species prevailing closer to mainland. Theoretically, it is not very well understood how the two processes should interact when both are operating. Here we will study the buildup of diversity under the action of (i) mutation/selection and (ii) occasional invasions of species from outside the community, in a model where species interactions are governed by a competition-colonization trade-off. We will show how species invasions can qualitatively change the type of (co)evolutionary dynamics, and how the two processes can act synergistically. An effort will then be made to evaluate the generality of conclusions in the context of other modes of ecological interactions, such as symmetric competition along a niche axis, or body-size mediated trophic interactions.

[1]CEFE/CNRS Campus du CNRS, Montpellier 5, France

[2]Station d'Ecologie Expérimentale du CNRS, Moulis, France

[3]CNRS, Université Montpellier 2, France

Pablo Catalan, Grupo Interdisciplinar de Sistemas Complejos (GISC), Universidad Carlos III de Madrid, Spain.

toyLIFE: the complexities of the genotype-phenotype map

(joint work with Clemente F. Arias[1], Susanna Manrubia[1] and José A. Cuesta[1])

Abstract

The understanding of the genotype-phenotype map is arguably one of the biggest challenges faced by modern evolutionary biology. Different theoretical models have been proposed to explore it, from RNA and protein folding, to gene regulatory and metabolic networks, and many important insights have been gained from them.

However, most of these theoretical models focus only on one level of the map: folded molecules, regulation or metabolism. As a result, they miss two important features of the complex genotype-phenotype map: the coexistence of different levels of expression – i.e. molecules, regulation and metabolism occur at the same time and place in the cell – and the bidirectional interaction between those levels.

Here we present toyLIFE, a multi-level model for the genotype-phenotype map that includes simple genes, proteins and metabolites. These molecules interact through the laws of a simplified chemistry to form complex regulatory networks and develop metabolic functions. toyLIFE allows us to investigate how different levels are coupled, in particular how and where mutations affect the phenotype or how the presence of certain metabolites determines the dynamics of toyLIFE gene regulatory networks. The model can easily incorporate evolution through more complex mutations, recombination, or gene duplication and deletion, thus opening an avenue to explore extended genotype-phenotype maps.

[1]Grupo Interdisciplinar de Sistemas Complejos (GISC), Universidad Carlos III de Madrid, Spain

Christina Cobbold, University of Glasgow, UK

Modelling the evolution of cold tolerance and adaptation to temperature changes: application to the mountain pine beetle

Abstract

Ectotherms rely on environmental heat sources to control their body temperature and as such temperature can play an important role in determining the development time and phenology and hence fitness of the organism. Mountain pine beetle is an important example of an ectotherms. In response to warmer temperatures, the mountain pine beetle has substantially increased its geographic range killing trees as far north as the Yukon Territory in Northern Canada. Recent work has suggested that MPB has been able to spread beyond its previous range by adjusting its cellular and metabolic functions, via selective adaptation, enabling populations to better withstand cooler climates.

The focus of my talk will be to examine how insect populations might adapt to changes in temperature and novel thermal environments. I will give an overview of some approaches for integrating climatic information into models of insect development. I will then present a quantitative genetics model for the evolution of cold tolerance, the ability to survive winter freezing, and explore how mountain pine beetle may adapt and evolve in response to warming temperatures and discuss the population consequences of these adaptations.

Caroline Colijn, Imperial College London, UK

Phylogenetic trees and outbreaks of pathogens: mapping one kind of tree onto the other.

Abstract

Sequencing technologies have made it possible to detect genetic variation with very fine resolution. As a consequence, it is possible to use sequences from pathogens spreading in a population to obtain a more clear understanding of transmission events than was possible previously. However, the best way to do this is not clear. Pathogen sequence data are usually interpreted with the aid of a phylogenetic tree; outbreaks are usually reconstructed with the aid of a transmission tree describing who infected whom. These trees are related, and one would think that each is quite informative of the other, but they are very different: phylogenetic trees are typically bifurcating trees with individual taxa (here, the pathogens from the human hosts) as tips and inferred common ancestors as nodes, with edges reflecting ancestry. Transmission trees have individual hosts as nodes and edges reflecting transmission events. The situation is further complicated by the fact that host can have more than one lineage of a pathogen inside them. Here we describe a way to use colouring to map transmission trees onto phylogenetic trees, in a way that can capture in-host diversity. We use this mapping to write the likelihood of a transmission tree given a phylogenetic tree. The approach is flexible, so the transmission process can be defined with a renewal model or an SIR-type model, and can cope with unsampled individuals. We use this likelihood to perform Bayesian inference of the transmission tree. Using data from an outbreak of tuberculosis in Canada, we find that sequence data (via phylogenetic trees) can aid in the understanding of transmission events in outbreaks, but that the inference of individual transmission events remains quite uncertain. However, we robustly detect small transmission clusters and identify the source case. We conclude with a discussion of the role of sequencing studies in understand the spread of infections.

Tamas David-Barrett, University of Oxford, UK

The evolution of constrained sociality

Abstract

All group living species face the problem of collective decision making, action coordination, and in – in many cases – of free-riding. A subset of species solved this problem via constrained sociality, that is, the behaviour in which positive social affiliation is limited to a small number of others. Empirical studies of primate behaviour suggest that the underlying behaviour, the need to build long-term cooperative relationships among non-kin, arises due to complex ecological environments that are particularly difficult to exploit. At the same time a recent theoretical paper suggests that in a behavioural synchrony framework the presence of constrained sociality in large groups results in highly structured social networks. This talk merges these results, in an evolutionary agent-based model of collective action in which agents can inherit their traits driving their ego network constructing behaviour; and shows that constrained sociality can be an evolutionary adaption to group living in high dimensional environments.

Andrew Dean, Department of Biology, University of York, Heslington, York

Modelling the evolution of symbiosis

Abstract

Mutualistic symbioses, in which both host and symbiont benefit from the relationship, are a process fundamental to ecosystems and have repeatedly evolved. In order to understand the evolutionary transition from free-living species to stable symbiosis, we have developed a model of the relationship between a heterotrophic host and its phototrophic symbionts. This is informed by working closely with experimentalists studying an existing symbiosis between the host ciliate Paramecium bursaria and the green algae Chlorella. Our model shows the resulting trade-off between the costs (nutrition, membrane maintenance, etc.) and benefits (increased access to carbon) selects a symbiont distribution with a clear maximum. Allowing the host a level of control over its symbionts shifts this maximum to the point at which nutrient uptake is optimal for host growth. Metabolic reconstructions have enabled us to understand the underpinning exchange metabolites which offer insights into model construction.

Florence Débarre, University of Exeter, UK / Wissenschaftskolleg zu Berlin, Germany / Centre for Interdisciplinary Research in Biology, CNRS UMR, Paris, France

(Co)evolution in multiple dimensions: how does the number of traits under selection influence evolutionary and coevolutionary processes?

(joint work with S. Nuismer[1], M. Doebeli[2])

Abstract

The complexity of biotic and abiotic environmental conditions is such that the fitness of individuals is likely to depend on multiple traits. Using a synthetic framework of phenotypic evolution that draws from adaptive dynamics and quantitative genetics approaches, we explore how the number of traits under selection influences convergence stability and evolutionary stability in models for coevolution in multidimensional phenotype spaces. Our results allow us to identify three different effects of trait dimensionality on stability. First are (i) a "combinatorial effect": without epistasis and genetic correlations, a higher number of trait dimensions offers more opportunities for equilibria to be unstable; and (ii) epistatic interactions, that is, fitness interactions between traits, which tend to destabilize evolutionary equilibria; this effect increases with the dimension of phenotype space. These first two effects influence both convergence stability and evolutionary stability, while (iii) genetic correlations (due, e.g., to pleiotropy or linkage disequilibrium) can affect only convergence stability. We illustrate the general prediction that increased dimensionality destabilizes evolutionary equilibria using examples drawn from well-studied classical models of frequency-dependent competition for resources, adaptation to a spatially heterogeneous environment, and antagonistic coevolution. In addition, our analyses show that increased dimensionality can favour diversification, for example, in the form of local adaptation, as well as evolutionary escape.

[1]Department of Biological Sciences, University of Idaho, Moscow, Idaho, USA

[2]Department of Zoology and Biodiversity Research Centre, University of British Columbia, Vancouver, Canada

Zhilan Feng, Purdue University, USA

Coupled within –host and between-host dynamics and evolution of virulence Part I: Disease dynamics of the coupled system

Abstract

Mathematical models coupling within- and between-host dynamics can be helpful for deriving trade-off functions between disease transmission and virulence at the population level. Such functions have been used to study the evolution of virulence and to explore the possibility of a conflict between natural selection at individual and population levels for directly transmitted diseases. In this two-part talk, we present a new model for environmentally-driven diseases, which couples a standard SI type of epidemiological with a standard model for cell-pathogen interactions. It is analyzed to study similar biological questions related to evolution of virulence. The model exhibits emerging dynamical behaviors including the possible occurrence of a backward bifurcation. It is demonstrated that optimal parasite strategies will maximize these reproduction numbers at the two levels, and a conflict may exist between the two levels. The results highlight the role of inter-dependence of variables and parameters in the fast and slow systems for persistence of infections and evolution of pathogens in an environmentally-driven disease. The results also demonstrate the importance of incorporating explicit links of the within- and between-host dynamics into the computation of threshold conditions for disease control.

