Dynamics of Biological Systems
Dynamique des systèmes biologiques
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MO'TASSEM AL-ARYDAH, Khalifa University
Applications of Mathematical Modelling in Managing the Spread of Chronic Wasting Disease (CWD) in Wild Deer

We describe the application of a recently developed mathematical model for predicting the spread of chronic wasting disease (CWD) in wild deer under different scenarios where harvesting is employed in disease management. A process-based mathematical model for CWD transmission in wild deer populations was recently developed and parameterized by Al-arydah et al. (2012) to provide a scientific basis for understanding the factors that affect the spread of CWD and to evaluate concomitant disease control strategies. The impact of gender on CWD transmission was shown to have a significant influence on the spread of the disease in the wild. Our model shows a range of harvesting rates in which CWD is controlled and the deer population survives. However, if harvesting rates are too low, the disease remains endemic for decades whereas the Canadian deer population becomes extirpated if harvesting rates are excessive. Future work includes building the model to assess the spread of CWD under different disease management scenarios.

SARDER MOHAMMED ASADUZZAMAN, University of Victoria
The coexistence or replacement of two subtypes of influenza

A pandemic subtype of influenza A sometimes replaces (e.g., in 1918, 1957, 1968) the previous seasonal subtype. However, the reintroduced subtype H1N1 in 1977 has been co-circulating with H3N2 since then. To understand these alternatives, we formulate a hybrid model for the dynamics of influenza A epidemics. Our model takes into account the cross-immunity between seasonal strains and the cross-immunity between seasonal and pandemic subtypes. A combination of theoretical and numerical analyses shows that for very strong cross-immunity between seasonal and pandemic subtypes, the pandemic cannot invade, whereas for strong and weak cross-immunity there is coexistence, and for intermediate levels of cross-immunity the pandemic may replace the seasonal subtype.

MARIE AUGER-MÉTHÉ, Dalhousie University
Tackling the challenges of fitting movement models to marine data

Understanding animal behavior by applying statistical models to movement data is becoming increasingly common in ecology. While applying movement models can be challenging in terrestrial systems, fitting such models to marine data is hindered by many formidable challenges. First marine movement data are often associated with large amounts of error because accurate positioning systems (e.g., GPS) are not well suited to the marine environment. Models therefore often include additional parameters to account for the effect of measurement error. Second, environmental data are often spatiotemporally coarser than movement data, requiring models that accommodate for discrepancies between these scales. Third, unlike terrestrial systems, the marine habitat has 3 dimensions (latitude, longitude, and depth). Fourth, the ocean is not static. Factors, such as tides and winds, create advection and turbulence. Thus models need to disentangle ocean drift from the voluntary movement of animals, as well as incorporate how animals react to changes in the strength and direction of currents. While all of these aspects are essential for our understanding of the behavior of marine animals, they result in highly complex models that are difficult to fit to data. Fitting these models to large datasets often requires colossal amounts of computational power and may give inaccurate results. We discuss these challenges and potential solutions. In particular, we show how methods based on automatic differentiation and the Laplace approximation, like those implemented in Template Model Builder, reduce computational time and increase the robustness of both state and parameter estimates.

DAVID CHAMPREDON, McMaster University
An agent-based model to disentangle the epidemiology of sexually transmitted co-infections

Sexual transmission of HIV (the virus that causes AIDS) can be significantly increased when one partner is co-infected with another sexually transmitted disease (STI). The prevalence of such co-infections in populations affected by HIV being relatively high, one natural question is to what extent other STIs can affect HIV incidence and HIV-focused interventions. To answer this question, the complex interactions between the demographic, epidemiological and biological dynamics need to be understood. Mathematical modelling can definitely help, but many challenges arise when a "realistic" framework is considered. In this presentation, I will briefly describe the agent-based model I'm currently working on to try to decipher this problem.

