Frontiers of Mathematical Biology: Modeling, Computation and Analysis

May 2-4, 2018
University of Central Florida
Orlando, Florida, United States

Co-sponsored by

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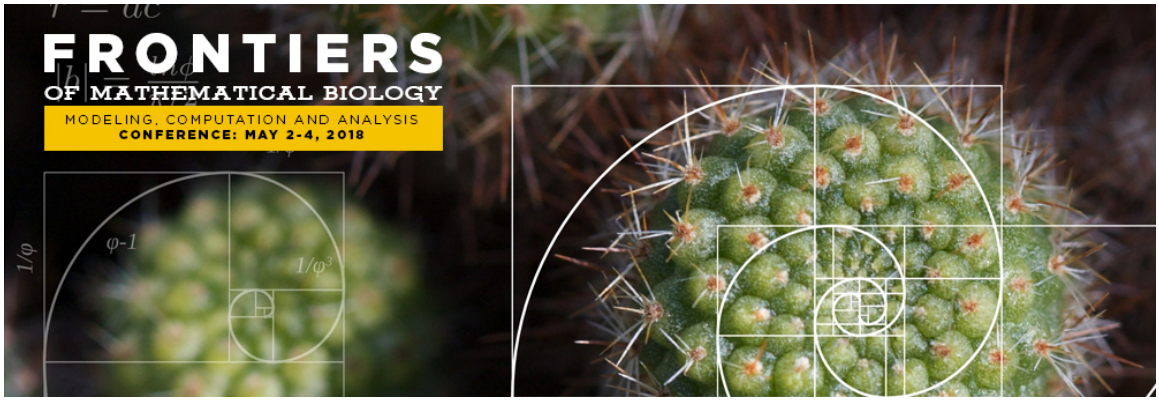

The conference "*Frontiers of Mathematical Biology: Modeling, Computation and Analysis*" is held on the Main Campus of the University of Central Florida, Orlando, FL during May 2-4, 2018. The theme is on modeling, computation and analysis in mathematical biology. Conference topics include cancer modeling and computation, pattern formation and neurosciences, mathematical ecology and epidemiology, and other related fields.

Scientific Committee

- Chris Cosner, University of Miami
- Suzanne Lenhart, University of Tennessee
- Yuan Lou, The Ohio State University
- Hal Smith, Arizona State University
- Pauline van den Driessche, University of Victoria, Canada
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- Maia Martcheva, University of Florida
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- National Science Foundation
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Department of Mathematics

Dear FMB Conference Participants,

It is my pleasure to welcome you to the Conference on Frontiers of Mathematical Biology: Modeling, Computation and Analysis.

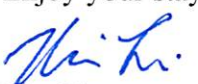
The mission of the Department of Mathematics at the University of Central Florida is to offer the opportunity for excellence and development to students with visionary and relevant programs of high quality at the undergraduate and graduate levels and to advance mathematical research to benefit society. To accomplish this mission the faculty and the staff of the department have set high goals. We aim to provide the best undergraduate mathematics program in the state of Florida. We offer doctoral and masters programs that encourages student to develop into mathematicians. We also foster a dedication to word-class research by faculty and students at all levels in a broad variety of areas of mathematics.

Mathematics is in the broadest sense the study of structure. Through quantification, relationships, and modeling, mathematics provides us with the intellectual structural framework that permits us to develop an understanding of the phenomena that envelops us. The skills that are developed in mathematical training have broad application in the workplace. Graduates with degrees in mathematics are well prepared for a variety of careers and are in high demand from employers who require analytical skill, data organization, and problem solving. Training in mathematics also earns the approbation of admission committees for legal, medical, and other professional training. This means that graduates in mathematics are able to pursue job careers with high salaries and high job-satisfaction.

It is from these efforts that the future begins.

On behalf of the Organizing Committee, I invite you to make the most of this conference by not only attending talks in a variety of research areas but also by reconnecting with friends and collaborators and meeting new colleagues. Also, I encourage you to take a walk together and explore the many different parts of our campus.

Enjoy your stay with us!



Xin Li
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Frontiers of Mathematical Biology: Modeling, Computation and Analysis