Tobias Galla, School of Physics and Astronomy, University of Manchester, UK

Stochastic evolutionary delay dynamics in epidemiology and gene regulation

(joint work with Tobias Brett[1])

Abstract

Traditionally biological systems are modelled by deterministic differential equations. This is undoubtedly because of the relative mathematical simplicity of this approach – the theory of ordinary and partial differential equations is well developed. These deterministic descriptions systematically neglect the effects of noise though. Stochasticity and intrinsic uncertainty can be relevant to the outcome of dynamical systems though, and induce phenomena such as fixation and extinction, noise-driven cycles, patterns and waves. This has been demonstrated in numerous examples, ranging from birth-death processes in evolutionary game theory, over models of epidemic spread to predator-prey dynamics and gene regulation. Analytical tools to characterize these effects are now well established and in place, these include the theory of stochastic processes and ideas from statistical physics.

Most of this work focuses on Markovian dynamics, in which the future evolution of the system depends only on its present state, but not on the path taken to arrive at the current state. Intrinsic delays are of importance though in many biological systems: transcription and translation in genetic systems only complete after a certain time, individuals are subject to gestation periods in population dynamics, and the recovery from diseases is not an exponential process, but instead occurs after characteristic recovery periods.

Existing work on non-Markovian dynamics is frequently restricted to deterministic delay differential equations. Stochastic individual-based systems with delay have only been considered more systematically relatively recently, and a more comprehensive theory is still very much under construction. The theory of Markovian random processes does not apply, and approaches based on master or Fokker-Planck equations is not easily transferred.

In my talk I will present recent work on systematic Gaussian approximations to individual-based models with non-Markovian dynamics. I will first describe how these can be derived from a path-integral approach, and I will then discuss a more heuristic derivation. I will apply the resulting theory to several exemplars, in particular delay models of epidemic spread, models of gene regulation, and an excitable chemical system derived from the well-known Brusselator. I will illustrate the effects intrinsic noise has on these effects, and I will detail how systematic Gaussian approximations can be used to analyse or simulate such individual-based systems efficiently.

[1]School of Physics and Astronomy, University of Manchester, UK.

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Andy Hoyle, Computing Science and Mathematics, University of Stirling, UK

Evolution of antibiotic resistance in aquatic bacteria – biofilms vs well-mixed models.

Abstract

Antibiotic resistance is one of the biggest problems faced by humans in the modern day. However, little is known about the size of the problem in the environment, in particular in aquatic systems. The key to controlling the problem lies at the bacterial level. There are several ways bacteria resist or tolerate antibiotics, all providing a selective advantage to resistant cells. However, one resistance mechanism is unique, the release of beta-lactamase (an enzyme) into the environment. This mechanism changes the typical selective pressures and provides an advantage to both resistant and susceptible cells. By using a range of modelling techniques, from well-mixed population to a fully spatially-structured population, I will show how this mechanism affects the bacterial population on both a short-term time scale, identifying what conditions allow susceptible cells to survive where they previously could not, and on a long-term time scale, allowing the strength of resistance to evolve, and show how the optimal resistance strength (amount of enzyme released) changes depending on spatial structure.

Weini Huang, Max-Planck-Insitute for Evolutionary Biology, Plön, Germany

Stochastic game dynamics under demographic fluctuations

(joint work with Christoph Hauert[1] and Arne Traulsen[2])

Abstract

Interactions between different types can lead to frequency dependent selection, where the fitness of an individual depends on the frequencies of its own type and the other interacting types. Under frequency dependent selection, the average fitness of the whole population may increase or decrease, as new interactions arise under mutation. This can cause fluctuation in population size even in a constant environment. Thus, frequency dependent selection and demographic stochasticity are indivisible in some biological systems. Here, we propose a stochastic approach to combine these two evolutionary ingredients naturally. We assume competitive interactions based on evolutionary games, which result in changing population size. In the limit of large populations, the averaged stochastic dynamics can be captured by the deterministic rate equations. In small populations, stochastic dynamics can lead to the extinction of the whole population.

[1]University of British Columbia, Vancouver

[2]Max-Planck-Insitute for Evolutionary Biology, Plön, Germany

Vincent Jansen, Royal Holloway University of London, UK

Inclusive fitness models that include ecological detail: the evolution of investment in siderophore production

Abstract

The bacterium Pseudomonas aeruginosa sequesters iron from the environment through the secretion, and subsequent uptake, of iron-binding molecules. As these molecules can be taken up by other bacteria in the population than those who secreted them, this is a form of cooperation through a public good. Traditionally, this problem has been studied by comparing the relative fitnesses of siderophore-producing and non-producing strains, but this gives no information about the fate of strains that do produce intermediate amounts of siderophores. Here, we investigate theoretically how the amount invested in this form of cooperation evolves using assumptions for a simple ecological setting. We formulate a model for the local dynamics which describes the competition and cooperation of the bacteria. From this dynamical model we derive the fitness following the adaptive dynamics method. The results show how selection is driven by local siderophore production and local competition. Because siderophore production reduces the growth rate, local competition decreases with the degree of relatedness (which is a dynamical variable in our model). The adaptive dynamics approach allows us to assess evolutionary stability, and we found that selection can lead to an intermediate strategy which in our model is always evolutionarily stable. Our model describes the evolution of a public good in the context of the ecology of the microorganism, which allows us to relate the extent of production of the public goods to the details of the interactions.

Jose Jimenez, FHMS, University of Surrey, UK

Comprehensive experimental fitness landscape and evolutionary network for RNA

Abstract

The origin of life is believed to have progressed through an RNA world, in which RNA acted as both genetic material and functional molecules. In this prebiotic context, the evolutionary fate of the first functional sequences of RNA would be determined by the structure of the evolutionary fitness landscape of the molecules, which would condition their survival during processes of natural selection. Although fitness landscapes are the subject of much speculation, their structure is essentially unknown. In this work we describe the first comprehensive map of an experimental fitness landscape, exploring nearly all of sequence space, for short RNAs surviving selection in vitro. The landscape was generated synthesizing a library of aptamers of RNA including almost all possible combinations of length 24 with a 1000 times coverage of each unique sequence. The resulting pool of 10^{17} molecules was subject to screening for binders to GTP attached to a resin. The evolutionary dynamics of the experiment in successive rounds were monitored by deep-sequencing and the biochemical properties of the most relevant hits were characterized. Based on the results obtained by that approach we estimated the fitness of all sequences surviving the selection and clustered them in families according to their sequence similarity. With the exception of a small evolutionary network, we find that fitness peaks are largely isolated from one another, highlighting the importance of historical contingency and indicating that natural selection would be constrained to local exploration of the space of sequences in the RNA world.

Gereon Kaiping, Computational Engineering and Design, University of Southampton, UK

Structured populations facilitate cooperation in policed Public Goods Games

(joint work with T. J. Sluckin[1] and S. J. Cox[2])

Abstract

Societies consisting of cooperative individuals seem to require for their continuing success that defectors be punished. The precise connection to benefits, population structure, and division of labour do however remain illunderstood. While most models assume costly “Peer punishment” to enforce cooperation in a system, results from the economics literature cast doubt on the general validity of this assumption. Starting from such observations, we present several extensions to the public goods game with punishment, and evaluate their influence on the level of cooperation seen in a system.

We find that even a weak metapopulation structure can have a strong effect on the evolutionary dynamics of a system and facilitate cooperation. Enabling fitness transfers to punishers has a small positive effect when voluntary, but leads to very cooperative systems if compulsory. Forcing defectors to contribute can support cooperation, but in the presence of group competition it can actually be deleterious

[1]Computational Engineering and Design, University of Southampton, UK

[2]Applied Mathematics, University of Southampton, UK

Anne Kandler, Department of Mathematics, City University London, UK

Inference of learning strategies from frequency data

Abstract

Cultural change can be quantified by temporal frequency changes of different cultural artefacts. Based on those (observable) frequency patterns researchers often aim to infer the nature of the underlying cultural transmission processes and therefore to identify the (unobservable) causes of cultural change. Especially in archaeological and anthropological applications this inverse problem gains particular importance as occurrence or usage frequencies are commonly the only available information about past cultural traits or traditions and the forces affecting them. Matters are further complicated by the fact that observed changes often describe the dynamics in samples of the population of artefacts whereas transmission processes act on the whole population. In this talk we start analyzing the described inference problem. Assuming the situation where two samples of cultural artefacts are known at time points t_1 and t_2 we firstly generates population structures from which the observed sample could have been drawn randomly at a time t_1 and then determine theoretical samples at time t_2 produced under the assumption that changes in frequencies are caused by a specific transmission process (in particular we explore neutral evolution, frequency- and age-dependent selection). Subsequent statistical comparisons (e.g. using Bayesian inference) of the theoretical and observed samples at t_2 can establish which processes could have produced the observed frequency data. In this way we aim to infer underlying transmission modes directly from available data without any optimality or equilibrium assumption. Importantly this approach allows us to explore the theoretical limitations of inference procedures based on population-level data and to start answering the question of how much information about the underlying transmission processes can be inferred from frequency patterns. Our approach might help narrow down the range of possible processes that could have produced observed frequency patterns, and thus still be instructive in the face of uncertainty. Rather than identifying a single transmission process that explains the data, we focus on excluding processes that cannot have produced the observed changes in frequencies. We demonstrate the applicability of the developed framework to archaeological case studies.

Michelle Kendall, Imperial College London, UK

A new metric for the comparison of phylogenetic trees

(joint work with Caroline Colijn[1])

Abstract

We propose a new metric for rooted, labelled, binary trees which distinguishes trees in a different way from existing phylogenetic metrics. In particular, our metric captures something of the difference in the `biological story' told by the trees, with an emphasis on comparing shape, root placement, lineages and, if desired, branch lengths. We will outline some of the applications where our metric has already proven useful. These include the comparison of tree inference methods, comparing gene trees to full sequence trees, sorting the trees from an MCMC chain, and visualising tree space, which enables us to detect `islands' (collections of trees with similar likelihood and topology). Finally we will describe some work in progress where we use the metric to find informative sites in sequence alignments.