CLAY CRESSLER, Queen's University

Disentangling the interaction among energy, immunity, and pathogens

The interaction between the immune system and pathogens is often characterised as a predator-prey interaction. This characterisation ignores the fact that both require host resources to reproduce. Here, we propose novel theory that considers how these resource requirements can modify the interaction between the immune system and pathogens. We derive a series of models to describe the energetic interaction between the immune system and pathogens, from fully independent resources to direct competition for the same resource. We show that increasing within-host resource supply has qualitatively distinct effects under these different scenarios. In particular, we show the conditions for which pathogen load is expected to increase, decrease or even peak at intermediate resource supply. We survey the empirical literature and find evidence for all three patterns. These patterns are not explained by previous theory, suggesting that competition for host resources can have a strong influence on the outcome of disease.

ERIC FOXALL, University of Victoria

Social contact processes and the partner model

We consider a version of the contact process on the complete graph with N vertices in which edges open and close dynamically, in this case modelling formation and breakup of monogamous partnerships. We obtain a formula for the basic reproduction number R_0, and show that if $R_0 < 1$ the extinction time for the infection is of order $\log N$ while if $R_0 > 1$ the extinction time is at least $e^{\gamma N}$ for some $\gamma > 0$. Analyzing the mean-field equations, we find a unique and globally attracting (except for the disease-free state) endemic state when $R_0 > 1$ which is also attracting (up to small constant fluctuation in proportion with probability $\geq 1 - e^{-\gamma N}$ up to time $e^{\gamma N}$) for the stochastic model whenever the initial proportion of infectious individuals is positive. Finally, when $R_0 = 1$ the extinction time appears to be of order $\sqrt{N}$, and we give some informal arguments to support this claim.

TARIK GOUHIER, Northeastern University

Coral disease dynamics under environmental change

Reef-building corals have experienced significant declines in recent years due to temperature-induced bleaching events and disease outbreaks. Here, we use a series of spatially-implicit mathematical models to understand how coral-microbial interactions mediate the effects of such environmental change. First, we show the existence of reciprocal feedbacks between coral genetic diversity and disease outbreaks when a tradeoff exists between growth and susceptibility to infection. Under low disease-induced mortality (virulence), genetic diversity amplifies (dilutes) disease outbreaks when transmission is high (low). Amplification occurs when high transmission and low virulence allow the fixation of susceptible but fast-growing genotypes because infected corals occupy patches for a long time and thus make it harder for resistant but slow-growing genotypes to colonize empty patches. Conversely, dilution occurs when low transmission and virulence lead to the fixation of resistant but slow-growing genotypes because there are fewer infected corals and thus more empty patches available.

Next, we show that coral-microbial systems can persist in the face of environmental change via a form of 'extended evolutionary rescue'. Under this scenario, environmental change both decreases coral abundance and destabilizes the dynamics of coral-microbial (meta)communities by inducing high-amplitude limit cycles, which increase the risk of stochastic extinction. However, microbial competition within diverse communities leads to a form of 'ecological rescue' whereby corals maintain higher abundances and experience stable dynamics under environmental change.

Overall, these results suggest that coral-microbial interactions are critical for predicting the effects of environmental change and devising management strategies to ensure the persistence of these important ecosystems.

XI HUO, Ryerson University
An age-structured epidemic disease model with delayed contact tracing

We developed a partial differential equation model about the spread of an infectious disease in a closed community in order to consider the efficacy of several basic public health interventions: (i) identifying and isolating symptomatic cases, (ii) tracing and quarantine of the contacts of identified infectives, and (iii) delayed tracing based on the real-world conduct of contact tracing strategies. Simulations and different aspects of contact tracing effects will be presented and discussed. The model can be applied as a rational basis for decision makers to guide interventions and deploy public health resources in future epidemics. Joint work with Professors Kunquan Lan and Jianhong Wu.

XINGPENG JIANG, Drexel University
Exploring Microbiome Dynamics by Sparse Vector Autoregessive Modeling

Microbial interactions play important roles on the structure and function of complex microbial communities. With the rapid accumulation of high-throughput metagenomic or 16S rRNA sequencing data, it is possible to infer complex microbial interactions. Co-occurrence patterns of microbial species among multiple samples are often utilized to infer interactions. There are few methods to consider the temporally interacting patterns among microbial species. In this work, we present a Graph-regularized Vector AutoRegressive (GVAR) model to infer causal relationships among microbial entities. The new model has advantage comparing to the original vector autoregressive (VAR) model. Specifically, GVAR can incorporate similarity information for microbial interaction inference—i.e. GVAR assumed that if two species are similar in the previous stage, they tend to have similar influence on the other species in the next stage. We apply the model on a time series dataset of human gut microbiome which was treated with repeated antibiotics. The experimental results indicate that the new approach has better performance than several other VAR-based models and demonstrate its capability of extracting relevant microbial interactions.

