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Book of abstracts
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Empirical space-time modeling of emerging forest disease in a heterogeneous landscape of environmental change

HAAS, Sarah 1; Dr. MEENTEMEYER, Ross 2
1 University of North Carolina at Charlotte

Corresponding Author: shaas1@uncc.edu

The dynamic and inherently spatial nature of epidemiological processes presents unique challenges to studying and managing the spread of emerging infectious diseases in natural communities. Long-term ecological studies that analyze conditions of environmental change are needed to understand the spatiotemporal dynamics of the spread and persistence of wildland epidemics. Here, we use spatially-explicit survival analysis to model space-time dynamics of the emerging infectious forest disease, sudden oak death (caused by Phytophthora ramorum) across an extensively monitored plot network (198 plots across 275-km² visited annually from 2004–11). We integrate data on fine-scale forest conditions—stand structure, hourly microclimate variability, and daily precipitation patterns—with broader-scale landscape attributes—edge effects, topography, and force of infection—to identify interactions among environmental factors governing host infection and mortality. During the eight-year study period, oak infection increased from 41–186 trees, while disease mortality rose from 5–57 trees. We found a dilution effect in which sites with high plant species richness had lower disease risk after accounting for other biotic factors, including plot-level host density and the amount of host vegetation surrounding each plot. The diversity effect held after accounting for key abiotic drivers sampled across spatiotemporal scales. Our analyses suggest that coast live oak is highly susceptible to disease across the study area, signaling the potential for sudden oak death to dramatically change oak woodlands through selective removal of a dominant host. This empirical space-time modeling approach illustrates how integrating spatially-structured, longitudinal data sets of real-world spatial, environmental, climatic and species-specific heterogeneity can help us better understand the complex nature of disease impacts to host communities in natural ecosystems.

Modeling concurrent Allee effects in pest control: sterile insect release and scarce mating encounters

GORDILLO, Luis F. 1
1 Department of Mathematics and Statistics, Utah State University

Corresponding Author: luis.gordillo@usu.edu

The release of sterile insects is one of the safest pest control methods currently known. Difference or differential equations based on Knipling’s model provide satisfactory qualitative descriptions of pest dynamics subject to sterile release with relatively high mating encounter rates, but fail otherwise. In this talk I explore numerically new deterministic models suitable for pest populations with scarce mating encounters and subject to sterile release. When insects spatial spread is incorporated through diffusion terms, computations reveal the possibility of steady pest persistence in finite size patches, even if the sterile release threshold for suppression in the non-spatial case is surpassed. In particular, this result holds in presence of density dependence regulation, where sterile release might contribute to induce sudden suppression of the pest population.
Chemotactic adhesion in bacterial flocs

Dr. SIRCAR, Sarthok 1; Dr. BORTZ, David 1
1 University of Colorado, Boulder

Corresponding Author: sircar1981@gmail.com

To understand the adhesion-fragmentation dynamics of bacterial aggregates (i.e., flocs), we model the aggregates as two ligand-covered rigid spheres. We develop and investigate a model for the attachment/detachment dynamics in a fluid subject to a homogeneous planar shear-flow. The binding ligands on the surface of the flocs experience attractive and repulsive surface forces in an ionic medium and exhibit finite resistance to rotation (via bond tilting). For certain range of material and fluid parameters, our results predict a nonlinear or hysteretic relationship between the binding/unbinding of the floc surface and the net floc velocity (translational plus rotational velocity). We show that the surface adhesion is promoted by increased fluid flow until a critical value, beyond which the bonds starts to yield. Moreover, adhesion is not promoted in a medium with low ionic strength, or flocs with bigger size or higher binder stiffness. The numerical simulations of floc-aggregate number density studies support these findings.

Effect of the infectious period distribution on discrete epidemic models

Ms. HERNANDEZ CERON, Nancy 1
1 Purdue University

Corresponding Author: hernann@math.purdue.edu

Although several epidemiological models with arbitrarily distributed infectious period have been developed, the exponential distribution seems to be the most popular assumption in continuous-time models. This assumption produces tractable ODE models, but it is often biologically unrealistic. A similar phenomenon is observed in discrete models, for which constant recovery rates are assumed. The underlying assumption of geometric distribution for such models does have an effect in the model outcomes, including the possibility of producing contradictory predictions, when other discrete distributions are assumed.

In this talk, both a deterministic and stochastic discrete-time model with arbitrarily distributed infectious are presented. The deterministic model can be used to evaluate the impact of waiting-time distributions on epidemiological processes or public health interventions. Model results and simulations highlight the inconsistencies in forecasting that emerge from the use of specific parametric distributions, as well as the relevance of realistic distribution to the dynamics of single epidemic outbreaks.

The stochastic model is used to analyze the repercussions of the choice of this distribution, particularly on the value of \( \mathcal{R}_0 \) and the probability of a major epidemic.
Stability and Bifurcation of the populations in a Fish Farm under the Competition of Fish and Mussel

Dr. GAZI, Nurul Huda

Aliah University

Corresponding Author: nursha@rediffmail.com

The paper is considered for a system with fish and mussel populations in a fish farm where external food is supplied. The ecosystem of the two species is represented by a set of nonlinear differential equations involving nutrient (food), fish and mussel populations. We have described the functions of the fish and mussel populations. The state of eutrophication is a matter of concern. It is found that many lakes have excess load of external food. As a result the ecosystem becomes eutrophic. The mussel in fish farm can help in reducing the eutrophication condition. It consumes the excess food from the water which in turn improves the water quality. This phenomena is modeled and analyzed. We have analyzed the existence of the equilibria of the model populations. We have obtained several criteria for the existence of the equilibrium states. Stability analysis is carried out. It is found that the system becomes stable under certain level of the external food. We have taken into account the gestation time of the fish and mussel. We have incorporated discrete type gestational delay of fish and mussel. The effects of such delays are analyzed extensively. The delay parameter is taken as a bifurcation parameter. We have found that the delay has significant role in shaping the dynamics of the populations of the model system. The delay parameter complicates the dynamics depending on the external food from changing the stable state to unstable periodic trajectories. Applying center manifold theorem and Poincare’ theorem we have determined the stability and period of the periodic oscillation of the populations. We have carried out the numerical simulation to verify the analytical results. The entire study reveals that the external food supply controls the dynamics of the system.

A RESULT AND IT’S APPLICATION ON THE ANALYSIS OF A KIND OF TRANSCENDENTAL CHARACTERISTICS EQUATION

Prof. AKINWANDE, Ninuola

Department of Mathematics Statistics Federal University of Technology P.M.B 65, Minna, Nigeria.

Corresponding Author: aninuola@yahoo.com

In this work, we present a result in the form of a lemma and its application to the stability analysis of some kind of transcendental characteristics equation arising from the perturbation of the steady states of some dynamical systems. Also presented is an application of the result to the non-linear age-structured population model of Gurtin and MacCamy. Sufficient condition for the asymptotic stability of the equilibrium state of the model is obtained in the form of constrained inequality on the vital parameters of the model. The result obtained is compared with those of earlier works.
Modelling the Role of Diagnosis, Treatment, and Health Education on Multidrug-Resistant Tuberculosis Dynamics

Mr. MALIYONI, Milliward ¹; Prof. TCHUENCHE, Jean Michel ²

¹ University of Malawi, Chancellor College, Zomba, Malawi-Southern Africa
² University of Dar es Salaam, Tanzania, East Africa

Corresponding Author: mmaliyoni@gmail.com

Tuberculosis, an airborne disease affecting almost a third of the world’s population remains one of the major public health burdens globally, and the resurgence of multidrug-resistant tuberculosis in some parts of sub-Saharan Africa calls for concern. To gain insight into its qualitative dynamics at the population level, mathematical modeling which require as inputs key demographic and epidemiological information can fill in gaps where field and lab data are not readily available. A deterministic model for the transmission dynamics of multi-drug resistant tuberculosis to assess the impact of diagnosis, treatment, and health education is formulated. The model assumes that exposed individuals develop active tuberculosis due to endogenous activation and exogenous re-infection. Treatment is offered to all infected individuals except those latently infected with multi-drug resistant tuberculosis. Qualitative analysis using the theory of dynamical systems shows that, in addition to the disease-free equilibrium, there exists a unique dominant locally asymptotically stable equilibrium corresponding to each strain. Numerical simulations suggest that, at the current level of control strategies (with Malawi as a case study), the drug-sensitive tuberculosis can be completely eliminated from the population, thereby reducing multi-drug resistant tuberculosis.
Pattern Formation in Epidemic Models with Application in Disease Spread and Control

Mr. MUHAMMAD, Abdullahi Yau 1

1 University of Sussex

Corresponding Author: ma526@sussex.ac.uk

Reaction-diffusion (RD) systems have been used over the years to model the distribution and interaction of populations in their spatial environment. In this work we consider an SIS spatial epidemic model with nonlinear incidence rate that comprises of susceptible $S$ and infective $I$ individual populations which interact randomly. We derive the conditions for local and global bifurcations and prove that global bifurcation of spatially non homogeneous stationary patterned solutions occurs in the domain of parameters. Turing instability conditions are established and analyzed for the model to exhibit patterns. In particular, the exact Turing domain is found in the parameter space. Conditions for predicting spots or stripes (labyrinthine) in the parameter regime are obtained. Also, we derive the basic reproduction ratio $\mathcal{R}_0$ using the next generation matrix method and prove that for $\mathcal{R}_0 > 1$ the disease free steady state is globally asymptotically stable and for $\mathcal{R}_0 < 1$ the endemic state is stable to initial perturbations.

In our numerics we use the Implicit-Explicit (IMEX) Euler's method, which compute diffusion terms in fourier space and reaction terms in real space. We benchmark our model to prove the stability of the method and find that the method converges with correct order. Rigorous numerical simulations reveal that the model has rich dynamics and complex patterns, for a fixed $r$, on increasing the bifurcation parameter $\beta$. We observe that whenever $\beta \ge r$ there is always an outbreak and for $\beta < r$ there is no outbreak. Therefore, to keep an epidemic under control we have to keep $\beta < r$. The results obtain extend well the findings of pattern formation in epidemic models and may have direct implications for the study of disease spread and control and perhaps the mechanistic impact of public health interventions on epidemics.