[1]Imperial College London, UK

Bhavin S. Khatri, National Institute for Medical Research, London, UK

A simple biophysical model of protein binding DNA predicts higher rates of speciation in small populations

(joint work with Richard A. Goldstein[1])

Abstract

Speciation is fundamental to understanding the huge diversity of life on Earth. Evidence suggests reproductive isolation arises most commonly in geographic isolation (allopatry). Current theory does not address how the speciation rate varies with population size in the important weak mutation regime of evolution, despite some evidence that smaller populations develop reproductive isolation more quickly. In addition, increasing data suggest that many species differences involve a divergence in the regulation of gene expression and that a number of speciation genes involve transcription factors. Here, we address an underlying biophysical basis of speciation by using a simple model of transcription factor-DNA binding and examine how their co-evolution in two allopatric lines leads to incompatibilities. We tackle this using both theory and simulations of sequence evolution. To develop a tractable analytical theory, we derive a coarse-grained Smoluchowski Equation for the dynamics of binding energy evolution due to the co-evolution of protein and DNA sequences; the high dimensionality of sequence evolution is accounted for by defining a Boltzmann sequence entropy for the binding energy of transcription factors, such that the flux of populations go up gradients in Iwasa's free fitness [1,2]. We find these simple considerations lead to a new prediction for the monomorphic regime of evolution, born out by theory and simulations, that smaller populations should develop incompatibilities more quickly; this arises as the effect of sequence entropy is to poise the common ancestor of smaller populations more closely to incompatible regions of phenotype space, so less substitutions are needed on average for incompatibilities to arise. Overall, these predictions are consistent with observations of large species diversity in small habitats such as Cichlids in the East African Great Lakes, contrasted with the observed smaller rate of developing reproductive isolation in marine animals and birds, which have large ranges and population sizes. In particular, our results directly predict how hybrid binding energies change with divergence time, which we suggest can be directly tested using ChipSeq analysis of transcription factor intra- and inter-species genomic binding.

[1]Division of Infection and Immunity, University College London, UK

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Ádám Kun, Parmenides Conceptual Foundation of Science, Parmenides Foundation, Germany; ELTE-MTA-MTM Ecology Research Group, Pázmány Péter sétány 1/C, Budapest, Hungary

The minimal genome and functionality of a ribo-organism

Abstract

The first living cell emerged in the RNA world, a stage in the origin of life when RNA acted both as information carrier and enzyme. This ribo-organism was simpler than the last universal common ancestor (LUCA) of all extant living being, for example it had no translation yet. Estimates of the minimal genome of bacterial life are around 200-300 genes. These set of genes mainly deal with transcription, translation, peptide metabolism, membrane functions, cellular level processes and intermediate metabolism. Intermediate metabolism is assumed to take many of its smaller constituents up from the environment, leaving energy production (glycolysis), nucleotide assembly, cofactor assembly and lipid metabolism to ribozymes.

A ribo-organism can function with fewer genes, as it has no translation (peptide synthesis) and no DNA (no ribonucleotide reductases are required, for example). On the other hand, many of the estimates of minimal gene content – in my opinion – underestimate the number of cellular processes that requires active control. Theoretical biology can help uncover the requirement for dynamical stability of the system and for the coexistence of all its components. Coexistence is not guaranteed as cheating elements, RNA that accept the catalytic help provided by the cell, but which does not contribute to it, abound. Tight control, for example, over what is passed on to the next generation helps to alleviate this problem. Chromosome (i.e. all genes strung together into one molecule) is a necessity for stable inheritance, but then it also requires function to “liberate” the genes from it, so that they can function (transcription). Chromosomes need to be divided into daughter cells, which raises the question of the early origin of cytoskeleton or cytoskeleton like function. Furthermore, the cell need transport processes so it won’t leak important components out, but can still take up what it needs.

In the end we propose that around a 100 genes were necessary for the first cellular ribo-organism. This is still a long jump from the cooperative ensemble of a few enzymes that could coexist on mineral surfaces. We also discuss possible pathways for this transition.

Oleg Kuzenkov, Lobachevsky State University of Nizhni Novgorod, Russia.

Revealing patterns of optimal zooplankton diel vertical migration on the basis of dynamics of the underlying measure.

(joint work with A. Morozov[1], E. Ryabova[2])

Abstract

When modelling evolving biological systems one of the most efficient approaches is based on the considering dynamics of the underlying measure. This approach was first proposed by A. Gorban in 1985 [1] and it is now used for describing self-replicating systems, for example, in works of G. Karev [2,3]. In such systems, the dynamics of species densities is considered in the space of genotypes and selection processes are studied in this space. Applying the method allows us to formulate and prove a variational principle of the survival of genotypes, which would eventually provide the maximum of the reproduction coefficient.

Here we use the above approach to build a mathematical model of diel vertical migrations of zooplankton in deep lakes and the ocean, which has been a long standing a hot topic in ecology. We consider the dynamics of species in the space of inherited behavior strategies in accordance with the Verhulst's law of the biomass growth. The order of preference is introduced on the set of genetic strategies of behavior in the infinite dimensional space as a result of selection. Unlike earlier publications on the topic, we consider here continuous strategies of organisms (the choice of optimal depth at each moment of time), i.e. the strategies are elements of an infinite dimensional functional space (in particular, we take into account both the current position of the organism and its instantaneous velocity). The introduced order of preference is expressed mathematically via a certain functional, maximization of which results in a variational principle of natural selection. Based on this principle we derive the optimal strategy for vertical migration of zooplankton and investigate the dependence of the shape of the resultant trajectories on the key model parameters (the load of predators, water resistance, food abundance, etc) . The results obtained through modelling are compared to the real data from the ocean.

[1]Department of Mathematics, University of Leicester, UK
[2]Lobachevsky State University of Nizhni Novgorod, Russia

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Galina Kuzenkova, Lobachevsky State University of Nizhni Novgorod, Russia.

Controlled selection process of self-replicating systems

(joint work with O. Kuzenkov[1])

Abstract

The problem of control for self-replicating systems is of great practical importance and it is very relevant now. This problem has been considered by many authors, for example, Zaslavsky, Poluektov, Svirezhev, Maillert, Grognard, and others.

In this paper, the controlled self-replicating system with initial conditions is considered:

$$\dot{z}_i = \left(A(z,t)\phi((b_i + u_i(t))z_i) + B(z,t)\right)z_i, \quad z_i(0) = z_i^0, \quad i = 1, \quad n.$$

Here $z = (z_1, \ldots z_n)$ – numbers of coexisting species, A, B, ϕ_i – smooth bounded functions, ϕ_i - monotone functions, u_i – control functions satisfying the constraints $|u_i| \le c_i, i = 1, \ldots n$; b_i, c_i – constants.

This system is the generalization of a class of mathematical models: Volterra's, Lotka's, Monod's ets. The control time is unlimited. The aim of control is to achieve the selection of one of the species. The quality criterion of control is formalized in the form of the time-average value reproduction coefficient.

We find that the necessary and sufficient conditions for the control resource solving this problem have been obtained in the form of an inequality

$$c_1 > \max_{j=2,\ldots n} \left(b_j - c_j\right)\frac{z_j^0}{z_1^0} - b_1.$$

Control synthesis has been done to solve the problem. The results are applied for various models: Verhulst's, Volterra's, Monod's ets.

Fabien Laroche, CEFE – CNRS, Montpellier, France

Evolution of dispersal impacts species diversity patterns in a heterogeneous metacommunity.

Abstract

A metapopulation model recently suggested that variation in carrying capacity among patches can select for various degree of dispersal polymorphism. Here, we extend this result to the community level: we splice dispersal evolution in an otherwise neutral metacommunity model and vary community size distributions. Dispersal polymorphism could readily emerge within but also among species. When several stable levels of dispersal emerge in the metacommunity, groups of species with different dispersal levels emerge and small communities are occupied by dispersing species while large communities harbor species with limited dispersal, leading to a significant turnover of species composition between small and large communities. By contrast, when dispersal is distributed around a unique evolutionarily stable strategy, no significant turnover of species occurs between small and large communities, in agreement with neutral expectations. Spatial turnover of species among communities of different carrying capacities may thus indicate strong among-species dispersal polymorphism coming from stable coexistence of several dispersal strategies within the metacommunity.

Ester Lázaro, Centro de Astrobiología (INTA-CSIC), Madrid, Spain

Transient increases in the error rate can open new pathways for adaptation to new selective pressures

Abstract

The replication error rate is one of the main parameters influencing the extension of genetic diversity contained in a population. High error rates are associated to a wider exploration of the genotype space, which, within a range of values, can increase the ability to adapt to new selective pressures. However, since most mutations are deleterious, there must be an upper limit for the error rate which is compatible with the survival of populations. Above this limit, beneficial mutations probably appear in genomes that already contain a certain number of deleterious mutations. This fact can limit the spread of beneficial mutations generated at increased error rate, reducing the population adaptability. To get a deeper insight into this point, we propagated two populations of an RNA virus, the bacteriophage Qβ, at either the standard error rate of the virus or at artificially increased error rate through the presence of a mutagenic nucleoside analogue (5-azacytidine or AZC). Following this, populations were exposed to a new selective pressure that consisted in an increase in the replication temperature from 37ºC to 42º C.

The results obtained show that both populations increased their fitness at 42º C although through different adaptive pathways. It is intriguing that some of the mutations selected in the virus evolved at increased error rate had previously been detected as polymorphisms in a long-term evolution experiment in which Qβ was exposed to a gradual increase in the AZC concentration [1], suggesting that some of the mutations favoured at increased error rate can have selective value at high temperature. We are currently developing some experiments to understand the reasons underlying this unexpected relationship. The results obtained will be described in this presentation.