LINDSAY KEEGAN, McMaster University
Analytic calculations of finite-population reproductive numbers

The basic reproductive number, $\mathcal{R}_0$, provides a foundation for evaluating how various factors affect the incidence of infectious diseases. Recently, it has been suggested that, particularly for vector-transmitted diseases, $\mathcal{R}_0$ should be modified to account for the effects of finite host population within a single disease-transmission generation. Here, we use a transmission-factor approach to calculate such "finite-population reproductive numbers", under the assumptions of homogeneous and heterogeneous mixing, for both vector-borne and directly transmitted diseases. In the case of vector-borne diseases, we estimate finite-population reproductive numbers for both host-to-host and vector-to-vector generations, assuming that the vector population is effectively infinite. We find simple, interpretable formulas for these three quantities. In the direct case, we find that finite-population reproductive numbers diverge from $\mathcal{R}_0$ before $\mathcal{R}_0$ reaches half of the population size. In the vector-transmitted case, we find that the host-to-host number diverges at even lower values of $\mathcal{R}_0$, while the vector-to-vector number diverges very little over realistic parameter ranges.

ALI KHANAFER, University of Illinois at Urbana-Champaign
Stability of SIS Models over Directed Graphs: A Positive Systems Approach

We study the stability properties of a susceptible-infected-susceptible (SIS) diffusion model, so-called the n-intertwined Markov model, over arbitrary directed network topologies. As in the majority of the work on infection spread dynamics, this model exhibits a threshold phenomenon. When the curing rates in the network are high, the all-healthy state is the unique equilibrium over the network. Otherwise, an endemic equilibrium state emerges, where some infection remains within the network. Using notions from positive systems theory, we provide conditions for the global asymptotic stability of the equilibrium points in both

cases over strongly connected networks based on the value of the basic reproduction number, a fundamental quantity in the study of epidemics. When the network topology is weakly connected, we provide conditions for the existence, uniqueness, and global asymptotic stability of an endemic state, and we study the stability of the all-healthy state. Finally, we demonstrate that the n-intertwined Markov model can be viewed as a best-response dynamical system of a concave game among the nodes. This characterization allows us to cast new infection spread dynamics; additionally, we provide a sufficient condition for the global convergence to the all-healthy state, which can be checked in a distributed fashion.

BERNHARD KONRAD, The University of British Columbia
Mathematical models to quantify and optimize HIV interventions in British Columbia

The BC Centre for Disease Control has developed an internet-based service for testing for HIV and other sexually-transmitted diseases, primarily targeting MSM.

We want to learn how to quantify and optimize the impact and cost-effectiveness of this tool to reduce the number of new cases of HIV and the number of people who were not aware they were infected. We start with a simple ODE model of the MSM community and show how survey and behavioural data is used to guide the mathematical modelling process, and how model parameters are extracted.

NICOLE MIDEO, University of Toronto
Explaining the complex lives of malaria parasites

Despite a wealth of biomedical research into the pathogenesis of infectious diseases, little is known about the basic biology of their etiological agents. For many parasites, we lack satisfying answers to questions such as: what is it specifically about the interaction between hosts and parasites that results in disease symptoms? How do these interactions differ between closely related parasite strains or species? And, which factors have shaped parasite traits that determine harm to host and infectiousness? Using a combination of theoretical and experimental approaches, my work has revealed processes that underlie within-host dynamics of experimental rodent malaria infections and how differences in these processes give rise to the variation observed in patterns of disease across parasite genotypes. I will present results that demonstrate the importance of resource availability and competition and show that such 'bottom-up' mechanisms can explain phenomena that are often attributed to immune-mediated processes.