Finally, we also prove that in certain parameter regime it is possible to have localized hexagon patches (patterns). This is good news for disease control and localization.

Optimal Control Applied in Coupled Within-host and Between-host Models

NUMFOR, Eric 1; Prof. LENHART, Suzanne 1

1 University of Tennessee

Corresponding Author: numfor@math.utk.edu

Despite advancements made with the study of immunological and epidemiological models, the outbreak of some diseases cannot still be predicted. Thus, we formulate an immuno-epidemiological model of coupled “within-host” model of ODEs and “between-host” model of ODE and PDE. Existence and uniqueness of solution to the “between-host” model is established, and an explicit expression for the basic reproduction number of the “between-host” model derived. Stability of disease-free and endemic equilibria is investigated. An optimal control problem with drug-treatment control on the within-host system is formulated and analyzed. Numerical simulations based on the forward-backward sweep method are obtained.
C3_Immunology / 17

Are Small Granulomas Stable?

Prof. FRIEDMAN, Avner ¹, Dr. LAM, King-Yeung ²

¹ Ohio State University
² Mathematical Biosciences Institute, Ohio State University

Corresponding Author: lam.184@osu.edu

Approximately one third of the human population are infected by Mycobacteria tuberculosis, yet only a few millions are clinically sick. The reason for this disparity is that under small amount of bacteria inhalation the granulomas formed by the macrophages are small and either remain stable or eventually shrink to zero. In the present paper we develop a simple free-boundary type model of granuloma. The model involves just macrophages and bacteria, and was introduced earlier, in the radially symmetric case, in [Friedman-Kao-Leander, submitted]. We first establish the existence of radially symmetric steady state granulomas with any radius $R$, $0 < R < R^*$, where $R^*$ is given explicitly by one of the model's parameters. Then we consider mathematically the linearized stability/instability of small radially symmetric steady states. This is joint work with Avner Friedman (OSU).

C2_Chemotaxis/Population / 18

Parameter Estimation and Sensitivity Analysis of Delay Differential Equations with Application to Population Models

Dr. BURNS, John ¹; Dr. CLIFF, Gene ¹; Dr. FARLOW, Kasie ¹

¹ Virginia Tech

Corresponding Author: jaburns@vt.edu

In this paper we consider the problems parameter estimation and sensitivity analysis for systems governed by delay differential equations. We consider the case where one assumes both modeling and measurement errors and show that in some cases using hierarchical modeling can help identify modeling errors. We apply these ideas to several examples involving delay differential equation models of population dynamics.
Dynamics of Phytoplankton Communities Under Photoinhibition

Prof. HSU, Sze-Bi 1; Dr. LIN, Chiu-Ju 2; Prof. HSIIEH, Chih-Hao 3; Prof. YOSHIYAMA, Kohei 4

1 National Center for Theoretical Sciences, Department of Mathematics, National Tsing-Hua University
2 Department of Mathematics, National Tsing-Hua University
3 Institute of Oceanography and Institute of Ecology and Evolutionary Biology, National Taiwan University
4 River Basin Research Center, Gifu University

Corresponding Author: d9621806@oz.nthu.edu.tw

We analyzed a model of phytoplankton competition for light in a well-mixed water column. The model, proposed by Gerla et al. (Oikos 120:519–527, 2011), assumed inhibition of photosynthesis at high irradiance (photoinhibition). We described the global behavior through mathematical analyses, providing a general solution to the multi-species competition for light with photoinhibition. We classified outcomes of 2- and 3-species competitions as examples, and evaluated feasibility of the theoretical predictions using empirical relationships between photosynthetic production and irradiance. Numerical simulations with published p–I curves indicate that photoinhibition may often lead to strong Allee effects and competitive facilitation among species. Hence, our results suggest that photoinhibition may play a major role in organizing phytoplankton communities.

Predicting Major Outbreaks for Diseases Transmitted Through the Environment

Dr. LAHODNY JR., Glenn 1; Dr. GAUTAM, Raju 2; Dr. IVANEK, Renata 2; Dr. BANI-YAGHOUB, Majid 3

1 Texas A&M University
2 Texas A University
3 University of Missouri - Kansas City

Corresponding Author: glahodny@cvm.tamu.edu

Indirect transmission, shedding of a pathogen by infectious hosts, growth of the free-living pathogen within the environment, and environmental decontamination can all play important roles in the emergence and spread of infectious diseases. To account for these factors, an epidemic model is introduced which includes both direct and indirect transmission as well as a compartment representing the free-living pathogen load in the environment. Based on the assumptions of the deterministic model, a corresponding continuous-time Markov chain model is developed and a result from the theory of multitype branching processes is used to estimate the probability of a major outbreak. Numerical examples illustrate the results for cholera in a human population as well as salmonellosis in a dairy herd. It is shown that environmental decontamination alone does not significantly affect the probability of a major outbreak. However, decontamination paired with additional control efforts is effective in preventing an outbreak. The stochastic results quantify the initial load of free-living pathogen required to produce an outbreak with the same probability as in the presence of one infectious host. The results have implications for disease control and farm management.
Measuring the efficacy of influenza antivirals

BEGGS, Noah 1; Dr. DOBROVOLNY, Hana 1

1 Texas Christian University

Corresponding Author: h.dobrovolny@tcu.edu

Influenza is a serious illness that causes thousands of deaths annually and has the potential to cause pandemics leading to millions of deaths. There are currently two classes of antivirals available to treat influenza: adamantanes, which prevent uncoating of the virus, and neuraminidase inhibitors (NAIs), which prevent release of new virions. Antivirals are the first line of defense against influenza, so the efficacy of current treatments must be evaluated for each new strain. Mathematical models of the infection process can help in this assessment, but we first need to accurately measure the parameters characterizing the efficacy of antivirals. We use a mathematical model to simulate in vitro experiments to determine which measurements will accurately determine drug efficacy parameters. We find that the efficacy of NAIs can be determined by measuring viral load during a single cycle assay, while the efficacy of adamantanes can be determined by measuring infected cells during the preparation stage for the single cycle assay.

Stochastic model of E. coli O157:H7 transmission in a cattle population

WANG, Xueying 1; Prof. ALLEN, Linda 2; Prof. IVANEK, Renata 3; Dr. GAUTAM, Raju 3; Prof. PINEDO, Pablo 3

1 Washington State University
2 Texas Tech University
3 Texas A University

Corresponding Author: xueying.wsu@gmail.com

E. coli O157:H7 is an important foodborne pathogen with a natural reservoir in the cattle population. To understand the spread and persistence of E. coli O157:H7 infection in cattle so that better infection control strategies can be designed, we propose a stochastic model for E. coli O157:H7 transmission in cattle. In this work, the extinction and outbreak of infection is studied by solving Kolmogorov equations associated with statistics of the time to extinction and outbreak. Our results provide insight into E. coli O157:H7 transmission and extinction, and suggest ways for controlling the spread of infection in a cattle herd. Specifically, this study highlights the importance of ambient temperature and sanitation, especially during summer.
M8_Stochastic and Deterministic Modeling and Analysis of Biological Systems / 25

On Parameter Variation in the Mathematical Modeling of Malaria Transmission

Mr. LYATUU, Lyatuu; Dr. SATHANANTHAN, Sivapragasam

1 Ifakara Health Institute
2 Tennessee State University

Corresponding Author: ilyatuu@ihi.or.tz

Mathematical modeling of malaria began in 1911 from Ross SIR population compartment models and followed by a major extension done by MacDonald in 1957. Recently, there have been a number of modifications to suit different types of scenarios [1] [2]. Of the many challenges that faces this type of modeling is the availability and variation of parameter values. Differences in the type of the parasite involved in the infection, variation of mosquito species, environmental variability and changes in the ecological system bring about disparity in parameter variation [3] [4]. Pointed out by Koella [4], the amount of variability in transmission parameters strongly affects the outcome of control measures and that predictions of the outcome can be misleading. Using the ODE type, we develop a tool to measure sensitivity analysis in the mathematical modeling of malaria transmission.

C5_Epidemiology / 27

Anti-pathogen genes and the replacement of disease vector populations: a model-based evaluation of hybrid strategies

Mr. ROBERT, Michael; Dr. OKAMOTO, Kenichi; Dr. GOULD, Fred; Dr. LLOYD, Alun

1 North Carolina State University

Corresponding Author: marober5@ncsu.edu

In recent years, genetic strategies aimed at controlling populations of disease vectors have received considerable attention as alternatives to traditional control measures. Theoretical studies have shown that Female-Killing (FK), Anti-Pathogen (AP), and Reduce and Replace (R) strategies can each cause reductions in competent vector populations. In this talk, we utilize a relatively simple ordinary differential equations model to evaluate the impacts on competent populations of the dengue vector Aedes aegypti of FK-only, AP-only, and R-only; releases as well as hybrid strategies that result from combinations of these three approaches. We show that while the relative efficacy of each of the strategies depends upon the release ratio, release duration, inclusion of females, and switch time in hybrid strategies, AP-only releases rather surprisingly lead to the greatest reduction in competent vectors in most scenarios. R releases followed by AP releases are also very effective at reducing competent vector population density in many scenarios. In all scenarios we considered, FK and FK followed by R releases led to the least long-term reduction in competent vectors. We discuss the role that linkage disequilibrium and inclusion of females play in the efficacy of the strategies relative to one another, and we motivate the continued development of AP strategies by discussing their benefits over other strategies.
C1_Epidemiology / 28

An SIR Metapopulation Model with the Allee Effect, with application to Controlling Plague in Prairie Dog Colonies

EKANAYAKE, Amy J. 1; EKANAYAKE, Dinesh B. 1

1 Western Illinois University

Corresponding Author: aj-ekanayake@wiu.edu

Reduced per-capita birth rates at low population densities have been observed among some wildlife species. This so-called Allee effect has important ramifications when disease causes a sudden population decline. For species living among fragmented or patchy habitats, disease combined with the Allee effect may quickly drive a patch’s population to extinction. Yet local extinction may not be indicative of global extinction, and a patch may, in fact, become recolonized by individuals dispersing from nearby patches. We introduce a deterministic, and corresponding stochastic, SIR epidemic model to study the effect of disease on metapopulation dynamics in the presence of the Allee effect.

