Rapid adaptation to novel environments

Abstract Traditional models both in population genetics and ecology assume that evolutionary and ecological processes happen at different time scales and can consequently be studied separately. However, genetic adaptation to novel environmental conditions can be rapid and accompanied by demographic change. Individuals might encounter a new unfavorable environment either in situ when the habitat itself deteriorates or when they invade a new habitat. In the first case, the environmental change, if severe, will drive the whole (possibly large) population extinct unless the population succeeds in adapting to the new environment ("evolutionary rescue"). In the second case, a small founder population enters a habitat to which it is maladapted. Rapid adaptation is necessary in order to establish a permanent population. Understanding which factors impact the survival/establishment probability of a group of individuals is important when we seek to maintain a population, but also when we seek to eliminate it. The latter aspect is of particular relevance in a medical context where we aim to inhibit the evolution of drug resistant pathogens or a pathogen host shift.

Thursday, June 19

9:00–9:30:	Olivier Cotto
9:30–10:00:	Helen Alexander
10:00–10:30:	Coffee break
10:30–11:00:	Matthew Hartfield
11:00–11:30:	Troy Day
11:30–11:40:	Break
11:40–12:10:	Yoav Soen

Evolution of senescence in heterogeneous landscapes

Olivier Cotto
University of Zurich, Institute of Evolutionary Biology and Environmental Studies, Winterthurerstrasse 190, CH-8057 Zurich, Switzerland
Email: olivier.cotto@ieu.uzh.ch

The current theory of senescence is developed in a very simple ecological and demographic context, with a unique population at equilibrium in a homogeneous habitat. In the wild, species live in a variable environment in space and time, where the assumption of equilibrium is often transgressed. In this study, we use models of quantitative genetics in structured populations in order to investigate the evolution of senescence in a variable environment. Adaptation to local environment depends on phenotypic traits which expression varies with age. We study different scenarios where the environment changes abruptly, gradually or cyclically with time and where the environment is heterogeneous in space with different populations connected by migration. The strength of selection decreases with age, which predicts slower adaptation of traits expressed late in the life cycle, potentially generating stronger senescence in habitats where selection changes in space or in time. This prediction is however complicated by the fact that the genetic variance also increases with age. With numerical calculations, we found that in most cases the rate of senescence is enhanced when the environment varies. Especially, migration between different habitats is a durable source of senescence in heterogeneous landscapes. We also show that the rate of senescence can sometime decrease transiently, when the population is not at equilibrium, with possible implications in experimental evolution and in the study of invasive species. Our results highlight the need to study age-specific adaptation, as a changing environment can impact differently each age-class with different consequences on demography.

Evolutionary rescue of a pathogen: emergence of viral drug resistance

Helen Alexander, Guillaume Martin, Sebastian Bonhoeffer
Institute for Integrative Biology, ETH Zürich, CHN H.74,
Universitätsstrasse 16, Zürich, CH-8092, Switzerland
Email: helen.alexander@env.ethz.ch

"Evolutionary rescue" is a term used primarily in conservation biology to describe the situation in which genetic adaptation prevents extinction of a declining population. This scenario arises, for example, in macro-organisms facing habitat destruction or climate change. It also arises in micro-organisms and cell populations, including pathogens or cancerous tumors in a patient taking drug therapy. The emergence of drug resistance, which can be seen as evolutionary rescue of a pathogen population facing severe environmental change, is a major problem in public health, compromising successful treatment of infections. Here I will present new theoretical results on the within-host emergence of drug resistance in viruses. We are particularly interested in whether rescue occurs from pre-existing (standing) genetic variation, or de novo mutation during therapy. I will describe an extension of a widely-used viral dynamics model, which allows us to investigate the roles of viral "life history", competition between strains, and stochasticity. Analytical approximations for the probability of rescue can be derived from a simplified stochastic process description, allowing identification of the parameters playing a key role in determining rescue. Finally, I will discuss some links between the largely separate drug resistance and evolutionary rescue literature and the potential for greater integration between these fields.

Epidemiological feedbacks affect evolutionary emergence of pathogens

Matthew Hartfield and Samuel Alizon
Laboratoire MIVEGEC (UMR CNRS 5290, IRD 224, UM1, UM2), 911 avenue Agropolis, B.P. 64501, 34394 Montpellier Cedex 5, France.
Email: matthew.hartfield@ird.fr

The evolutionary emergence of new pathogens via mutation poses a considerable risk to human and animal populations. Here, I present work from two models investigating the emergence of new strains in non-homogeneous populations.

At the epidemiological level, previous studies have investigated cases where a potentially pandemic strain emerges from a maladapted strain (that is, its basic reproductive ratio $R_0 < 1$). However, an alternative cause of pathogen emergence is where a weakly-adapted strain (with $R_0 \approx 1$) mutates into a strongly-adapted strain (with $R_0 \gg 1$). In this case, a proportion of the susceptible population is removed as the first strain spreads, but the impact this feedback has on mutated strain emergence has yet to be quantified. We produce a model that takes into account changes in the susceptible population over time, and find that the ongoing depletion of susceptible individuals by the first strain has a drastic effect on the emergence probability of the mutated strain, above that assumed by just scaling the reproductive ratio. We subsequently apply our model to the case of Chikungunya virus on La Réunion island, and demonstrate that the emergence probability of the mutated strain was reduced approximately 10-fold, compared to models assuming that susceptible depletion would not affect outbreak probability.

We next show how these models can be extended to emergence at the within-host level, where a pathogen needs to escape immune-cell proliferation. Using this extended model, we show that contrary to previous results, it is more beneficial for a pathogen to increase its growth rate, rather than tolerating immunity.

These results highlight the importance of taking population feedbacks into account when predicting disease emergence.

Insights into the evolution of drug resistance from simple models

Troy Day
Department of Mathematics and Statistics & Department of Biology,
Queen's University, Kingston, Ontario, Canada
Email: tday@mast.queensu.ca

The evolution of drug resistance is one of the most serious public health problems in modern medicine. In this talk I will present two insights into the problem that are obtained from relatively simple mathematical models involving a separation of ecological and evolutionary timescales: (1) contrary to current orthodoxy, a lighter drug dosage can be most effective for controlling resistance evolution; and (2) cofactors aimed an manipulating within-host ecological interactions can prevent the evolution of resistance. I will also present preliminary data from an experimental test of the latter idea.

Epigenesis and Symbiosis – A missing link in adaptation to new environments?

Yoav Soen
Department of Biological Chemistry, Weizmann Institute of Science,
Rehovot 76100, Israel
Email: yoavs@weizmann.ac.il

The development of organisms must be robust enough to maintain adaptive patterns and flexible enough to enable coping with fluctuating environmental, epigenetic and symbiotic conditions. How this flexibility is achieved and whether and how it is connected to longer-term establishment of new adaptations are not clear.

We are addressing these questions by studying stress-induced induction and inheritance of altered developmental patterns in flies. We identified epigenetic and symbiotic-mediated mechanisms which promote increased developmental flexibility under stress and contribute to non-Mendelian transfer of influences across generations.

I will present these mechanisms and discuss the potential of epigenesis and symbiosis to bridge between ecological and evolutionary processes.