BRYCE MORSKY, University of Guelph
Homophilic replicator equations

Tags are conspicuous attributes of organisms that affect the behaviour of other organisms toward the holder, and have previously been used to explore group formation and altruism. Homophilic imitation, a form of tag-based selection, occurs when organisms imitate those with similar tags. Here we further explore the use of tag-based selection by developing homophilic replicator equations to model homophilic imitation dynamics. We assume that replicators have both tags (sometimes called traits) and strategies. Fitnesses are determined by the strategy profile of the population, and imitation is based upon the strategy profile, fitness differences, and similarity in tag space. We show the characteristics of resulting fixed manifolds and conditions for stability. We discuss the phenomenon of coat-tailing (where tags associated with successful strategies increase in abundance, even though the tags are not inherently beneficial) and its implications for group dynamics. We extend our model to incorporate mutations and invasions to explore their implications upon tag and strategy diversity. We find that homophilic imitation based upon tags significantly affects the diversity of the population, although not the ESS. We classify two different types of invasion scenarios by the strategy and tag compositions of the invaders and invaded. In one scenario, we find that novel tags introduced by invaders become more established with homophilic imitation than without it. In the other, total diversity decreases.

JAMES O'DWYER, University of Illinois
From Bacterial Phylogenies to Ecological Complexity

Macroecological patterns are aggregated over large numbers of individuals, and often display a kind of universal behavior across different systems, independent of differences in their underlying ecological processes. Physicists benefit from several principles which can underly universal behavior: for example, laws of large numbers. Can we identify similar principles in ecology, and understand which phenomena are universal and which are more contingent on mechanism? In this talk I will put these questions in context, and present new models and data which shed light on when and why ecological systems display emergent patterns. The final part of the talk will focus on what these patterns can tell us about the complexity of ecological systems.

IRENA PAPST, McMaster University
(Don't) Panic! — Modelling changes in human behaviour over the course of an infectious disease outbreak

Suppose there is a new and serious infectious disease circulating in your area. Would you go about your daily life just as you do when there is no threat of becoming ill? Probably not: instead, you might wash your hands more often, avoid contact with clearly infected people, be more diligent about using hand sanitizers before meals, or even avoid public places over the course of the outbreak – all in an attempt to reduce your personal risk of becoming infected. These special disease-induced behaviours have been proposed to explain certain observed epidemic patterns (such as multiple waves of infection), but they are often ignored in mathematical modelling of infectious disease spread.

In this talk, I will provide an overview of some work that has been done to model self-initiated changes in human behaviour due to fear of an infectious disease over the course of an epidemic. One approach has been to define a new class of compartmental models where "fearful" susceptible individuals (those that modify their behaviour to reduce their infection risk) are explicitly incorporated into the model as a distinct disease state. I will discuss these models, as well as on-going work to investigate whether it is possible to distinguish the dynamics of these explicit models from those of the simpler SIR model by using incidence data.

SÉBASTIEN PORTALIER, McGill University
Size-related Effects of Physical Factors on Trophic Interactions

Predator and prey interactions are the basis of food web studies and are one of the most important processes acting on the community structure. It is well known now that a given predator feeds on a few prey species, which are only a subset of all available species within the same ecosystem. However, constraints driving prey choice remain challenging to define. Body size is often used as a descriptor of trophic position, but predator and prey body size differences vary widely between aquatic and terrestrial habitats. In this study, we consider how physical aspects of a habitat medium (such as density and viscosity) related to body size can predict the optimal range of prey size for a given predator. Our model leads to several interesting conclusions. First, swimming or flying predators are more constrained than predators living on the floor. Thus, the range of edible prey is narrower for swimming and flying predators. Second, predator strategy (e.g., hunt, ambush, scavenger) influences the range of prey this predator can feed on. Last, the results fit available data, which tends to prove that physical constraints play a major role in predator and prey relationship. The last point is usually neglected in predator and prey studies. These results will be used to parameterize a food web model. It will allow investigating how physical factors can affect the size structure of food webs.