The Allee effect is generally represented by altering the logistic equation: \( \frac{dN}{dt} = \gamma N(N-\theta)(\kappa-N) \), where \( \gamma > 0 \), \( \kappa \) is the carrying capacity, and \( \theta \) is the Allee threshold. However, this mathematical representation neither accommodates a weak Allee effect (having no threshold) nor allows for the distinguishing of separate birth and death rates, which are important for stochastic models. We discuss a mathematical representation that allows a weak Allee effect and obtain a spatially explicit stochastic differential equation model for a large system of patches. Then we consider several strategies for control of a disease spreading among such a patch system.

Finally, we apply the models to evaluate the dynamics of a population of prairie dogs living in patch-like colonies and suffering from the slyvatic plague. Existing prairie dog models have not been suitable for studying how the spatial structure of prairie dog metapopulations could be exploited to protect the species against extinction due to plague or to develop control methods. In this example, we use the SIR stochastic model to evaluate the effects of spatial structure, dispersal, and the Allee effect on extinction. We further evaluate the effects of: control of the flea population, control by removing individuals from affected patches, and control by immunization, on eradicating the plague from a large patch system connected through dispersal.

C6_Ecology / 30

Dynamics of Adult trees, Seedlings and Herbivores in Presence of Plant Toxin Defense

Mr. ZHENG, Yiqiang 1; Prof. FENG, Zhilan 1

1 Department of Mathematics, Purdue University

Corresponding Author: zheng30@math.purdue.edu

One of the possible consequences of global warming in the northern hemisphere is the invasion of Arctic Tundra by woody plants, which is influenced by the chemical defense of plants against mammal browsing. In this paper, we explore the toxin-mediated plant-herbivore interaction with size structure in plants using a simplified mathematical model. The size structure of plants is important as it seems to play a significant role on the woody plant invading. We formulate a three dimensional ordinary differential equations model of the simplified dynamics of adult-trees, seedlings and herbivores in the presence of chemical defense of plants. Then we investigate the mathematical features of the model, analyze the stability of equilibria. When choosing plant toxin measurement \( G \) and herbivore health measurement \( \mu \) as bifurcation parameters, we analytically show the existence of different bifurcations, including a saddle-node bifurcation and a Hopf bifurcation. A Bogdanov-Takens cusp with codimension 2 is also identified. A Bogdanov-Takens bifurcation occurs when bifurcation parameters are perturbed at the cusp.
C3_Immunology / 31

Transient spikes after vaccine introduction

Mr. PANDEY, Abhishek¹; Dr. MEDLOCK, Jan²; Dr. MUBAYI, Anuj³

¹ Clemson University
² Oregon State University
³ Northeastern Illinois University

Corresponding Author: abhishe@g.clemson.edu

A dengue vaccine is expected to be available within few years. Once vaccine is available, policy makers will need to find suitable vaccine allocation policies. Because of seasonal oscillations in mosquito abundance, dengue models may predict a transient period after vaccine introduction where incidence can spike higher than in pre-vaccine period which could lead to doubts about vaccination program and possibly public panic. Using a simple dengue transmission model, we show that transient spikes in incidence is a robust phenomenon and it occurs as long as vaccine efficacy and vaccination rate are not both either very high or very low. Despite the presence of transient spikes in incidence, the total number of infections in the 15 years after vaccine introduction was always lower than without the vaccine. Therefore, the transient spikes might mislead the public and policy makers into concluding incorrectly that the vaccine program is ineffective. Policy makers should prepare for spikes in incidence after vaccine introduction to mitigate the burden of these spikes and resulting perception of inefficacy of the vaccine program.

C7_Epidemiology / 32

Modeling Study of Sterile Insect Technique in Fighting Mosquito-Borne Diseases

Prof. LI, Jia¹

¹ University of Alabama in Huntsville

Corresponding Author: li@math.uah.edu

To prevent the transmission of malaria or other mosquito-borne diseases, one of the effective weapons is using the sterile insect technique (SIT) to release sterile mosquitoes to reduce or eradicate the wild mosquito population. To study the impact of SIT on the diseases transmission, we formulate simple mathematical models for the interactive wild and sterile mosquitoes, incorporating different strategies in releasing sterile mosquitoes. We investigate the model dynamics and compare the different release strategies. Numerical examples are also given to demonstrate rich dynamical features of the models.
A case of sitting pups: Special predators, endangered prey, and the importance of structural uncertainty in selective predator removal programs

Mrs. CRAWFORD, Tara 1; Dr. SALAU, Kehinde 2
1 University of Georgia
2 University of Arizona

Corresponding Author: krsalau@email.arizona.edu

Selective removal of “problem” predators is sometimes proposed as a means to enhance endangered prey populations. Justification for these lethal control programs rests on two key assumptions: 1) only a specialized subset of the predator population is responsible for the conflict, and 2) that the conflict will cease after those individuals are removed. Many of these cases require prompt action and are of great public interest (e.g., Galapagos sharks/Hawaiian monk seals). It is important to evaluate the factors influencing management success in these cases. In particular, to what degree does violating the aforementioned assumptions influence the long-term success of management as it relates to increasing the size of an endangered prey population? A theoretical agent-based model is used to test the effectiveness of selective removal programs in scenarios that differ in three key ways: 1) the degree to which individual predators specialize on endangered prey; 2) the mode by which individuals become specialized; and 3) the accuracy of lethal removal efforts. Further model development and analyses are warranted; however, these interim results suggest that the success of selective predator removal programs intended to enhance endangered prey populations is more dependent on accurate identification of problem individuals than the degree and mode of their specialization in endangered prey.

A Structured Avian Influenza Model with Imperfect Vaccination

GULBUDAK, Hayriye 1; Prof. MARTCHEVA, Maia 1
1 University of Florida

Corresponding Author: hgulbudak@ufl.edu

Vaccination of poultry is an important control strategy for avian influenza. In general, it is clear that vaccination does not induce perfect immunity in populations, instead producing partial protection from disease. The mechanisms of this partial protection offered by vaccination include reducing the probability of infection and decreasing the severity of infection. In mathematical models of vaccination, the partial protection is usually modeled by considering reduction in probability of infection and loss of immunity is assumed to occur with constant per-capita rate. However, this may not capture all of the complexities of vaccination.

We introduce a model of avian influenza in domestic birds with imperfect vaccination and age-since-vaccination structure, which includes the two mechanisms of partial protection. The basic reproduction number, $\mathcal{R}_0$, is calculated. The disease-free equilibrium is found to be globally stable when $\mathcal{R}_0<1$ under certain conditions. When $\mathcal{R}_0>1$, existence of an endemic equilibrium is proved (with uniqueness for a special case), and uniform persistence of the disease is established. The inclusion of both mechanisms of partial protection from vaccination can have important implications for disease control. We analytically and numerically demonstrate that vaccination can paradoxically increase the total number of infected, resulting in the “silent spread” of the disease.
M3_Population-Level Epidemiological Modeling with an Ecological Perspective / 35

A size-structured population model with distributed states-at-birth: numerical approximations

LI, Xinyu 1; Prof. FARKAS, Jozsef 2; Prof. ACKLEH, Azmy 1; Dr. MA, Baoling 3

1 University of Louisiana at Lafayette
2 University of Stirling
3 Louisiana Tech University

Corresponding Author: baolingm@latech.edu

A size-structured population model where individuals may be recruited into the population at different sizes is considered. First and second order finite difference schemes are developed to approximate the solution of the mathematical model. The convergence of the approximations to a unique weak solution with bounded total variation is proved. We then show that as the distribution of the new recruits become concentrated at the smallest size, the weak solution of the distributed states-at-birth model converges to the weak solution of the classical Gurtin-McCamy-type size-structured model in the weak-star topology. Numerical simulations are provided to demonstrate the achievement of the desired accuracy of the two methods for smooth solutions as well as the superior performance of the second-order method in resolving solution-discontinuities.

M8_Stochastic and Deterministic Modeling and Analysis of Biological Systems / 36

Stochastic Modeling of Parasites in Host Populations

Dr. ARCINIEGA, Armando 1

1 The University of Texas at San Antonio

Corresponding Author: armando.arciniega@utsa.edu

The complexity of the host-parasite relationship, and its depression of host populations has been investigated and successfully modeled for large populations using deterministic methods. These models fail to accurately predict interactions within smaller populations, and require a statistical component to reclaim some degree of accuracy. This research employs stochastic differential equations to better forecast changes in small populations, and, in a practical setting, seeks to write software that will automate this procedure.
**M6_Mathematical Modeling of Prion Dynamics and Transmission / 38**

**Homogenization, Sex, and Differential Motility Predict Spread of Chronic Wasting Disease in Mule Deer in Southern Utah**

Dr. GARLICK, Martha ¹; Dr. POWELL, James ²; Dr. HOOTEN, Mevin ²; Ms. MCFARLANE, Leslie ³

¹ South Dakota School of Mines and Technology  
² Utah State University  
³ Utah Division of Wildlife Resources

**Corresponding Author:** martha.garlick@sdsmt.edu

Chronic wasting disease (CWD) is an infectious prion disease that mule deer, along with other Cervids. It is a slow-developing, fatal disease which is rare in the free-ranging deer population of Utah. We present a sex-structured, spatial model for the spread of CWD over heterogeneous landscapes, incorporating both horizontal and environmental transmission pathways. To connect the local movement of deer to the regional spread of CWD, we use ecological diffusion with motility coefficients estimated from mule deer movement data. Ecological diffusion allows for aggregation of populations in desirable habitats and therefore allows for an interaction between density dependent disease transmission and landscape structure. The major innovation presented is use of homogenization to accelerate simulations of disease spread in southeastern Utah, from the La Sal Mountains near Moab to the Abajo Mountains near Monticello. The homogenized model provides accuracy while maintaining fidelity to small-scale habitat effects on deer distribution, including differential aggregation in land cover types with high residence times, with errors comparable to the order parameter measuring separation of small and large scales.