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Olof Leimar, Department of Zoology, Stockholm University, Sweden

Social evolution and genetic polymorphism

Abstract

There are many situations where relatives interact while at the same time there is genetic polymorphism in traits that are involved in the interaction. Examples include cheater-cooperator polymorphisms and interactions between microbial pathogens and their hosts. These situations are often characterized by environmental heterogeneity. There is, however, no general theoretical framework of social evolution that encompasses genetic polymorphisms in heterogeneous environments. Here I will show how traditional theoretical approaches to social evolution can be integrated into the evolutionary ecology of heterogeneous environments, including situations where there is adaptively maintained genetic polymorphism, by applying the concept of genetic cues. I will present a model of social evolution in a two-habitat situation, with limited dispersal between habitats, where the average relatedness at the time of helping and/or other benefits of helping differ between habitats. A novel aspect is that alleles at a polymorphic locus play the role of genetic cues, in the sense that the presence of an allele contains statistical information about the current environment, including information about relatedness. Another novel aspect is the perspective of genetic conflicts between genes at or tightly linked to the genetic cue locus and genes that are more loosely linked. An overarching idea is that the genetic cue can be regarded as playing a similar role as an environmental cue, in the sense of providing information about selectively relevant circumstances. This idea forms the basis for integrating genetic polymorphism into the traditional theories of social evolution. A main result of the model is that genes unlinked to the genetic cue locus will act to modify the effects of tightly linked genes to make them less extreme. The reason is that unlinked genes become adapted to exist in both habitats, be transferred between them, and to use the information in the genetic cue to adjust the phenotype in an optimal way. Tightly linked genes, on the other hand, will be selected to perform well in one of the habitats, even at the expense of performance in the other habitat. The reason is that an allele tightly linked to the cue locus becomes concentrated on one of the habitats, with the other habitat acting as a sink for that allele, to which little adaptation takes place.

Magnus Lindh, Umeå University, Sweden

Early starters beat optimal reproduction strategy in evolutionary game with annual plants

(joint work with Jacob Johansson[1])

Abstract

Understanding phenology is becoming increasingly complex as green house gases causes temperature, precipitation and CO_2 levels to drift. Annual plants use all these abiotic parameters as cues for germination and flowering, and plant phenology is already responding in unpredictable ways the new disturbances. We explore how germination and flowering times in annual plants respond to changes in productivity and mortality. Using an eco-evo model, based on previous optimal control models for annual plants, we want to understand when competition for a common resource gives an evolutionarily stable strategy different from the strategy maximizing reproductive output.

Plants are often assumed to maximize for example reproductive output, even though they are usually involved in an evolutionary race with other species competing for common resources. Examples of evolving plant organs are excessively long tree trunks among plants competing for light, and over proliferating roots among plants competing for water. In our model plants are competing for a common resource, e.g. water, light and/or nutrients, giving a constrained vegetative growth.

We show that plants growing in seasonal productivity and constant mortality, starting at germination, have an evolutionary stable strategy far from the strategy maximizing reproductive output. When competing for a common resource we find that plants generally evolve to germinate earlier and flower later, compared to the optimal reproduction strategy. This gives a clue to why growing seasons tend to get longer as an effect of global warming, even though biological development is expected to speed up as temperatures are rising.

[1]Lund University, Sweden

Sebastien Lion, CEFE, CNRS, Montpellier, France

Spatial structure, host heterogeneity and parasite evolution: implications for vaccination

Abstract

Evolutionary epidemiology aims at understanding how host-parasite interactions evolve in response to various ecological factors. However, theoretical studies often assume that the host population is well-mixed, thereby neglecting potential selective pressures caused by genetic and epidemiological spatial structuring. I will present some theoretical and experimental results in order to elucidate the evolutionary impacts of parasite and host dispersal patterns. I will first focus on a population where all hosts have the same quality, and show that the predictions of non-spatial theory are altered by kin competition for susceptible hosts. I will then examine what happens in a heterogeneous population in which a fraction of the hosts are vaccinated. I show that different types of vaccines may lead to different evolutionary outcomes, which depend on the interplay between vaccine efficacy, vaccination coverage, and spatial structure. Kin selection is shown to be a useful conceptual tool to understand the ecological feedbacks on parasite traits and to generate predictions for the management of infectious diseases.

Carlos A. Lugo-Vélez, the Sainsbury Laboratory, UK

Genomic evolution of pathogens as a consequence of host shifts.

Abstract

Comparative genomic analysis of pathogens belonging to a single clade alongside their free-living counterparts, exhibit extraordinary differences in genome complexity and length mostly in regions related to the regulation of the host-specific pathogenic activity. Whereas, the regions not related to such specific activity the genomes exhibit high degree of conserved domains.

The emergence of pathogen clade elements as the results of abrupt changes in the environmental conditions such as host shifts or "jumps" may be held responsible as evolutionary triggers of those differences. I will present results obtained by applying ideas of near neutral evolution and a simple genotype-phenotype mapping into an evolutionary model of genome evolution. The framework of the model is very general and can be used to explore several different mechanisms which might drive the processes behind the observed genome differences in the elements of the clade.

Rupert Mazzucco, International Institute for Applied Systems Analysis, Austria.

Virulence evolution in fragmented host populations with infectivity–mobility trade-offs

(joint work with Marieke Jesse[2], Ulf Dieckmann[1], and Johan A. J. Metz[1,3])

Abstract

Agents of infectious diseases evolve rapidly, which impedes control and eradication efforts. With fragmented host populations, the problem is compounded by agents surviving extinction in one patch and re-infecting other patches from there. Here, we study the evolution of infectivity with a simple SIS compartment model assuming a negative effect of increasing infectivity on host mobility, using the R_m fitness proxy as endemicity/invasibility indicator. We find that if mobilities increase quickly when infectivities decrease from their maximum, SIS-diseases generally become endemic in fragmented host populations for a wider range of conditions and at higher infectivities than if mobilities increase only slowly. Unless they are already close to extinction, diseases can generally sustain for a wide range of infectivities, while trade-off shapes determine the evolutionary outcome within that range. Evolutionary suicide never occurs: extinction results when the range of sustainable infectivities shrinks to 0. When diseases are close to extinction, evolutionarily stable infectivities are highest if mobilities increase only slowly with decreasing infectivities. When diseases are not close to extinction, trade-off shape is, however, often not as influential as maybe naively expected.

[1]Evolution & Ecology Program, International Institute for Applied Systems Analysis, Schlossplatz 1, 2361 Laxenburg, Austria

[2]Faculty of Veterinary Medicine, Utrecht University, the Netherlands

[3]Institute of Biology and Mathematical Institute, Leiden University, the Netherlands

Géza Meszéna, Institute of Biological Physics, Eötvös University, Hungary

Niche theory in ecology and evolution: A mathematical exercise, or help in biology?

Abstract

Development of evolutionary theory is seriously hindered by the controversial status of theory in ecology. While a significant amount of ecological theory has been developed, it is still very fragmented and subordinate to the essentially non-theoretical ecological thinking. I argue that deeply rooted illogical misconceptions are the main stumbling blocks for a consistent ecological theory. There are a long history, and current debates, of ideas about weakened (slowed down) competitive exclusion by several reasons other than niche segregation. If any of them would be correct then validity of natural selection were similarly iffy, a proposition nobody makes. I show that a mathematical niche theory of a sufficiently general level can clarify these controversies. If we understand that niche segregation (in a well-defined and sufficiently general sense) is the only generic way to avoid competitive exclusion, then it becomes parsimonious to assume that the main story behind a speciation event is adaptation to a new niche – a picture that is in line with biological common sense and current empirical findings.

Complementary nature of theory and empirical research is well established in physical sciences. It is clear that the two-way traffic between theory and reality, induction and deduction, should proceed in separate lanes: Induction and deduction have requires different mind-sets and methodologies. In contrast, the role of theory has remained controversial in ecology. While a significant amount of ecological theory has been developed, it is still very fragmented and subordinate to the non-theoretical ecological thinking, which has become very sophisticated in induction, but not in deduction. Lack of theoretical understanding of ecology, as a whole, is a significant handicap for evolutionary theory also – despite that fact that nobody questions the importance of theory for evolution.

Ville Mustonen, Wellcome Trust Sanger Institute, UK

Using time-resolved genetic data to monitor evolving populations

Abstract

Populations can evolve to adapt to external challenges. Of particular interest are adaptive processes in cancer or in bacterial, parasitic and viral infections, enabling the populations to escape from selective pressures exerted by drugs or the immune system.

To study the dynamics of such adaptation we experimentally evolved heterogeneous populations of budding yeast (*Saccharomyces cerevisiae*) under selective chemotherapy conditions. We followed the evolution of these populations over the course of 200 generations using whole-genome sequencing of the bulk population. The allele frequency patterns observed across time reveal different modes of adaptation: initially, selection acts on standing variation at segregating sites, later replaced by the emergence of macroscopic subclones. A set of *de novo* mutations within these subclones shows a validated resistant phenotype. The subclones also show genomic hallmarks of an active loss of heterozygosity dynamics that enables the resistance mutations to become homozygous. This is a collaboration project with the groups of Jonas Warringer and Gianni Liti.

The inference of the subclonal evolution from the data described above is underpinned by our probabilistic inference algorithm, cloneHD, which we use to learn the number of emerging subclones, their population fractions and their genotype posterior probabilities. A similar inference problem arises in the evolution of cancer where intra-tumour heterogeneity can underpin the emergence of resistance. Therefore, the ability to track subclonal dynamics and changes in clonal composition can inform therapy. We theoretically link the ability to monitor subclonal evolution to the development of novel therapy paradigms.