DORA ROSATI, McMaster University
Song popularity as a contagious process

How is it that a song becomes popular? How do certain songs become so much more popular than others? The rapidity with which some songs gain popularity often leads to them being described as 'contagious' or 'infectious'. Upon closer examination, the download time series for some of these songs do resemble epidemic curves derived from case report data for infectious diseases. This suggests that ideas from epidemiological modelling might be useful in investigating how such songs 'spread' through human populations, and that employing disease epidemic models might help to better understand the mechanisms underlying song popularity. Download data were obtained from Nokia based on daily song downloads through Nokia cell phones in 28 countries over a five year period. Songs were characterized by fitting standard epidemic models to song download time series and using these to estimate epidemiological parameter values, providing new insights about popularity of music.

MATTHEW SCOTT, University of Waterloo
Using mathematics to untangle mechanism from physiology

Bacterial growth environment strongly influences the efficacy of antibiotic treatment, and slow growth is often associated with decreased susceptibility. Focusing on ribosome-targeting antibiotics, we find that the interplay between physiology and antibiotic action is more subtle, and in fact faster growth decreases susceptibility for some antibiotics within this class. Remarkably, these observations can be explained by a simple mathematical model that combines drug transport and binding with physiological constraints. Our model reveals that growth-dependent susceptibility is controlled by a single parameter characterizing the 'reversibility' of antibiotic transport and binding. Drug action and bacterial metabolism are mechanistically complex; nevertheless this study illustrates how coarse-grained mathematical models can be used to integrate pathogen physiology into drug design and treatment strategies.

KATHERINE SCRANTON, Yale University
Life history variation and environmental fluctuations jointly shape extinction risk of a population

For many species, life history processes and individual fitness are highly dependent on temperature. Fluctuations around some mean temperature can promote coexistence, prolong persistence times, and alter the overall species composition in a community. These effects are mediated by the thermal response of individual fitness to temperature. In this study we investigate how a population's extinction risk is altered by its environment (mean and variance of temperature fluctuations) using individual thermal response curves. We identify the amount of variation that creates different levels of population extinction risk over timescales between 10 days and 200 years. We also ask how life history variation changes the relationship between the mean and variance of the environment and the risk of extinction. We approximate a set of ordinary differential equations with a Gillespie algorithm where waiting times between birth, death, and fluctuation events are Exponentially distributed. We allow one parameter of individual fitness to follow a Gamma distribution creating intraspecific life history variation. Individual's traits are not heritable but are instead stochastic outcomes of a single distribution, leading to standing variation within a population. We find that population extinction risk is nonlinearly dependent on the mean and variance of the environment, where a population can tolerate higher variation at temperatures below its thermal optimum. Intraspecific variation in the thermal response curve buffers against extinction risk, leading to longer persistence times for populations in environments with mean temperatures around and just above its thermal optimum regardless of the variation present.

ALEXANDRA TESLYA, McMaster University
Predator-prey models with distributed delay: numerical exploration

We are considering predator-prey models with the delay between the consumption of prey to the conversion to predator biomass incorporated. Doing so results in oscillatory behaviour of solutions even in the systems with predator functional responses as simple as the Holling I. In this study we have considered distributed delays and hence modelled the dynamic with integro-differential equations. The point of interest is how different would the resulting dynamics be across various distributions? Or, what is the set of characteristics across parameter space and distributions that will result in the model exhibiting similar dynamics? Local stability analysis shows that systems with various distributions of delay share quite a few properties.

From both mathematical and biological points of view the most interesting dynamics are these of sustained population of both predator and prey. Due to tractability issues these dynamics are hard to establish theoretically. Therefore, we employed numerical bifurcation tools such as XPPAUT and DDEbifTool software to study the local dynamics around coexistence equilibrium and to establish conditions upon which the behaviour of solutions across the systems with various distributions are similar to each other and comparable to the solutions of the system with the discrete delay.

STEVE WALKER, McMaster

YANYU XIAO, University of Alberta
Modeling the effect of human activities on the transmission of vector-borne diseases

Mosquito-borne diseases/zoonoses bring significant amount of deaths among human and other hosts, such as livestock and birds, and cause huge economic costs and losses. Human activities enormously contribute the outbreak and spread pattern of vector-borne diseases/zoonoses. I will present mathematical models to explore that how socioeconomic factors in conjunction with other well-known factors, i.e., disease latency and seasonality, will impact disease dynamics.