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**C6_Ecology / 39**

**Sensitivity Analysis for a Nonlinear Size-Structured Population Model**

Dr. DENG, Keng ¹

¹ University of Louisiana at Lafayette

**Corresponding Author:** deng@louisiana.edu

In this talk, we consider a nonlinear size-structured population model with vital rates depending on the total population. We derive sensitivity partial differential equations for the sensitivities of the solution with respect to the reproduction and mortality rates. We also present numerical results to illustrate the use of these sensitivity equations.
**M1_Persistence and Permanence in Multi-Species Population Models / 40**

**Permanence of a general discrete-time two-species-interaction model with nonlinear per-capita growth rates**

Dr. KANG, Yun

1 Arizona State University

**Corresponding Author:** yun.kang@asu.edu

Abstract for the minisymposium "Persistence and Permanence in Multi-Species Population Models":

The per-capita growth rate of a species is influenced by density-independent, positive and negative density-dependent factors. These factors can lead to nonlinearity with a consequence that species may process multiple nontrivial equilibria in its single state (e.g., Allee effects). This makes the study of permanence of discrete-time multi-species population models very challenging due to the complex boundary dynamics. In this talk, we explore the permanence of a general discrete-time two-species-interaction model with nonlinear per-capita growth rates and derive a simple sufficient condition for guaranteeing the permanence of the system by applying and extending the ecological concept of the relative nonlinearity to estimate systems' external Lyapunov exponents. Our method allows us to fully characterize the effects of nonlinearities in the per-capita growth functions and implies that the fluctuated populations may devastate the permanence of systems and lead to multiple attractors. These results are illustrated with specific two species competition and predator-prey models with generic nonlinear per-capita growth functions. Finally, we discuss the potential biological implications of our results.

**M5_Chemotactic Modeling of Inflammatory Type Diseases / 41**

**A Reaction-Diffusion Model of Chemotactic Processes in Atherogenesis**

Prof. RITTER, Lake 1; Prof. IBRAGIMOV, Akif 2; Prof. WALTON, Jay 3

1 Southern Polytechnic State University

2 Texas Tech University

3 Texas A M University

**Corresponding Author:** lritter@spsu.edu

Atherosclerosis is a disease of the vasculature characterized by chronic inflammation. It is understood that low density lipoproteins (LDL) trapped inside the arterial wall are subject to oxidative modification, and that the resulting oxidized LDL interferes with normal immune function. In particular, macrophages may become engorged with lipids—forming foam cells through failed phagocytosis—initiating a lesion in the tissue as well as instigating an inflammatory feedback loop driven by chemotaxis. A mathematical model of these biochemical-immune processes of atherogenesis will be presented. The model takes the form of a coupled, nonlinear system of partial differential equations including elements based on the classic Keller-Segel-Patlak model of chemotaxis. The equations, governing the densities of chemical and cellular species, are assumed to hold in some fixed domain representing an artery. Initiation of an inflammatory spiral is viewed as resulting from a mathematical instability off of an equilibrium state characterized as healthy (i.e. lacking certain disease features). Particular attention is given to the competing processes of diffusion and chemotaxis both within the interior of the domain (modeling the subendothelial intima) and at the domain boundary (corresponding to the endothelial layer).
A DISCONTINUOUS GALERKIN LEAST-SQUARES FINITE ELEMENT METHOD FOR SOLVING FISHER’S EQUATION

Dr. LIN, Runchang
Texas A&M International University
Corresponding Author: rlin@tamiu.edu

In the present study, a discontinuous Galerkin least-squares finite element algorithm is developed to solve Fisher’s equation. The present method is effective and can be successfully applied to problems with strong reaction, to which obtaining stable and accurate numerical traveling wave solutions is challenging. Numerical results are given to demonstrate the convergence rates of the method and the performance of the algorithm in long-time integrations.

On a Discrete Selection-Mutation Model

SALCEANU, Paul; Prof. ACKLEH, Azmy; Prof. SACKER, Robert
University of Louisiana at Lafayette; University of Southern California
Corresponding Author: salceanu@louisiana.edu

A discrete-time population model in which individuals are distributed over a discrete phenotypic trait-space is studied. It is shown that when the mutation matrix $\Gamma$ is irreducible then all strains persist uniformly and that when, in addition, mutation is small, then an interior equilibrium exists, in which all traits coexist. When mutation is not necessarily small, but under some additional conditions, the global attractor for the interior of the positive cone is still an equilibrium. When $\Gamma$ is block-diagonal, sufficient conditions are given for competitive exclusion among phenotypes.

Modeling reproductive conflict in group-living organisms

Dr. HAMILTON, Ian
The Ohio State University
Corresponding Author: hamilton.598@osu.edu

Skew models predict how reproduction or resources produced by the group are divided between dominant and subordinate group members. These include transaction models, in which one group member may allocate some group productivity to another to prevent group dissolution and tug-of-war inefficiency models, in which group members compete for access to resources at a cost to group productivity. However, empirical support for existing models is limited and the assumptions of these models do not fit many group-breeding organisms. I will present two recent extensions of skew models. In the first, I expand transactional and tug-of-war models to allow one individual (the follower) to respond plastically to a fixed strategy of its partner (the leader). For the second, I allow other members of the group to influence pairwise interactions over the division of reproductive benefits.
**M3_Population-Level Epidemiological Modeling with an Ecological Perspective / 45**

**Modeling ecological and evolutionary risks in the dynamics of Trypanosoma cruzi strains in sylvatic hosts**

Dr. MUBAYI, Anuj 1; Mr. MEEKS, Jonathan 1; Ms. SOLIS, Gabriela 1; Prof. KRIBS-ZALETA, Christopher 2

1 Northeastern Illinois University
2 University of Texas-Arlington

**Corresponding Author:** a-mubayi@neiu.edu

In this study, ecological evolutionary, and epidemiological factors are incorporated in a model that capture the transmission dynamics of Trypanosoma cruzi (T. cruzi) parasite in sylvatic hosts. Using an example of Chagas disease, an infectious disease caused by a parasite T. cruzi and transmitted to humans and other mammals by blood-sucking Triatominae bugs, we study the dynamics of two competing strains of T. cruzi enzootic to North America as a context to understand the role that local stochasticity in demographical and epidemiological factors may play in the dynamics of invasion risk, wherein one strain is introduced to a population of hosts and vectors in which the other strain is resident. The study uses mathematical models that incorporate dynamics of two T. cruzi strains and attempts to understand the role that adaptations of each strain to distinct modes of transmission may play in the competition between the two strains. A deterministic model incorporating contact process saturation predicts competitive exclusion, and reproductive numbers for the infection provide a framework for evaluating the competition in terms of adaptive trade-off between distinct transmission modes and the risk associated with it. Results highlight the importance of oral transmission in mediating the competition between horizontal (stercorarian) and vertical transmission; its presence as a competing contact process advantages vertical transmission even without adaptation to oral transmission, but such adaptation appears necessary to explain the persistence of (vertically-adapted) T. cruzi IV in raccoons and woodrats in the southeastern United States. Whereas the stochastic models illustrate the impact of stochastic effects during the (often long) transient dynamics at the outset of an invasion, in ways that deterministic models do not.

**Reception & Poster Session / 46**

**Climate change and influenza: public health implications of warmer than average winters**

Dr. TOWERS, Sherry 1

1 Arizona State University

**Corresponding Author:** smtowers@asu.edu

The 2012-13 influenza season was unusually early and severe, following on the heels of the record mild influenza season of 2011-2012 which occurred during the fourth warmest and driest winter on record. We examine past US influenza seasons, and show that epidemic severity not only depends on current climate, but also the climate and epidemic severity of the season before. We discuss resulting influenza public health implications of global warming.

We employ a Susceptible, Exposed, Infected, Recovered (SEIR) compartmental disease model to simulate the spread of avian influenza amongst poultry in live bird markets. We show that the population dynamics of the live bird market markets, where large numbers of birds are regularly shipped in and then sold within a few days of arrival, can lead to high endemic prevalence of influenza in birds in the market.
Adapting Agent-based Models for Mosquito Borne Disease Using a Network-Patch Framework

MNISZEWSKI, Susan

Los Alamos National Laboratory

Corresponding Author: smm@lanl.gov

Agent-based models (ABM) are commonly used to simulate the spread of infectious disease such as influenza through a population. Detailed human movement, demography, realistic business location networks, and in-host disease progression are available in existing ABMs, such as the Epidemic Simulation System (EpiSimS). These capabilities make possible the exploration of pharmaceutical and non-pharmaceutical mitigation strategies through simulation used to inform the public health community. There is a similar need for the spread of mosquito borne pathogens due to the re-emergence of diseases such as chickungunya, dengue fever, and malaria. A network-patch model for mosquito dynamics has been coupled with EpiSimS, known as EpiPatch. Mosquitoes are represented as a "patch" or "cloud" associated with a location. Each patch has an ordinary differential equations (ODE) mosquito dynamics model and mosquito related parameters relevant to the location characteristics. Activities at each location can have different levels of potential exposure to mosquitoes based on whether they are inside, outside, or somewhere in-between. The probability of disease transmission depends on the mosquito dynamics in the patch a person enters. This probability can depend on mosquito densities, the infection status of humans that previously entered the patch, environment, weather, types of activities, disease properties, proportion of infected mosquitoes, and mitigation strategies. A description of EpiPatch will be presented with preliminary results. This coupled model will help quantify the importance of heterogeneity in predicting the spread and invasion of mosquito borne pathogens.