Robert Noble, Institut des Sciences de l'Évolution, University of Montpellier, France

Eco-evolutionary models of tumour heterogeneity

(joint work with Patrice Lassus[1], Urszula Hibner[1], Katarina Bacevic[1], Liliana Krasinska[1], Daniel Fisher[1] and Michael Hochberg[2])

Abstract

The interacting ecological and evolutionary processes that shape tumour progression and therapeutic outcomes remain poorly understood. I will show how simple mathematical models, based on empirical data, can be used to characterize processes such as competitive and cooperative cell interactions, and adaptation to changing, heterogeneous microenvironments. I will further show how methods of dynamical systems, game theory and cellular automata can inform forecasts of tumour development. My findings add to understanding of the dynamics of tumour heterogeneity and the emergence of drug resistance. The ultimate aim is to achieve a fundamental understanding of tumorogenesis, which may be exploited to improve cancer treatment and prevention.

[1]Institut de Génétique Moléculaire de Montpellier, France

[2]Institut des Sciences de l'Évolution, University of Montpellier, France

Lorenzo Pellis, University of Warwick, UK

Is HIV short-sighted? Insights from a multistrain nested model.

(joint work with Katrina Lythgoe[1] and Christophe Fraser[1])

Abstract

Within-host evolution can change the composition of pathogen genotypes within a host during the course of an infection, thus altering the availability of genotypes at the time of transmission and affecting the pathogen genotype distribution at the epidemiological level. We investigate the impact of short-sighted within-host evolution on the evolution of virulence of human immunodeficiency virus (HIV).

We develop a nested modelling approach that allows us to follow the evolution of pathogens at the epidemiological level by explicitly considering within-host evolutionary dynamics of multiple competing strains, their impact on between-host transmission events and the resulting epidemic dynamics.

We find that the topology of the within-host adaptive landscape strongly affects how virulence evolves at the epidemiological level. If viral reproduction rates increase significantly during the course of infection, the viral population will evolve a high level of virulence even though this will reduce the transmission potential of the virus (short-sighted evolution). However, if reproduction rates increase more modestly, as data suggest, our model predicts that HIV virulence will be only marginally higher than the level that maximizes the transmission potential of the virus.

This work provides a clear mathematical tool to investigate the contrasting evolutionary pressures a pathogen might be subjected to when comparing evolution at the within- versus between-host scale, and identifies or suggests possible reasons that can explain the currently observed HIV virulence patterns.

[1]Imperial College London, UK

Judith Perez-Velazquez, Helmholtz Zentrum München, Institute of Computational Biology, Germany

An age-structured model to analyze the evolutionary stability of bacterial quorum sensing

(joint work with Anne Mund[1], Christina Kuttler[1] and Burkhard A. Hense[2])

Abstract

Bacterial communication is enabled through the release and sensing of signalling molecules in a process called quorum sensing. This cooperative process can easily be destabilized by the appearance of cheaters, who contribute little or nothing at all to the production of common goods. This especially applies for planktonic cultures. In this work, we analyse the dynamics of bacterial quorum sensing and its evolutionary stability under two levels of cooperation, namely signal and enzyme production. The model includes mutation rates and switches between planktonic and biofilm state of growth. We present a mathematical approach to model these dynamics via age-structured models. We explore the conditions under which stability of cooperation is achievable and find that a compartmental structure in a part of the population can promote stability of cooperation in plankton. We furthermore identify parameters regions for maintenance of cooperativity.

[1]Technical University of Munich, Centre for Mathematical Sciences

[2]Helmholtz Zentrum München, Institute of Computational Biology

Michael J. Pocklington, Dept. of Genetics, University of Leicester, UK

The empirical genetic interaction map, the molecular ecosystem, and the nature of mathematical and computational abstraction

Abstract

The biochemical system of any organism can be represented graphically by a network of molecular types (nodes) and reactions (edges). Analogously, ecosystems consist of populations of organism types (nodes) and trophic interactions (edges). It is intuitively easy to represent both types of network approximately using ad-hoc process diagrams, but it is currently hopeless to predict their activity by mathematical or computational means for even the simplest systems. Instead, ways must be found for a principled description of biological networks which can then be translated into a useful generalised form. Systems biologist find the genetic interaction map useful for visualising the biochemical network of the organism, and this map can in principle be situated within a broader ecological context. The map arises out of mutations and environmental manipulations that make shared contributions to modular activities, recognisable by their phenotypes. Here I investigate the theoretical requirements needed to construct a comprehensive genetic interaction map for any organism, where the measured phenotype is overall fitness. A way is found to obtain the most general measurement of fitness. This is achieved by repeated competitive growth in a particular defined environment, a domain-independent heuristic device called the imaginary chemostat. Wide ranging implications follow, especially pertaining to niche structure and universality

Andrew Pomiankowski, Department of Genetics, University College London, UK

The evolution of larger sexual ornaments.

(joint work with Samuel Tazzyman[1] and Yoh Iwasa[2])

Abstract

Theoretical models predict that sexual selection should lead to reduction as often as exaggeration of sexual ornaments. Yet small sexual traits are very rarely seen. We analyze a simple quantitative genetic model of Fisher's runaway (the null model for sexual selection) and the handicap process. Our analysis shows that the imbalance cannot be obviously explained by larger ornaments being less costly than smaller ornaments, nor by asymmetry in the costs of preferences. Rather, the bias toward exaggeration can be best explained by signaling efficacy and/or the condition dependence of a trait increasing with size.

[1]Institute of Integrative Biology, ETH Zurich, Switzerland

[2]Department of Biology, Kyushu University, Japan

Christopher Quickfall, University of Sheffield, UK

Evolution of Maternally-Transmitted Symbionts

(joint work with William Hughes[1], James Marshall[2])

Abstract

Maternally transmitted symbionts, such as *Wolbachia*, have exhibited a multitude of methods of proliferating within insect populations, and indeed this includes mixed horizontal and vertical transmission. We seek to understand the general principles involved in mixed modes of transmission, with a general model of maternal symbiont transmission. In addition, we also wish to investigate how mixed modes of transmission may interact with parasitism, and the formation of mutualisms.

We consider a differential equation model of maternally-transmitted symbionts in an arbitrary host population. Novel elements of our model include a decomposition of virulence into components separately affecting horizontal and vertical transmission, and the inclusion of maternal transmission of symbionts. The purpose of decomposing virulence into two components is to model trade-offs between virulence and the different modes of transmission; in addition, we allow the components to be negative, in order to model fitness benefits conferred to hosts. We present results for a simple initial scenario, before extending the model to consider host control of sex ratios. In particular, we investigate which parameters, and host and symbiont strategies, allow for mutualism or parasitism, and mixed modes of symbiont transmission.

[1]University of Sussex, UK

[2]Kroto Research Institute, University of Sheffield, UK

Juan C. Ramírez, Department of Computer Science, University of Sheffield, UK

Self-deception Can Evolve Under Appropriate Costs

(joint work with James A. R. Marshall[1])

Abstract

Apparent biases in decision making by animals, including humans, seem to present an evolutionary puzzle, since one would expect decisions based on biased (unrealistic) information to be suboptimal. Although cognitive biases are hard to diagnose in real animals [3], we investigate Trivers' proposal that individuals should self-deceive first in order to better deceive others [4]. Although this proposal has been scrutinised extensively [1], it has not been formally modelled. We present the first model designed to investigate Trivers' proposal.

We introduce an extension to a recent model of the evolution of self-deception [2]. In the extended model individuals make decisions by taking directly into account the benefits and costs of each outcome and by choosing the course of action that can be estimated as the best with the information available. It is shown that in certain circumstances self-deceiving decision-makers are the most evolutionarily successful, even when there is no deception between these. In a further extension of this model individuals additionally exhibit deception biases and Trivers' premise (that effective deception is less physiologically costly with the aid of self-deception) is incorporated. It is shown that under Trivers' hypothesis natural selection favors individuals that self-deceive as they deceive others.

[1]Department of Computer Science, University of Sheffield, UK

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Daniel Ritterskamp, ICBM, University Oldenburg, Germany

Evolutionary Dynamics in Food Webs: Influence of Interaction Range, Resource Distribution and Space

(joint work with Bernd Blasius[1])

Abstract

Food webs encode feeding interactions in ecological communities, originating from an interplay of evolutionary and ecological processes. Here we develop an evolutionary food web model in which feeding interactions between species are related to the relative distance of their adaptive traits in niche space. We present three model variants, which are analyzed using numerical simulations in combination with adaptive theory. First, considering a single trait (bodysize), we uncover novel evolutionary dynamics, characterized by oscillations of bodysize within whole trophic guilds. Next, we study a system driven by two basal resources at different niche positions. For certain resource configurations this can lead to a dynamic instability of the food web, because the predators can not optimize their trait position towards both resources. This results into tight coupling of ecological and evolutionary dynamics, giving rise to biomass oscillations and intermittence in the population dynamic. Finally, we embed the model into a two dimensional niche space, where the additional niche axis might describe a spatial coordinate. The model is able to produce a wide range of static and dynamic food webs, depending on the width of the interaction kernel. The structure of these food webs has common features with empirical data (e.g. intervality) that other models lack to describe. We conclude that the interaction of ecological and evolutionary dynamics can give rise to complex behaviour, such as sustained oscillations, intermittency, and ongoing evolution. Combined with spatial considerations, our studies have the potential to predict evolutionary behaviour in real food webs.

[1]ICBM, University Oldenburg, Germany

Axel Rossberg, Centre for Environment, Fisheries & Aquaculture Science, UK

Are there species smaller than 1mm?

(joint work with Tim Rogers[1] and Alan J. McKane[2])

Abstract

Rapid advances in genetic sequencing technologies have fuelled the vision that these data would allow the identification and counting of species, represented as tight clusters of similar genomes separated by a "barcoding gap". Theoretical considerations, however, suggest that, for populations occurring in large numbers, as most small organisms do, such a sharp distinction and enumeration of species will not be possible. Surprisingly, empirical data used to quantify meiofaunal species richness do indeed become ambivalent at best once the empirical support for the paradigm that distinct species do indeed form becomes part of the research question. We conclude that, as things stand, the burden of proof is on showing that meiofaunal species do form. Meanwhile, exciting new approaches to ecology and evolution that do not rely on the notion of species are beginning to take shape.

[1]Department of Mathematical Sciences, University of Bath, Claverton Down, Bath, UK

[2]Theoretical Physics Division, School of Physics and Astronomy, University of Manchester, UK

Nadav Shnerb, Physics Department, Bar-Ilan University, Israel

Emergence of structured communities through evolutionary dynamics.

Abstract

Species-rich communities, in which many competing species coexist in a single trophic level, are quite frequent in nature, but pose a formidable theoretical challenge. In particular, it is known that complex competitive systems become unstable and unfeasible when the number of species is large. Recently, many studies have attributed the stability of natural communities to the structure of the interspecific interaction network, yet the nature of such structures and the mechanisms behind them remain open questions. We introduce an evolutionary model, based on the generic Lotka-Volterra competitive framework, from which a stable, structured and diverse community emerges spontaneously.

Michael Sieber, University of Potsdam, Germany

Beyond trade-offs: how life cycle complexity limits parasite host ranges

Abstract

Parasite host ranges are a dynamic ecological trait and there is ample evidence for the potential of rapid host shifts. Nevertheless most parasites show a high degree of host specificity. Host-use trade-offs are a common theoretical explanation for the prevalence of host specialism, but empirical evidence for such host-use trade-offs is rare. I will present an alternative model for limited host-ranges which is based solely on known basic features of the parasitic life cycle, namely host selection and subsequent intra-host replication.

This theory provides an explanation for the observed host specificity even when there is no apparent cost for extended host ranges.

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Suzanne Sindi, University of California, Merced, USA

A Mathematical Test for Selection in Word Frequencies.

(joint work with Rick Dale [1])

Abstract

In biological evolution traits may rise and fall in frequencies due to either genetic drift, where variant frequencies change by chance, or selection where advantageous variants will rise in frequency. The neutral model of evolution, first developed by Kimura in the 1960s, has become the standard against which selection is detected. While the balance between these two important forces - drift and selection - has been well established biology there are other domains where these beliefs are still coming together.

The dominant account of cultural change has been the neutral theory. This powerful representation has been applied to a myriad of disparate phenomena from pre-history pottery to modern baby names. Intriguingly, there is accumulating evidence regarding cultural processes that the neutral theory cannot account for all features of the data. As such, there has been a renewed interest in determining whether there is selection amongst the drift.

Detecting selection in biological data sets is a comparatively well-studied problem. Adapting selective tests to cultural evolution has been complicated by significant differences between cultural and biological evolution. There are not well-defined analogues of “alleles” - heritable elements which selection is thought to operate on - and “inheritance” - the transmission of cultural units among individuals. Thus, it is not possible to directly impose evolutionary models from the biological context to cultural data. However, cultural data also presents many opportunities. Most notably, cultural archives may offer more complete samplings of historical data than is possible for many biological data sets.

Inspired by the Moran and Wright-Fisher models in population genetics, we developed a neutral model of word frequency variation to assess when linguistic data appears departs from neutral evolution. As such, our model represents a possible “test for selection” in the linguistic domain. While alternative approaches have focused on specific subclasses of vocabulary - such as domain specific terms or baby names. We focus our attention on a set commonly used words and explore how their frequencies have changed in time. Specifically, we explore how the distribution of word use has changed for sets of words in American English over 100 years (1901-2009) as expressed in vocabulary usage in published books, made available by Google Ngram. When comparing empirical word frequency changes to our neutral model we find pervasive and systematic departures from neutrality.

[1] Cognitive and Information Sciences, University of California - Merced, Merced, USA

Yoav Soen, Depart.of Biological Chemistry, Weizmann Institute of Science, Israel

Bridging Ecology and Evolution by Symbiosis and Epigenesis

Abstract

While adaptations to novel environments extend over evolutionary timescales, a new environment can emerge already within a single generation and can immediately impact the physiological and epigenetic state of the organism. Whether and how the initial response might be connected to longer-term establishment of new adaptations are not clear. We address these questions experimentally by studying how flies cope with novel scenarios of stress. We identified epigenetic- and symbiotic-mediated mechanisms which promote increased developmental plasticity under stress, influence the germline, and contribute to non-Mendelian transfer of variation across generations. I will discuss these epigenetic- and symbiotic-mediated processes and their potential contribution to the establishment of initial adaptations that can bridge part of the gap between ecological and evolutionary processes.

Andrea Sottoriva, the Institute of Cancer Research, UK

Neutral evolution and star-like phylogenies in next-generation sequencing data

Abstract

Despite extraordinary efforts to profile cancer genomes on a large scale, interpreting the vast amount of genomic data in light of cancer evolution and in a clinically relevant manner remains challenging. This is complicated by inter- patient variation and extensive intra-tumor heterogeneity. In particular, the relationship between the cancer genotype and phenotype remains largely unknown as phenotypical characteristics are hard to measure. A critical issue is the lack of a theoretical framework of reference able to make predictions on existing data. Using a multi-sampling strategy we have recently shown how colorectal cancers grow as a single "Big Bang" expansion populated by many intermixed sub-clones that are not subject to stringent selection. Here we demonstrate that this signature of *effectively-neutral* evolution can be detected in next-generation sequencing data from bulk samples as well, as a result of the star-like tumor phylogenies predicted by the Big Bang growth. In particular, we present a simple mathematical model of cancer expansion based on neutral evolutionary dynamics that predicts the spectrum of alterations reported by next- generation sequencing in a large proportion of cases. This hidden feature of cancer evolution is common to multiple tumor types and can be detected in different independent cohorts. Importantly, this allows the direct measurement in each individual patient of the *in vivo* mutation rate per division and the timing of mutations using currently available sequencing data. This result provides a new way to interpret the wealth of cancer genomic data available to date, shedding new light on the relationship between the cancer genotype and phenotype.

Max Souza, Departamento de Matemática Aplicada, UFF, Brazil

Fixation in large populations: a continuous view of a discrete problem.

Abstract

We study fixation in large, but finite populations with two types, and dynamics governed by birth-death processes. By considering a restricted class of such processes, which includes most classical evolutionary processes, we derive a continuous approximation for the probability of fixation that is valid beyond the weak-selection (WS) limit. Indeed, in the derivation three regimes naturally appear: selection-driven, balanced, and quasineutral the latter two require WS, while the former can appear with or without WS. The continuous approximations then yield asymptotic approximations for evolutions in the selection-driven regime. An interesting point is that the scalings of such regime do not preclude a weak-selection regime. As an application, we show that the fixation pattern for the Hawk and Dove game satisfies what we term the one-half law: if the Evolutionary Stable Strategy (ESS) is outside a small interval around 1/2, the fixation is of dominance type. We also show that outside of the weak-selection regime the dynamics of large populations can have very little resemblance to the infinite population case. In addition, we also show results for the case of two equilibria. Finally, we present a continuous restatement of the definition of an ESSN strategy, that is valid for large populations. In the quasi-netrual regime, we have a further asymptotic approximation that recovers the one-third law under linear fitness and, as a generalisation, we introduce the concept of critical-frequency.

Andrzej Swierniak, Silesian University of Technology, Gliwice, Poland

Mixed Spatial Evolutionary Games in modeling cancer cell interactions

Abstract

Spatial tools were first used in modeling of carcinogenesis in [1]. The line of reasoning presented there has been the starting point for our analysis, as the most suited to the applications focused on in our investigations. The spatial evolutionary games (SEGT models) are played iteratively on a lattice forming torus and each tie in a competition is solved randomly. The following steps are performed every iteration [1]: payoff updating - the sum of local fitness in the neighborhood, removing players - cell mortality, reproduction - defining which phenotype will be on an empty place. In the case of application of SEGT to analyze cancer cells behavior, the question that arises is whether each cell has only one strategy (represents one phenotype) or rather it should be treated as containing different strategies. The new idea which we propose in modeling spatial effects associated with evolution of cancer cells is related to their heterogeneity. It leads to the conclusion that cancer cells should be considered as representing different phenotypes at the same time, described by frequency of occurrences. The spatial games resulting from this assumption will be called mixed spatial evolutionary games (MSEG).

Modification of the way spatial games are used requires the change in definition of the local fitness (adaptation). It is defined in a way similar to an expected result of the game with mixed-type strategies. The result given by each pair of strategies is multiplied by their frequency of occurrence. Hence, the analysis is more complex and difficult, due to an increased number of feasible spatial structures. Nevertheless, for simplification, both types of spatial games may be represented in a way similar to the mean-field models. The new formulation of spatial games also defines mortality of the cells in a different way. Here, chosen player stays alive and either its phenotypes ratio is changed or it affects cells in the neighborhood. Additionally to two basic reproductions (deterministic and probabilistic) at least three additional could be added for the mixed spatial games: weighted mean of the strongest players - the weighted mean accordingly to players payoffs is taken, weighted mean of the best clusters - players are organized into clusters and the weighted mean is calculated for players in the strongest cluster, spreading reproduction - mentioned previously possibility to impact surrounding cells. In our study we compare simulation results for different models based on spatial evolutionary games describing interaction of cancer cells representing four phenotypes [2].