MODELING INFLAMMATORY PROCESSES ASSOCIATED WITH ATHEROGENESIS AND ATHEROSCLEROSIS

Prof. WALTON, Jay

Department of Mathematics Texas A University

Corresponding Author: akif.ibragimov@ttu.edu

In this talk, both atherogenesis and atherosclerosis are viewed as vascular diseases driven by inflammatory processes fueled by low-density lipoproteins (LDL) undergoing oxidative damage. Atherogenesis is modeled as an inflammatory instability of a governing reaction-diffusion-chemotactic (RDC) system of partial differential equations over a fixed domain (an arterial wall, for example), while modeling atherosclerotic lesion growth requires coupling the RDC system to the linear momentum (LM) system governing the mechanical response of the affected soft-tissue. Conventional wisdom in this setting views high-density lipoproteins (HDL) as mitigating factors through reverse cholesterol transport. However, there is mounting evidence that HDL can be corrupted to a dysfunctional, pro-inflammatory, destabilizing state that can trigger atherogenesis and promote atherosclerotic lesion growth. This talk presents a model of this process, discusses the role played by anti-oxidents in stabilizing the inflammatory process, and presents numerical simulations of a (axisymmetric) free-boundary problem modeling lesion growth.
Designing a biosurveillance network for avian influenza outbreaks in Nigeria

BROWN, Mac 1; MOORE, Leslie 2; BERENDZEN, Joel 3; LOEPPKY, Jason 4; MCMAHON, Benjamin 3; FAIR, Jeanne 3; MANORE, Carrie 5; POWELL, Dennis 3; LABUTE, Montiago 6; HYMAN, James 3; RIVAS, Ariel 3; JANKOWKI, Mark 8

1 National Security Technologies LLC
2 Los Alamos National Laboratory
3 LANL
4 University of British Columbia
5 Tulane University
6 Lawrence Livermore National Laboratory
7 University of New Mexico
8 U.S. Fish and Wildlife

Corresponding Author: cmanore@tulane.edu

Minisymposium: Modeling for biosurveillance and risk assessment of emerging infectious diseases

Emerging infectious diseases from wildlife or livestock sources are of increasing concern and effective bio-surveillance important. However, especially in developing nations, resources for surveillance and mitigation are often limited and determining optimal surveillance networks for an emerging pathogen is difficult. Mathematical modeling can play a unique role in examining a wide range of scenarios of pathogen spread, helping us to determine good surveillance policies. We use a hybrid network-differential equation model (MuSE) designed to model a wide range of hosts, pathogens, and pathogen introduction locations to model the spread of avian influenza in Nigeria. We determine a surveillance network which is robust over a wide range of avian pathogen parameters and mitigation strategies and validate with data from an H5N1 outbreak in Nigeria in 2006-2007. The methods used here can apply to a variety of pathogens, hosts, and locations to help robustly determine optimal surveillance locations, particularly when resources are scarce but early detection is important.
How Many Suffice? A Computational Method for Sizing Sentinel Surveillance Networks

FAIRCHILD, Geoffrey ¹; POLGREEN, Philip ²; POSTER, Eric ²; RUSHTON, Gerard ²; SEGRE, Alberto ²

¹ University of Iowa / Los Alamos National Laboratory
² University of Iowa

Corresponding Author: geoffrey-fairchild@uiowa.edu

Data from surveillance networks help epidemiologists and state public health officials detect emerging diseases, conduct outbreak investigations, manage epidemics, and better understand the mechanics of a particular disease. In previous work, we showed how to apply facilities location algorithms to designing disease surveillance networks and primary stroke center networks. In this work, we introduce the notion that surveillance networks are used to determine outbreak intensity (i.e., disease burden), outbreak location, and outbreak timing (i.e., the start, peak, and end of the epidemic). In this work, we primarily consider outbreak intensity and outbreak timing. We focus on outpatient influenza surveillance, although these methods are general and applicable to many more disease surveillance networks.

We developed a web-based calculator that provides a simple user interface for public health officials to determine the best site placement for their state. The web-based calculator supports three different site placement algorithms: two algorithms based on the maximal coverage model and one based on the K-median facilities location model. The maximal coverage model (MCM) considers each site as having a fixed coverage radius. The MCM chooses the sites that maximize the total number of people within the specified distance of a site. Note that the standard MCM formulation places no restrictions on the number of cases a site can service (or in this case, detect). In the real world, however, surveillance sites cannot detect an infinite number of cases, as each site will have some established natural limit, for example, in terms of the number of patients it can service. Such site capacity constraints are explicitly modeled in the capacitated MCM formulation where each site is endowed with some intrinsic integer capacity. The K-median model (sometimes also referred to as the P-median model) minimizes the sum of the distances from each individual to the individual's nearest site.

Which of these algorithms produces the "best" set of surveillance sites? Are there conditions where the K-median model is "better" than the MCM (or vice versa)? Does either algorithm provide "better" site selection than the existing Iowa Department of Public Health (IDPH) network? We can answer many of these questions empirically by simulating the spread of influenza across the state of Iowa and calculating the probability of each case being detected by any surveillance site. Our dataset consists of two million de-identified Medicaid billing records representing eight complete influenza seasons from July 2000 to June 2008. These records comprise all of the Iowa Medicaid records from this time period that contain any one of 30 pre-specified ILI ICD-9 codes. We treat the Medicaid dataset as the record of all influenza cases that occurred in Iowa between 2000 and 2008. The probability of case detection is determined by the Huff model, a probabilistic model often used in geography literature to analyze and understand aggregate behavior.

We first analyze outbreak intensity and show that networks our algorithms design always outperform the existing IDPH network. In fact, our networks are able to detect the same number of cases that the existing network can detect using many fewer surveillance sites. To analyze outbreak timing, we correlate the percent difference time series for the detected cases and the full dataset of cases. We show that when algorithmically selecting surveillance sites, the size of the network plays only a minimal role in the quality of the outbreak timing detection. Selecting just a few strategically-placed surveillance sites is enough to reliably and accurately determine the onset, peak, and end of the influenza season in the state of Iowa.

The major contribution of this work is the introduction and study of two different metrics on which a surveillance network can be optimized: outbreak intensity and outbreak timing. We show that our methods allow us to design surveillance networks which match or beat the performance of the existing IDPH network using fewer, more strategically-placed, surveillance sites.
Chaos and Allee effect on a discrete-time logistic population model

Ms. DOUCETTE, Sarah

1 Texas Tech University

Corresponding Author: sarah.doucette@ttu.edu

Chaos is a term used in biological population systems that often carries implications of randomness. Instead, the accepted mathematical consensus considers a system to be in chaos only if this system displays the following three characteristics: topological mixing (different parts of a graph are superimposable on others), a deterministic nature, and sensitivity to initial conditions. Chaos occurs in a discrete logistic system as the growth rate $r$ increases beyond approximately 2.692. Using MATLAB, three programs were developed to model the population size of a discrete logistic model. These programs model population size versus growth rate, time, and previous population size in order to study the changing patterns in population size and clearly display each of the conditions of chaos. The values of $r$ for which a discrete logistic system bifurcates into 2, 3, 4, and 8 cycles were numerically derived. An investigation was then conducted that examined how complications like the addition of an Allee effect (positive density dependence) to the system affects the $r$ values at which the system bifurcates. Adding an Allee effect to the system increased the $r$ values at which the system bifurcated, implying increased system stability.

M2_Modeling for Biosurveillance and Risk Assessment of Emerging Infectious Diseases / 52

A national assessment of West Nile virus risk integrating climate and ecology.

Dr. JUSTIN, Davis

1 Tulane University

Corresponding Author: jdavis37@tulane.edu

Symposium: Modeling for biosurveillance and risk assessment of emerging infectious diseases.

Many attempts to characterize West Nile virus infections in humans use local, proprietary data sources yet these studies consistently report similar correlates. Additionally, after an apparent drop in numbers of cases reported to the CDC in 2008-2011, cases in 2012 rose perhaps unexpectedly. Here we consider a family of national models conditioned on publicly available data. These more general models accord with local models and demonstrate that the recent spike in cases is likely a straightforward consequence of simple climatological indices. Alternative formulations of the distributed lag to characterize weather data are considered.
M2_Modeling for Biosurveillance and Risk Assessment of Emerging Infectious Diseases / 53

Data Assimilation for Epidemic Models

Dr. HICKMANN, Kyle 1

1 Tulane University

Corresponding Author: khickma@tulane.edu

Minisymposium: "Modeling for Biosurveillance and Risk Assessment of Emerging Infectious Diseases"

To forecast disease spread using a complex epidemic model one must estimate both the current state of the model and an optimal parametrization. These must be estimated from public health data sources that are both sparse and, perhaps, only indirectly related to actual infection levels. In this talk I will detail the application of methods such as the Ensemble Kalman Filter and Sequential Monte Carlo to iteratively update epidemic model predictions. With these methods an epidemic simulation has its parametrization and current state adjusted as data becomes available. This allows an optimal model forecast to be derived that accounts for data and model error simultaneously.

M5_Chemotactic Modeling of Inflammatory Type Diseases / 54

Mathematical model of immune system response on the foreign body.

Dr. JICHEN YANG, Jichen Yang 1; Prof. SU, Jianzhong 2; Mrs. PERKINS, Larrissa 3; Prof. TANG, Liping 4; Prof. IBRAGIMOV, Akif 5

1 Department of Mathematics, University of Texas at Arlington, Arlington, TX 76019, USA
2 Professor and Chair Department of Mathematics The University of Texas at Arlington Arlington, Texas 76019 USA
3 Department of Mathematics The University of Texas at Arlington
4 The University of Texas at Arlington Arlington
5 TTU

Corresponding Author: akif.ibraguimov@ttu.edu

Implanted foreign body trigger immunological and inflammatory response from surrounding cells. The foreign body-mediated tissue responses may result in varying degrees of fibrotic tissue formation. There is an intensive research interest in the area of wound healing modeling, and quantitative methods are proposed to systematically study the behavior of this complex system of multiple cells, proteins and enzymes. However, the detailed mechanisms of macrophage responses, recruitment and activation, in foreign body reactions are not totally understood. Coincidentally, mathematical models have been proposed to systematically decipher the behavior of this complex system of multiple cells, proteins and biochemical processes in wound healing responses. Based on these early works, this study introduces a mathematical model in two spatial dimensions to investigate the transient behavior of macrophages, fibroblasts and their interactions during the formation of fibrotic tissue. We find that the simulation results are consistent with the experimental observations. These findings support that the model can reveal quantitative insights for studying foreign body reaction processes.
Death 1 Latency and the Effects on Predictability of Zombie Virus Spread

TYSON, Austin 1; Dr. WILLIAMS, Brock 1; CALHOUN, John 1
1 Texas Tech University

Corresponding Author: austin.tyson@ttu.edu

Zombie diseases provide a means of exploring new tools in virology without the typical restraints of real-life diseases such as lack of information or lack of control over the experiment. By studying a virus with no real-life context, all of the restraints of real-life diseases can be avoided. The zombie disease model used for this research was the Calhoun-Williams model of the zombie virus. This model employs random walks to imitate the analogous spread of diseases among human populations. A computer simulation was used to create a virtual city and disease wherein specific factors can be observed and manipulated. The factors explored by this simulation were infectivity of disease as an expression of the population’s resistance, initial population (both total and infected), shape of the city, and latency periods. This researcher focused on the trend between Death 1 latency (a measure of the time between recovering from a disease and becoming infected by the same disease again) and overall predictability of viral spread. This relationship can be seen in the coalescence of population graphs over time when the Death 1 latency period is modified.