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Nobuto Takeuchi, Department of Basic Science, University of Tokyo, Japan

Spontaneous symmetry breaking in complementary replication as a consequence of multilevel selection in a minimal model of protocells.

(joint work Paulien Hogeweg[1], Kunihiko Kaneko[2])

Abstract

Complementary replication provides the molecular basis of heredity in all life forms, accomplishing the fundamental prerequisite for evolution, probably since the emergence of the first, primitive replicating systems. However, complementary replication is not strictly necessary for evolution, as attested by numerous models of evolution that abstract away from complementarity (e.g., population genetics). Here, we use individual-based mathematical modeling to investigate the consequences of complementarity to the evolution of prebiotic replicating molecules enclosed in protocells. In the model, molecules can serve both as catalysts and as templates for replication. A catalyst and template first form a complex and subsequently dissociate, synthesizing the complementary strand of the template. This time lag in replication results in a trade-off between molecules spending time as templates and as catalysts. Consequently, the catalytic activity of molecules is driven toward evolutionary deterioration by selection between molecules within protocells. This tendency, however, is counteracted by selection between protocells, whose growth and division require internal molecules to replicate. This conflict between the two levels of selection exacerbates as the average number of molecules within protocells increases (the key parameter of the model). This exacerbation of the conflict leads to spontaneous symmetry breaking, whereby an initially symmetric replication cycle, where the two complementary strands of molecules perform an exactly identical role, evolves into an asymmetric system, where one strand becomes the minority and functions only as templates (like genomes), whereas the other strand becomes the majority and serves the dual function of templates and catalysts, performing both 'informational' and 'executive' roles. This compositional and functional asymmetry between the complementary strands enables protocells to survive under wider conditions than possible without complementarity. These results indicate that complementary replication and spontaneous symmetry breaking could provide greater stability to the earliest stages of life's evolution.

[1]Utrecht University, Theoretical Biology and Bioinformatics Group, the Netherlands.

[2]University of Tokyo, Department of Basic Science, Japan.

Jaspreet Toor, School of Mathematics and Statistics, University of Sheffield, UK

The evolution of host resistance to disease in the presence of predators

(joint work with Alex Best[1])

Abstract

Although host-parasite models have been widely studied, the inclusion of predators has often been overlooked until recently. In this study, we examine a host-parasite model with a predator and show that the predator changes the evolutionary behaviour of the host. We find that the hosts maximize their levels of resistance at intermediate predation rates, where the risk and cost of infection are both high due to the presence of infected hosts and predators. We show that the branching possibilities increase with the predation rate for regions where the susceptible and infected hosts coexist with the predator. Hence, the results reveal that the addition of a predator has a considerable impact on host-parasite populations.

[1] School of Mathematics and Statistics, University of Sheffield, UK

Tat Dat Tran, Max Planck Institute for Mathematics in the Sciences, Leipzig, Germany

The free energy method for the Wright-Fisher model

(joint work with Julian Hofrichter[1] and Jürgen Jost[1])

A short abstract

We develop here a new systematical approach about using free energy methods to consider the Wright-Fisher model of population genetics.

[1]Max Planck Institute for Mathematics in the Sciences, Leipzig, Germany

Minus van Baalen, CNRS/IHES/ENS, Paris, France

Adaptation, conflicting information and stress

Abstract

Information plays a critically important role in ecology and evolution. Very often biological information users have to assess the fitness values of different simultaneous sources of information. If these are concordant, there is no problem, but if different sources of information are in conflict, there is a problem. Here I will illustrate how conflicting information may lead to a form of stress, and suggest that such stress may have important consequences for our understanding of the role of information in evolution.

Daniel van der Post, School of Biology, University of St Andrews, UK

Learning mechanisms modulate the evolutionary trade-off between social learning and exploration

(joint work with Mathias Franz[1] and Kevin N. Laland[2])

Abstract

Social learning is of interest as a behavioral adaptation and as a prerequisite for cultural inheritance. To explain the evolution of social learning, evolutionary theory focuses on the contrast between social and asocial learning and a trade-off between them. It is assumed that only asocial learning can generate the information needed for adapting to environmental change. Social learning is therefore parasitic and dependent on asocial learning. Such theory makes use of the "phenotypic gambit", the idea that the evolution of behavior can be studied on a functional level without specifying underlying behavioral mechanisms, enabling general predictions to be formulated. In contrast, we study evolutionary simulations of group foragers, where psychological learning mechanisms and grouping processes are explicitly implemented. In this model, where foragers learn "what" and "how" to eat, we find that the existence and nature of the trade-off between social and asocial learning depends strongly on the social learning mechanism that is considered.

In our model there are three kinds of social learning mechanisms. The first is group-level social learning, which arises spontaneously when foragers live in groups and learn by trial and error. In groups with only this kind of social learning, evolution does not cause exploration rates to decline relative to those in solitary foragers and there is no trade-off. The second mechanism is stimulus enhancement, which makes individuals more likely to choose an observed behavior. Since this process affects decision-making it can lead to social contagion. As a result, exploration (including associated costs) can be amplified in groups. Thus an evolutionary trade-off arises which causes exploration rates to decline. Interestingly, this is not the same trade-off as in most evolutionary theory. The third mechanism is social-skill-learning that allows individuals gain experience about processing resources by observing others. Here decisions are not affected directly, and there is no social contagion. As a result, when social-skill-learning evolves, there is no trade-off with exploration. Instead, exploration rates can even increase. When both stimulus enhancement and social-skill-learning evolve, social contagion due to stimulus enhancement dominates, causing exploration rates to decrease.

The overall implications of our results are: (i) when mechanistic detail is taken into account, the trade-offs between social learning and exploration can change or disappear, and as a consequence generalizations regarding this trade-off are not straightforward; (ii) the basic assumptions used in theory on the evolution of social learning do not hold up well in detailed simulation.

[1]Department of Biology, Duke University, USA
[2]School of Biology, University of St Andrews, UK

Matthijs Van Veelen, University of Amsterdam, CREED, the Netherlands

Inclusive fitness and group selection: the regression method vs. the counterfactual method

Abstract

A widespread claim in evolutionary theory is that every group selection model can be recast in terms of inclusive fitness. Whether or not that claim is true, depends on how costs and benefits are defined. When they are defined with the regression method, that claim is true. When they are defined by comparing fitnesses to what they would have been, had a player played defect instead of cooperate, this is not the case. I will discuss problems with the regression method, and illustrate how the counterfactual method results in two types of problems for a very simple group selection model.

Antje Vollrath, Institute of Computational Mathematics, Braunschweig, Germany

A framework for multi-gene-loci inheritance in resistance modelling

Abstract

We consider a time-continuous, polygenic model by means of which different scenarios of emerging resistance can be simulated and analysed in a systematic manner for organisms of different ploidies and for any number of different alleles. By means of a new approach based on tensor products of heredity matrices it is possible to not only analyse target-site resistance but also metabolic resistance, i.e., mechanisms were resistance is encoded as a property of multiple gene loci.

We will analyse how the genetic basis of fitness affects the ability of a population to adapt to changing environmental conditions. Under the assumption that the population would go extinct in the new environment without evolution, we discuss the concept of evolutionary rescue that occurs when a few preexisting adapted genotypes reproduce at a rate sufficiently large to avoid population extinction. This adaptation process can be either wanted or unwanted for various reasons. As an unwanted consequence of the genetic adaptation of plants we look at herbicide resistance due to the strong selection pressure following the application of a harmful substance. Evolution is acting on the parameters of dose-response curves, i.e., on the mortality rates and thus on the ED50-value. The resulting system of differential equations is analysed with respect to polymorphic equilibria, conditions for evolutionary rescue and the influence of resistance as property of a single genotype compared to resistance as property of a single allele.

The different scenarios of resistance development and evolutionary rescue are demonstrated in numerical experiments.

Andrew Whalen, School of Biology, University of St Andrews, UK

The Learning of Sequences of Actions through Low Fidelity Social Transmission

(joint work with Daniel Cownden[1], Kevin Laland[1])

Abstract

Recent theoretical models have examined the evolution of social learning. These models focus on the evolutionary and population level out comes of a learning processes, and abstract away from specific learning mechanisms. As a result such models are difficult to compare with animal behavioral data. To address this issue, we present an individual based learning formalism which integrates social information into Temporal Difference learning within a Markov Decision Process, and analyze how individuals learn sequences of actions. The goal of this formalism is to provide a setting where the merits of social learning can be tested theoretically, while maintaining the ability to empirically verify these results We demonstrate the usefulness of this formalism by exploring how animals might learn sequences of actions, a difficult problem due to the large number of possible sequences of actions given even a small repertoire of behaviors. We take the learning of nettle processing by mountain gorillas as a focal example, and estimate the value of social learning in this situation. Through simulations, we find that limited, low-fidelity social learning can combine with individual learning to facilitate the acquisition of complex sequences. We then investigate the types of learning problems for which social learning will be particularly beneficial. We find that social learning becomes increasingly helpful as the number of availble base actions grows, and as the length of high reward action sequences increases. Our analysis shows that a simple form of social learning dramatically enhances the rate at which sequences of actions are learned. This work calls attention to a widely applicable model of learning, Temporal Difference learning, and demonstrates how it can be readily extended to examine the value of social information in specific learning problems. Temporal Difference learning can provide individual level behavioural prediction on a fine temporal scale, and so can be directly verified with animal experiments. This work creates a much needed link between theory and empiricism in the study of animal social learning.

[1]School of Biology, University of St Andrews, UK.

Ke Yuan, CRUK Cambridge Institute, University of Cambridge, UK

Reconstructing intra-tumor phylogenies with Bayesian nonparametric models.

(joint work with Thomas Sakoparnig[2,3,4], Florian Markowetz[1] and Niko Beerenwinkel[2,3])

Abstract

Cancer has long been understood as a somatic evolutionary process, but many details of tumor progression remain elusive. Here, we present BitPhylogeny, a probabilistic framework to reconstruct intra-tumor evolutionary pathways. Using a full Bayesian approach, we jointly estimate the number and composition of clones in the sample as well as the most likely tree connecting them. We validate our approach in the controlled setting of a simulation study and compare it against several competing methods. In two case studies, we demonstrate how BitPhylogeny reconstructs tumor phylogenies from methylation patterns in colon cancer and from single-cell exomes in myeloproliferative neoplasm.

[1] Cancer Research UK Cambridge Institute, University of Cambridge, UK

[2] Department of Biosystems Science and Engineering, ETH Zurich, Basel, Switzerland.

[3] SIB Swiss Institute of Bioinformatics, Basel, Switzerland.

[4] Biozentrum, University of Basel, Basel, Switzerland.

Benjamin Werner, the Institute of Cancer Research, London, UK

Reconstructing the in vivo dynamics of hematopoietic stem cells from telomere length distributions

Abstract

Most mammalian tissues are hierarchically organized. Few self renewing stem cells give rise to shorter-lived progeny. Stem cells undergo both symmetric and asymmetric cell divisions with age specific proportions to ensure different needs of development, wound healing and homeostasis. Miss-regulation of these proportions potentially manifests in cancer.

Here, we investigate the in vivo patterns of symmetric and asymmetric stem cell divisions in the human hematopoietic system throughout life. We analyze the expected shape of telomere length distributions underlying stem cell behavior within individuals. These distributions contain a fingerprint of the initial telomere length and progressive telomere loss relevant for individualized disease prognosis.

We test our predictions on telomere length data of 356 healthy individuals, including 47 cord blood and 28 bone marrow samples. We find an increasing stem cell pool by rare symmetric stem cell self renewals during childhood and adolescence and an approximately maintained stem cell population in adults. Furthermore, our method is able to detect individual fluctuations of stem cell dynamics from a single tissue sample, i.e. a single snapshot. Prospectively, this allows us to compare cell proliferation between individuals and identify abnormal stem cell dynamics, which affects the risk of stem cell related diseases

Posters

(in alphabetic order)

Matthew Adamson (University of Leicester, UK). *Structural sensitivity in biological models revisited.*

Jack Aidley (University of Leicester, UK). *Modelling the behaviour of hypermutable regions in populations of Campylobacter jejuni under selective and non-selective conditions*

Paul Calcraft (University of Sussex) *Species Selection in the Solanaceae: Integrating Speciation and Extinction with Individual Competition*

Valentina Clamer (University of Trento, Itlay). *Dynamics of Host-Parasitoid Interactions and Coexistence of Different Hosts.*

Assaf Engel (Bar-Ilan University, Soreq NRC, Israel). *What bowl and doily spiders have to say about Zero Determinant strategies?*

Frédéric Fabre (INRA, Bordeaux, France). *Joint estimation of the strength of genetic drift and selection from Next Generation Sequencing time-sampled data: a case study on the adaptation of virus populations to host plant resistance.*

Lucas Dias Fernandes (University of Aberdeen, UK). *Interplay between selection and gene flow on coevolutionary dynamics on large spatial lattices*

James Ounsley (University of St. Andrews, UK). *Whom should I copy? The value of learning from the young in a complex and variable environment.*

Ferdinand Pfab (University of Trento, Italy). *Multiplicity of coexistence equilibria in a 2-parasitoids 1-host model.*

Giacomo Plazzotta (Imperial College London, UK). *Phylogenetic trees: cherries and basic reproduction number.*

Nomenjanahary Alexia, Raharinirina (Leibniz Center for Tropical Marine Ecology (ZMT) & Jacobs University, Bremen, Germany). *A simple trait-based model for describing the adaptive dynamics of symbiosis*

Carlos Reding (University of Exeter, UK). *Non-monotone antimicrobial response and emergence of resistance during antimicrobial treatments.*

Edith Ross (University of Cambridge, UK). *Inferring clonal evolution of tumours from single-cell sequencing data.*

Rafik Salama (University of Oxford, UK). *Somatic evolution of renal cancer presents an evidence for selective transcriptional pressure.*

Benjamin Schuster-Boeckler (University of Oxford, UK). *Biased random sampling: the effect of epigenetic marks on somatic genome evolution.*

Mircea T. Sofonea (University of Montpellier, France). *Epidemiological pleiotropy of within-host interactions.*

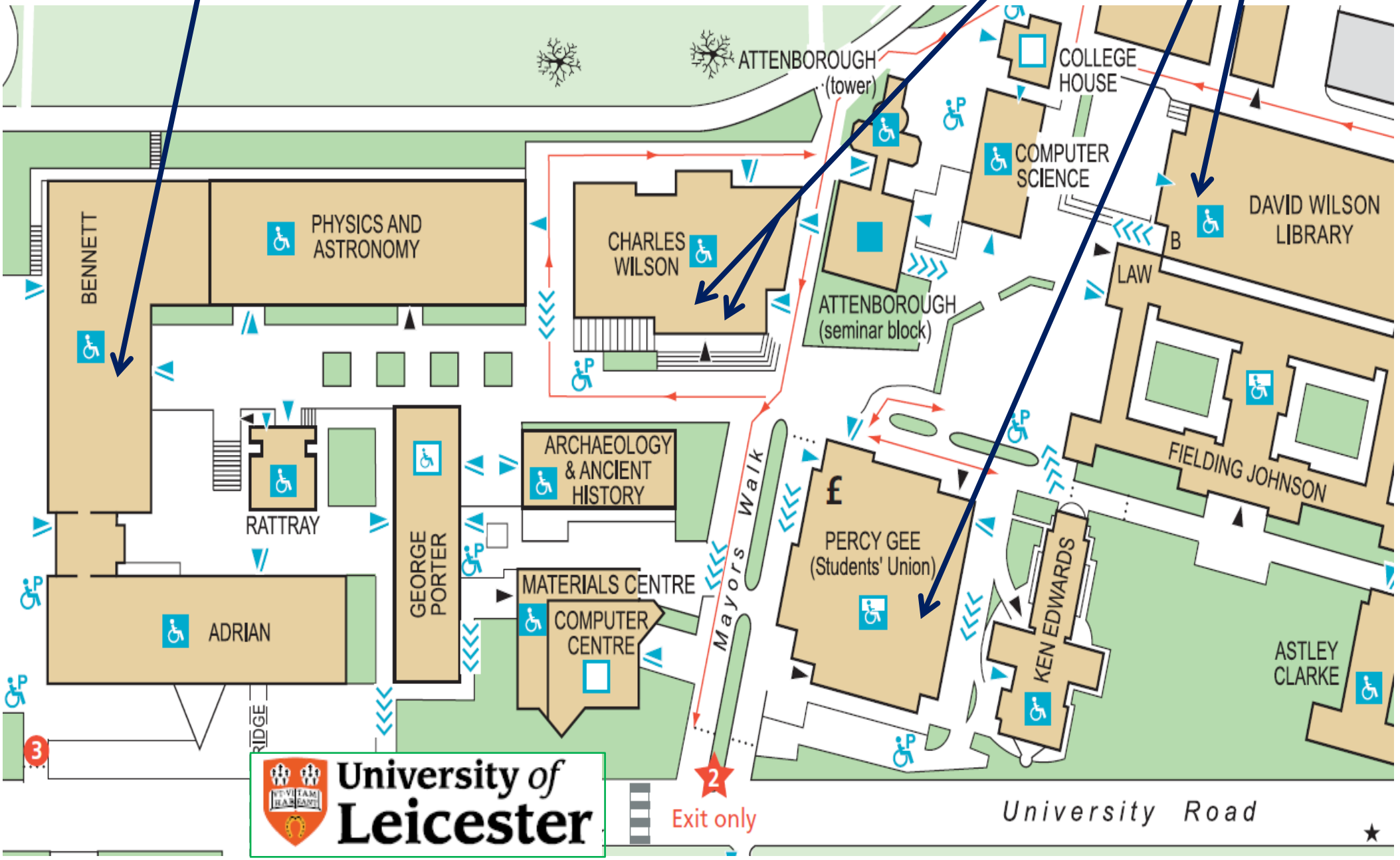
Dov Stekel (University of Nottingham, UK). *Mathematical model for spread of antibiotic resistance in a dairy unit in the UK: the importance of horizontal gene transfer*

Megan Sørensen (University of York, UK). *Modelling the metabolic exchange in a novel endosymbiosis*

Main Campus Map

All talks are here! (Bennett)

Lunch suggestions!!!



Recommended places **to eat and drink** close to the conference site

Suggestions for lunch (on Campus)

Charles Wilson Building, ground floor: cafeteria Piazza (recommended) and the student canteen.

Charles Wilson Building, first floor: academic staff canteen (recommended)

Percy Gee Students' Union, Nineteen Twenty Three restaurant (recommended) and other cafeterias in the same building

The Library Building: cafeteria

Suggestions for dinner (off Campus)

The Royal Dine (Indian food): the corner of London Road and De Montfort Street

Kayal (South Indian Restaurant: it is highly recommended!): in the beginning of Granby Street, near the train Station

La Tosca (Italian Restaurant), London Road, close to the Victoria Park (almost at the corner of London Road and Granville Road)

There is a really huge number and variety of restaurants and pubs in the city centre (and along London Road). You are encouraged to explore them by yourself!