Herbaceous Species Invasions in a Fire-Dominated Habitat: a Mathematical Simulation Model

Ms. HEARNS, Jessica 1; Mr. NEVAI, Andrew 2; Mr. QUINTANA-ASCENCIO, Pedro 1; Mr. MENGES, Eric 3
1 University of Central Florida Department of Biology
2 University of Central Florida Department of Mathematics
3 Archbold Biological Station

Corresponding Author: hearnsj@knights.ucf.edu

Presents a fire dominated density dependent growth model for various plants in Florida scrub habitat.
Deterministic and Stochastic Models of Bacteria-Phage Dynamics

Dr. VIDURUPOLA, Sukhitha 1; Dr. ALLEN, Linda 1
1 Texas Tech University

Corresponding Author: sukhitha.vidurupola@ttu.edu

Bacteriophages, more commonly known as phages, are viruses that kill bacteria. Phages are used to treat food or animals infected with bacteria, thereby killing the bacteria. Phages attach to a bacterium, inject their DNA or RNA, multiply inside the bacterium, then burst from the bacterium, releasing many new phage particles. Mathematical models for bacteria-phage dynamics that account for uninfected bacteria, B, two stages for phage-infected bacteria, L and I, and phage particles P are formulated and analyzed. The models are deterministic and stochastic, a system of ordinary differential equations, a Markov chain model, and a multitype branching process. The basic reproduction number in the deterministic model defines a threshold in terms of model parameters. If this threshold is less than one, phage cannot persist and will not kill the bacteria. This same threshold applies to the stochastic models. In addition, the branching process provides an estimate of the probability of successful phage growth when the threshold exceeds one. Numerical results applied to a particular bacteria-phage system illustrate the importance of initial phage density, phage death rate, and phage burst size in controlling bacterial infections.

Evolutionary dynamics of ecological systems with a component Allee effect

Mr. UDIANI, Oyita 1; Prof. KANG, Yun 1
1 Arizona State University

Corresponding Author: oudiani@asu.edu

We investigate, using continuous trait evolutionary game theory, ways in which natural selection can change the outcome of a single species predator-prey dynamics when prey are subject to component Allee effects such as mating limitations or predation satiation. One of the more interesting findings from our model is the potential for multiple evolutionary attractors. Thus, depending on initial species abundance and trait distribution, selection may favor traits that promote (or suppress) demographic Allee effects highlighting fitness tradeoffs that could be especially consequential in extinction-prone populations. Preliminary results on a co-evolutionary model with a generalist predator and type II functional response are also discussed.
Competing pathways in protein aggregation. Prion protein (PrP) polymerization

Dr. PRIGENT, Stéphanie 1; Ms. HAFFAF, Wafaâ 2; Dr. DOUMIC-JAUFFRET, Marie 2; Dr. REZAEI, Human 3

1 INRIA-UPMC-INRA
2 INRIA-UPMC
3 INRA

Corresponding Author: stephanie.prigent@inria.fr

Though aggregation of proteins into amyloid fibrils is involved in more than twenty neurodegenerative diseases, incorporation of additional proteins on fibril remains misunderstood. To clarify these mechanisms, kinetics of aggregation of units on fibrils were studied experimentally. We performed then an analytical and numerical approach using the ODE system from Becker-Döring as a basis. Our first model that considers one single compartment of fibrils does not reproduce the observed biological behaviour where a loss of aggregation ability is observed. Our second model involves a second compartment with fibrils unable to incorporate more units and can be seen on a biological point of view as e.g. a structural defect of fibrils upon monomer addition. This model leads to kinetic coefficients biologically plausible and correctly simulates the first experimental steps for prion aggregation. Such system of aggregation with a dead-end pathway can be applied not only to certain other aggregative proteins as tubulin, but also to other fields such as proofreading of DNA replication.

Models and Optimal Controls of Vancomycin-Resistant Enterococci with Colonization, Infection and Prevention

YAHDI, Mohammed 1

1 Ursinus College

Corresponding Author: myahdi@ursinus.edu

The rising prevalence of vancomycin-resistant enterococci (VRE) is a major health problem in many intensive care units (ICU) because of its association with increased mortality and high costs. We present a mathematical framework for determining cost-effective strategies for prevention and treatment of VRE infections and outbreaks in the ICU. Models are developed based on a colonization, infection, prevention and treatment stages using deterministic, Stochastic as well as continuous and discrete-time Markov chains approaches. The models encompass the uncertainty of the parameter values and transitions. In particular, critical health conditions are linked to the effectiveness on reducing VRE infections, mortality rates and the basic reproduction number, and results are compared to clinical data. Control variables representing special prevention, such as daily chlorhexidine baths, VRE treatment, compliance rate and antibiotic use are incorporated into an optimal control analysis to minimize VRE-related deaths and costs associated with treatment and prevention controls over a finite time period. Numerical solutions illustrate optimal single and dual allocations of the controls for various cost values. Results show that preventive care has the greatest impact in reducing the basic reproductive number, while treatment of VRE infections has the most impact in reducing VRE-related deaths.
M5_Chemotactic Modeling of Inflammatory Type Diseases / 61

Chaotic patterns in a chemotaxis-haptotaxis model of cancer invasion

Prof. THOMAS, Hellen ¹
¹ University of Alberta, Canada

Corresponding Author: akif.ibragimov@ttu.edu

In 2006 Chaplain and Lolas proposed a chemotaxis-haptotaxis model for cancer invasion into healthy tissue. Numerical solutions of this model show interesting spatial patterns in the wake of the invasion front. Together with M. Winkler and K.J. Painter we prove, that these patterns are organized by a chemotaxis model with logistic growth term. In my talk I will further analyse these patterns and show that, indeed, they show a form of spatio-temporal chaos.

M1_Persistence and Permanence in Multi-Species Population Models / 62

Bifurcation and global stability of nonlinear semelparous Leslie matrix models

Dr. KON, Ryusuke ¹
¹ Faculty of Engineering, University of Miyazaki

Corresponding Author: ryusuke.kon@gmail.com

This work studies the dynamics of an n-stage nonlinear semelparous Leslie matrix model. The model has a positive equilibrium point if and only if the basic reproduction number $R_0$ is greater than unity. The main concern of this work is the global stability of the positive equilibrium point. Our main result gives a sharp condition under which the positive equilibrium point is globally asymptotically stable near the bifurcation point $R_0=1$. This result affirmatively solves the conjecture posed by Cushing (2009). Our approach uses a certain Lotka-Volterra differential equation to approximate the discrete dynamics of nonlinear semelparous Leslie matrix models. The way of this approximation was first proposed by Diekmann and van Gils (2009). We see that our approach has a potential application to show that the approximation gives a faithful description of the dynamics of nonlinear semelparous Leslie matrix models even if there are no stable positive equilibrium points.

This is a joint work with Josef Hofbauer (University of Vienna).
Target Reproduction Number and Application to Infectious Disease Control

SHUAI, Zhisheng 1; Dr. VAN DEN DRIESSCHE, Pauline 2; Dr. HEESTERBEEK, Hans 3
1 University of Central Florida
2 University of Victoria
3 University of Utrecht

Corresponding Author: shuai@ucf.edu

A new quantity called the target reproduction number is defined to measure control strategies for infectious diseases with multiple host types such as waterborne, vector-borne and zoonotic diseases. The target reproduction number includes as a special case and extends the type reproduction number to allow disease control strategies that target contacts between host types. Relationships among the basic, type and target reproduction numbers are established. Several disease models in the literature and new cholera models are used to illustrate the applications of the target reproduction number.

In the Minisymposium "Recent Advances in Mathematical Epidemiology and Ecology".

Spatiotemporal mutualistic model of mistletoes and birds

Prof. SHI, Junping 1; Dr. LIU, Rongsong 2; WANG, Chuncheng 3; DEL RIO, Carlos 2
1 College of William and Mary
2 University of Wyoming
3 Harbin Institute of Technology

Corresponding Author: jxshix@wm.edu

A mathematical model which incorporates the spatial dispersal and interaction dynamics of mistletoes and birds is derived and studied to gain insights of the spatial heterogeneity in abundance of mistletoes. Fickian diffusion and chemotaxis are used to model the random movement of birds and the aggregation of birds due to the attraction of mistletoes respectively. The spread of mistletoes by birds is expressed by a convolution integral with a dispersal kernel. Two different types of kernel functions are used to study the model, one is Dirac delta function which reflects one extreme case that the spread behavior is local, and the other one is a general non-negative symmetric function which describes the nonlocal spread of mistletoes. When the kernel function is taken as the Dirac delta function, the threshold condition for the existence of mistletoes is given and explored in term of parameters. For the general non-negative symmetric kernel case, we prove the existence and stability of non-constant equilibrium solutions. Numerical simulations are conducted by taking specific forms of kernel functions. Our study shows that the spatial heterogeneous patterns of the mistletoes are related to the specific dispersal pattern of the birds which carry mistletoe seeds. This is a joint work with Chuncheng Wang, Rongsong Liu, Carlos Martinez del Rio (University of Wyoming).
Innate Dispersal in *T. confusum*: Using Laboratory Studies to Develop a Metapopulation Model

HOLMES, Kody \(^1\); OLLIVER, Perry \(^1\); TAYLOR, Chalet \(^2\); Dr. NAGY, John \(^3\)

\(^1\) Arizona State University
\(^2\) Scottsdale Community College
\(^3\) School of Mathematical and Statistical Sciences at Arizona State University; Department of Life Sciences at Scottsdale Community College

**Corresponding Author:** holmes.kody@gmail.com

Dispersal is a key life history trait in many vertebrates, especially those living in metapopulations—populations comprising discrete subpopulations connected by dispersal. Among the best known mammalian metapopulations is the American pika population (*Ochotona princeps*) inhabiting ore dumps in the ghost mining town of Bodie, California. Members of this lab have studied the evolution of dispersal in this metapopulation in the field and with models for decades. Nevertheless, key model elements—particularly individual dispersal probability, its functional relationship with patch density, dispersal survival probability and its functional relationship with distance dispersed—have proven nearly impossible to estimate precisely. Therefore, we have developed artificial metapopulations of confused flour beetles (*Tribolium confusum*) to examine unclear behavioral attributes of dispersal under controlled conditions. Here we show that dispersal propensity in *T. confusum* is "innate," that is, it is neither a function of number of opportunities to disperse nor a function of local patch density. Our artificial metapopulations comprise 5 patches—a central "hub" connected to 4 identical patches by tubes through which beetles could disperse. Both stochastic and deterministic mathematical models built under the assumption that beetles dispersed with a fixed probability (per capita) regardless of which patch they were in (central hub with 4 dispersal corridors vs. outer patches with only 1) fit the data very well, but models assuming beetles disperse with probability proportional to the number of outlets did not. We also varied initial density between 50 and 300 beetles with 35 g of media per patch, which had no impact on per capita dispersal probability. These results dovetail with previous studies performed by John C. Ogden, who in the 1970's showed that variation in dispersal amongst *Tribolium* spp. was genetic.

M5_Chemotactic Modeling of In ammatory Type Diseases / 66

Approximate Analysis to the Fisher Equation

ZHAOSHENG, Feng \(^1\)

\(^1\) Department of Mathematics, University of Texas—Pan American, Edinburg, TX 78539

**Corresponding Author:** akif.ibraguimov@ttu.edu

There is the widespread existence of wave phenomena in ecology and biology. In this talk, we give an approximate analysis on traveling wave solutions to a generalized Fisher equation, a partial differential equation used as a deterministic version of a stochastic model for the spatial spread of a favored gene in a population. We provide a connection between the Abel equation of the first kind and the Fisher equation, and present two integral forms of the associated Abel equation with the small initial condition. By virtue of the integral forms and the Banach Contraction Mapping Principle we derive the approximate expansion of traveling wave solutions in the Banach space, and use the approximate formula to construct blow-up solutions accordingly.
Sonification of EEGs

EHNIS, Kate 1; PHILIP, Stacy 1; Dr. SEAQUIST, Carl 1; RAMIREZ, Frederick 2; Dr. GONIK, Renato 2

1 TTU
2 TTUHSC

Corresponding Author: stacy.philip@ttu.edu

Ten patient Electroencephalogram (EEG) recordings were selected from a study conducted at a Lubbock hospital. These recordings include 8 normal and 2 abnormal EEGs, which were stripped of personal identifying information. Recent publications indicate that sonification (converting data to sound) allows the human ear to analyze series data and detect irregularities that might otherwise go unnoticed. Since brain rhythms are typically lower than the human hearing range, signal-processing techniques, including but not limited to modulation, Fourier transforms, wavelet analysis, and digital filtering, will be applied to convert EEGs to sound. Our objective is to demonstrate that in addition to traditional visual analysis, auditory acuity may be useful in the analysis of EEGs and aid in the early detection of abnormal EEG activity. The project will be a success if an algorithmic approach to sonification leads to the identification of important features of the EEGs by listening to the transformed signals.

C3_Immunology / 68

Modeling the role of altruism of antibiotic-resistant bacteria

Prof. ZOU, Xingfu 1

1 University of Western Ontario

Corresponding Author: xzou@uwo.ca

Based on the new findings in a recent experimental study [Lee et al, Nature, 467 (2010), pp.82-86] that antibiotic resistant mutants of bacteria produce indoles to protect the wild strain bacteria, we propose a mathematical model to describe the interaction of the wild strain, resistant strain and indoles with limited nutrient. We distinguish two cases: (i) mutation is negligible and a resistant strain pre-exists; (ii) mutation is not negligible. For (i), we establish conditions for co-persistence of both strains, which indicate that the wild strain can survive with the help from the altruistic resistant strain, whereas it dies out in the absence of such a benefit. This consolidates the experimental findings in Lee et al (2010). Further analysis and simulations also reveal some new phenomena that are not reported in Lee et al (2010), that is, periodic oscillations of the populations may occur within certain range of the parameters, and there exists bistability in the sense that a stable positive periodic solution coexists with a stable positive equilibrium. This is a joint work with Wendi Wang.
Systems Biology of Epidemiology: From Genes to Environment

Dr. GUTIERREZ, Juan B. ¹
¹ University of Georgia

Corresponding Author: juan@math.uga.edu

The traditional epidemiological approach to characterize transmission of infectious disease consists of compartmentalizing hosts into susceptible, exposed, infected, recovered (SEIR), and vectors into susceptible, exposed and infected (SEI), and variations of this paradigm (e.g. SIR, SIR/SI, etc.). Compartmentalized models are based on a series of simplifying assumptions and have been successfully used to study a broad range of disease transmission dynamics. These paradigm is challenged when the within-host dynamics of disease is taken into account with aspects such as: (i) Simultaneous Infection: An infection can include the simultaneous presence of several distinct pathogen genomes, from the same or multiple species, thus an individual might belong to multiple compartments simultaneously. This precludes the traditional calculation of the basic reproductive number. (ii) Antigenic diversity and variation: Antigenic diversity, defined as antigenic differences between pathogens in a population, and antigenic variation, defined as the ability of a pathogen to change antigens presented to the immune system during an infection, are central to the pathogen’s ability to 1) infect previously exposed hosts, and 2) maintain a long-term infection in the face of the host immune response. Immune evasion facilitated by this variability is a critical factor in the dynamics of pathogen growth, and therefore, transmission.

This talk explores an alternate mechanistic formulation of epidemiological dynamics based upon studying the influence of within-host dynamics in environmental transmission. A basic propagation number is calculated that could guide public health policy.

The interaction of migratory birds and domestic poultry and its role in sustaining avian influenza

Dr. LIU, Rongsong ¹
¹ University of Wyoming

Corresponding Author: rongsong.liu@uwyo.edu

We investigate the role of migratory birds in the spread of H5N1 avian influenza, focusing on the interaction of a migratory bird species with nonmigratory poultry. The model is of patch type and is derived with the aid of reaction-advection equations for the migratory birds in the air along the flyways. Poultry may reside at some or all of the four patches of the model, which consist of the breeding patch for the migratory birds, their winter feeding patch, and two stopover patches where birds rest and refuel on their migration. Outward and return migratory routes can be different. The equations for the migratory birds contain time delays which represent the flight times for migratory birds along particular sectors. Delays also appear in the model coefficients via quantities which represent flight survival probabilities for the various sectors. We establish results on positivity, boundedness, global asymptotic stability of the disease-free equilibrium, and the persistence of infection. We also discuss extensions of the model to include the seasonality of the migration phenomenon. Numerical simulations support the analytical findings; here we used data on H5N1 infected ducks in the Poyang Lake region of China.
Stability Analysis of a Model for Foreign Body Fibrotic Reactions

Prof. IBRAGIMOV, Akif 1; Prof. SU, Jianzhong 2; Ms. OWENS, Larrissa 3; Prof. TANG, Liping 4

1 Texas Tech University
2 University of Texas at Arlington
3 UTA
4 University of Texas at Arlington, USA

Corresponding Author: akif.ibraguimov@ttu.edu

Implanted medical devices often trigger immunological and inflammatory reactions from surrounding tissues. The foreign body-mediated tissue responses may result in varying degrees of fibrotic tissue formation. There is an extensive research interest in the area of wound healing modeling, and quantitative methods are proposed to systematically study the behavior of this complex system of multiple cells, proteins, and enzymes. This paper introduces a kinetics-based model for analyzing reactions of various cells/proteins and biochemical processes as well as their transient behavior during the implant healing in 2-dimensional space. In particular, we provide a detailed modeling study of different roles of macrophages and their effects on fibrotic reactions. The main mathematical result indicates that the stability of the inflamed steady state depends primarily on the reaction dynamics of the system. However, if the said equilibrium is unstable by its reaction-only system, the spatial diffusion and chemotactic effects can help to stabilize when the model is dominated by classical and regulatory macrophages over the inflammatory macrophages. The mathematical proof and counter examples are given for these conclusions.

Structured models for transmission dynamics of \emph{Mycobacterium marinum}

Dr. SUTTON, Karyn 1

1 University of Louisiana at Lafayette

Corresponding Author: sutton@louisiana.edu

\emph{Mycobacterium marinum} (Mm), a genetically similar bacterium to \emph{Mycobacterium tuberculosis}, affects a number of fish industries (fisheries, aquaculture, aquariums, and research stocks) on a comparable scale to tuberculosis in humans. Because of this, and the practical advantages of working with animal models as opposed to humans, Mm infections in recently established fish models provide a unique opportunity for the study of mycobacterial infections. We present two recently developed models of transmission dynamics of \emph{Mycobacterium marinum} in aquatic environments: (i) fish dynamics structured by the bacterial load or infection severity of the animal, and (ii) fish dynamics structured by size of the animal. Recent efforts have shown that effective transmission necessarily involves consumption of an infected host or a source of bacteria to ensure “activation” of Mm into a highly infectious state. Therefore, both are models of transmission within a food web, but each are particularly well suited to account for different sources of variability. The bacterial load structured model allows for differences in behavior as the infection progresses from a chronic (and seemingly asymptomatic), to an acute (symptomatic) state. This is a crucial component as the large chronically infected pool of fish are thought to play a similar important role in the persistence of these infections as do the large chronic pool of humans afflicted with TB. The size structured model allows one to consider metabolic differences, which likely play a key role in a food web. We present preliminary simulation results from both models, illustrating the distinct benefits of each formulation. We also show agreement between model solutions and experimentally collected data in a laboratory setting. We further present this framework as a useful tool to address key questions such as design in experimental settings and potential control strategies in large-scale situations.
Finite difference approximations for measure-valued solutions of a hierarchically size-structured population model

Prof. ACKLEH, Azmy; Mr. CHELLAMUTHU, Vinodh; Prof. KAZUFUMI, Ito

1 University of Louisiana at Lafayette
2 North Carolina State University

Corresponding Author: ackleha@gmail.com

We consider a quasilinear hierarchically size-structured population model formulated by Ackleh and Ito in 2005. In this model the growth, mortality and reproduction rates are assumed to depend on a function of the population density. The solution to this model can become singular (measure-valued) in finite time even if all the vital rates are smooth. In this paper we develop an implicit first-order finite difference scheme to compute the measure-valued model solution. Convergence analysis for this method is provided. We also present a high resolution second order explicit scheme to compute the solution of the model. Numerical simulations indicate that the second order method is superior in resolving solution-singularities.

Differential impact of sickle cell trait on symptomatic and asymptomatic malaria

Prof. SHIM, Eunha; Prof. FENG, Zhilan; Prof. CASTILLO-CHAVEZ, Carlos

1 University of Tulsa
2 Purdue University
3 Arizona State University

Corresponding Author: eunha-shim@utulsa.edu

Individuals who carry the sickle cell trait (S-gene) have a greatly reduced risk of experiencing symptomatic malaria infections. However, previous studies suggest that the sickle cell trait does not protect against acquiring asymptomatic malaria infections, although the proportion of symptomatic infections is up to 50% in areas where malaria is endemic. To examine the differential impact of the sickle cell trait on symptomatic and asymptomatic malaria, we developed a mathematical model of malaria transmission that incorporates the evolutionary dynamics of S-gene frequency. Our model indicates that the fitness of sickle cell trait is likely to increase with the proportion of symptomatic malaria infections. Our model also shows that control efforts aimed at diminishing the burden of symptomatic malaria are not likely to eradicate malaria in endemic areas, due to the increase in the relative prevalence of asymptomatic infection, the reservoir of malaria. Furthermore, when the prevalence of symptomatic malaria is reduced, both the fitness and frequency of the S-gene may decrease. In turn, a decreased frequency of the S-gene may eventually increase the overall prevalence of both symptomatic and asymptomatic malaria. Therefore, the control of symptomatic malaria might result in evolutionary repercussions, despite short-term epidemiological benefits.
A branching process model of prion dynamics

OLOFSSON, Peter 1; SINDI, Suzanne 2
1 Trinity University
2 UC Merced

Corresponding Author: polofsso@trinity.edu

Prions are infectious agents composed of misfolded proteins, responsible for illnesses such as mad cow disease in cattle and Creutzfeldt–Jakob disease in humans. We create a branching process model for yeast cells to describe how prions grow inside the cell and how they are transmitted from mother to daughter cell. We compare our model predictions to simulated data and use it to estimate parameters.

Age-structure model with periodically distributed delay

Dr. WANG, Xiang-Sheng 1
1 Southeast Missouri State University

Corresponding Author: xswang@semo.edu

We study an age-structure system with periodically distributed delay. Using a standard linear chain trick, we obtain an infinite dimensional ordinary differential system with periodic coefficients. Successive approximation can be applied to derive an asymptotic expansion for the solution. It remains an open and interesting problem to find an explicit formula for the basic reproduction number of the periodic system.

The LAZARUS Simulation Computer

Mr. CALHOUN, John 1; Dr. WILLIAMS, Brock 2
1 Texas Tech
2 Texas Tech University

Corresponding Author: john.m.calhoun@ttu.edu

The Texas Tech Math department is building a beowulf cluster named LAZARUS, through a joint effort between several grant programs at Texas Tech, for the purpose of outreach and for testing large scale simulations of viral spread such as in cities of several million people. The Computer specifically leverages the power of modern graphics processing units, we will outline the hardware of the system, how the simulation runs across 24 computers, and what kind of simulations will be well suited to LAZARUS.
Persistent Risk factors for Hyperendemic Visceral Leishmaniasis in Two Different Eco-Epidemiologically Different Regions

BARLEY, Kamal 1; Dr. MUBAYI, Anuj 2
1 Arizona State University
2 Northeastern Illinois University

Corresponding Author: kbarley@asu.edu

Visceral Leishmaniasis (VL) has been on the rise in the two highest prevalent regions: India and Sudan. Similar hosts and parasite characteristics are observed in these two countries but variance in estimates of epidemiological quantities are different. WHO aims to eliminate the disease by 2020, however, various unknown risk factors and undetected high incidence of asymptomatic cases may pose a barrier in achieving the target for the two countries. A vector-host epidemic model, accounting for asymptomatic and treated individuals, is developed to compare and contrast the mechanisms that contribute to the level of risk posed for VL for the two nations. The analysis of the model is uniquely determined by the basic reproductive number, $R_0$, measuring the transmission potential of the Leishmania parasite. Various public health department reports and data sets, including data from the literature, were thoroughly reviewed to obtain estimates of demographical and epidemiological parameters related to the two populations. Using these estimates, we conducted uncertainty and sensitivity analyses to assess the most influential risk factors that contribute to the high endemic levels of VL observed in Sudan and India.

Targeted Biology for Biological Control: Identifying Vulnerabilities in the Life History of the Invasive Adelges tsugae (Homoptera, Adelgidae) using Dynamic Life-History Simulations

Dr. TROTTER, Robert T. 1
1 USDA Forest Service, NRS, Yale School of Forestry and Environmental Studies

Corresponding Author: linda.j.allen@ttu.edu

The sustainability of hemlock (Tsuga) as a forest resource in the eastern United States is threatened by the invasive hemlock woolly adelgid (Adelges tsugae, Annand), a small piercing-sucking herbivore native to Asia and western North America. Here we present a simulation of the hemlock woolly adelgid life-cycle based on field studies to evaluate the potential impacts of predators, pathogens, and climatic variability on adelgid population dynamics. Simulations indicate that the two documented density dependent life stages of the hemlock woolly adelgid provide a buffering effect that makes population densities resilient to adelgid mortality in several life stages. However, subtle changes in the nature of the density dependence and the timing of adelgid mortality factors may have a large impact on the efficacy of biological controls.
Modeling the Dynamics of Woody Plant-Herbivore Interactions with Age-Dependent Toxicity

Dr. LIU, Rongsong ¹

¹ University of Wyoming

Corresponding Author: linda.j.allen@ttu.edu

In this paper we study the effects that woody plant chemical defenses may have on interactions between boreal hares that in winter feed almost entirely on twigs. We focus particularly on the fact that toxin concentration often varies with the age of twig segments. The model incorporates the fact that early in the growth of twigs, segments are often highly defended by toxins and are, therefore, highly unpalatable to hares. But, in the second year of twig growth, the toxin concentration of older twig segments begins to decline increasing the palatability of their biomass. This age-dependent toxicity of twig segments is modeled using age-structured model equations which are reduced to a system of delay differential equations involving multiple delays in the woody plant - hare dynamics. A novel aspect of the modeling was that it had to account for mortality of non-consumed younger twig segment biomass when older twig biomass was bitten off and consumed. Basic mathematical properties of the model are established together with upper and lower bounds on the solutions. Necessary and sufficient conditions are found for the linear stability of the equilibrium in which the hare is extinct, and sufficient conditions are found for the global stability of this equilibrium. Numerical simulations confirmed the analytical results and demonstrated the existence of limit cycles over ranges of parameters reasonable for hares browsing on woody vegetation in boreal ecosystems. This showed that age dependence in plant chemical defenses has the capacity to cause hare - plant population cycles, a new result.
An Enzymatic Model of Prion Aggregate Dynamics

Mr. DAVIS, Jason ¹; Dr. SINDI, Suzanne ²
¹ University of California, Merced
² UC Merced

Corresponding Author: jdavis8@ucmerced.edu

Prion proteins are responsible for a variety of neurodegenerative diseases in mammals such as Creutzfeldt-Jakob disease in humans and "mad-cow disease" (Bovine Spongiform Encephalopathy or BSE) in cattle. While these diseases are fatal to mammals, a host of harmless phenotypes have been associated with prion proteins in the yeast S. cerevisiae, making yeast an ideal model organism for prion diseases.

According to the prion hypothesis, misfolded versions of a protein appear and form prion aggregates, complexes of multiple misfolded proteins which range in size from tens to hundreds of proteins. The misfolded (prion) state is infectious and spreads to healthy proteins by conversion of the healthy confirmation to the misfolded state. (During this process, the newly misfolded protein joins the complex and the aggregate increases in size by one protein monomer.) Prion aggregates may also increase in number due to fragmentation, thus increasing the number of templates which act to convert healthy proteins.

The dynamics of prion aggregates have been investigated with a number of mathematical models. Most models assume that the fragmentation rate is proportional to the size of the aggregate; however, experimental data does not support this assumption. Instead, it suggests the presence of an enzymatic limitation to the fragmentation process. We generalize the mathematical models to include an additional protein that has been identified as the limiter, perform general analyses of the model, then compare it with experimental data.

Modeling Mammalian Geographic Ranges Using Georeferenced Environmental Data and Mathematical Models

Mr. BOVIO, Richard Stephen ¹; Dr. RIBBLE, David ²; Dr. ELAYDI, Saber ²; Dr. HASFURA, Roberto ²; Ms. WALLACE, Shalee ¹
¹ Research Student
² Professor

Corresponding Author: swallac3@trinity.edu

Species distribution models attempt to predict the presence of a given species based on a number of environmental features within its geographic range. These models are critical for conservation efforts and the potential impact of climate change. In this study, we expand upon previous species distribution models to include population growth rate and competition, as well as traditional climate parameters in order to predict the geographic range of a small mammal, Peromyscus maniculatus. Population growth rates were estimated from 60 years of georeferenced collection efforts across the United States made available through Natural History Museum data bases (Manis). We have tested various models incorporating rainfall and temperature, and results from these models will be presented.