May 2-4, 2018, University of Central Florida, Orlando, Floirda, United States

Conference Schedule

	Wednesday (May 2)			Thursday (May 3)			Friday (May 4)		
8:00-9:00	Registration (8:00-10:00) in CB2 204 Welcome Remarks (9:00-9:10) in CB2 201			Registration (8:00-10:00) in CB2 204			Registration (8:00-10:00) in CB2 204		
	CB2 201			CB2 201			CB2 201		
9:00-10:00	Wei-Ming Ni			Bard Ermentrout			Jianhong Wu		
10:00-10:30	Coffee Break			Coffee Break			Coffee Break		
	CB2 201	CB2 206	CB2 207	CB2 201	CB2 206	CB2 207	CB2 201	CB2 206	CB2 207
10:30-11:00	Stephanie Portet	Mansoor Haider	Fred Brauer	Jonathan Rubin	Winfried Just	Bingtuan Li	Guihong Fan	Azmy Ackleh	Connell McCluskey
11:00-11:30	Gianni Arioli	Yang Li	Wenjing Zhang	Pei Liu	Harsh Jain	Xueying Wang	Biao Tang	Leonid Hanin	Jing Li
11:30-12:00	Xiulan Lai	Qimin Huang	Joseph Tien	Chunhua Shan	Yiyuan Wang	Jonathan Welker	Evan Milliken	Xue Zhang	Necibe Tuncer
12:00-12:30	Xingfu Zou	Naveen Vaidya	Gail Wolkowicz	Trachette Jackson	Paula Grajdeanu	Wenxian Shen	Jia Zhao	Xianghong Zhang	Shigui Ruan
12:30-14:00	Lunch Break			Lunch Break			Lunch Break		
	CB2 201			CB2 201			CB2 201		
14:00-15:00	Peter Bates			Robert Gatenby			Qiang Du		
15:00-15:30	Coffee Break			Coffee Break			Coffee Break		
	CB2 201	CB2 206	CB2 207	CB2 201	CB2 206	CB2 207	CB2 201	CB2 206	CB2 207
15:30-16:00	H. Thomas Banks	Chris Cosner	Rongsong Liu	Shangbing Ai	Igor Belykh	Maia Martcheva	Xuefeng Wang	Wei Lin	Eric Numfor
16:00-16:30	Xiaoying Wang	Veronica Ciocanel	Cameron Browne	Xiaoxia Xie	Xiao Yu	Yu Jin	Xiaoming Zheng	Richard Schugart	Qiuyi Su
16:30-17:00	Yan Wang	Evan Haskell	Yanyu Xiao	Qingyan Shi	Xi Huo	Chengcheng Huang	Steve Cantrell	Zhaosheng Feng	Elena Braverman
17:00-17:30	Rebecca Everett	Fan Bai	Yixiang Wu	Qiliang Wu	Calistus Nhonghala	Hayriye Gulbudak	Panel Session		
17:30-18:00	Yaqin Feng	Sophia Jang	Junping Shi	Heiko Enderling	Partha Srinivasan	Julien Arino	Discussions and Concluding Remarks		
18:00-19:00	Welcome Reception/Poster Session (18:15-19:30)			Cash bar starts at 18:30			Conference concludes at 18:00		
19:00-21:00				Banquet (19:00-21:00) in Cape Florida Room, 2nd floor, Student Union					

* All conference talks are held on the 2nd floor of Classroom Building 2 (CB2)

Daily Schedule
Wednesday May 2nd

	8:00-9:00	Registration desk opens (CB2 204)
	9:00-9:10	Welcome Ceremony (CB2 201)

PLENARY TALK

Page	Room: CB2 201	Session Chair: Xin Li
12	9:10-10:10	**Wei-Ming Ni**, *Interaction of diffusion and spatial heterogeneity in ecology*
	10:10-10:30	COFFEE BREAK

PARALLEL TALKS

	Room: CB2 201	Session Chair: Xiulan Lai
35	10:30-11:00	**Stephanie Portet**, *Transport of intermediate filaments in cells*
14	11:00-11:30	**Gianni Arioli**, *Existence and stability of traveling pulse solutions of the FitzHugh-Nagumo equation*
29	11:30-12:00	**Xiulan Lai**, *Mathematical modeling about the synergy and antagonism of combination drugs with immune checkpoint inhibitors and anti-tumor drug*
51	12:00-12:30	**Xingfu Zou**, *Modeling the role of white-tailed deer in geographic spread of the black-legged tick Ixodes scapularis by a spatially nonlocal model*

PARALLEL TALKS

	Room: CB2 206	Session Chair: Qimin Huang
23	10:30-11:00	**Mansoor Haider**, *Nonlinear elastic vessel wall models for studying pulmonary hypertension in cardiovascular networks*
30	11:00-11:30	**Yang Li**, *Stage-structured discrete-time models for interacting wild and sterile mosquitoes and their global dynamics*
25	11:30-12:00	**Qimin Huang**, *Modeling the effect of antibiotic exposure on the transmission of Methicillin-resistant Staphylococcus aureus in hospitals with environmental contamination*
42	12:00-12:30	**Naveen Vaidya**, *Modeling the effects of antibody responses on HIV dynamics under drugs of abuse*

Wednesday May 2nd (cont.)

PARALLEL TALKS

Page	Room: CB2 207	Session Chair: Wenjing Zhang
16	10:30-11:00	**Fred Brauer**, *Early estimates of epidemic final size*
48	11:00-11:30	**Wenjing Zhang**, *Multiple attractors in the simple epidemic model*
41	11:30-12:00	**Joseph Tien**, *Disease spread on a dynamic multi-layer network: limit laws*
45	12:00-12:30	**Gail Wolkowicz**, *Growth on two limiting essential resources in a self-cycling fermentor*
	12:30-14:00	LUNCH BREAK

PLENARY TALK

	Room: CB2 201	Session Chair: Steve Cantrell
10	14:00-15:00	**Peter Bates**, *Gradient dynamics: motion near a manifold of quasi-equilibria*
	15:00-15:30	COFFEE BREAK

PARALLEL TALKS

	Room: CB2 201	Session Chair: Yan Wang
15	15:30-16:00	**H. Thomas Banks**, *Optimal Control of Immunosuppressants in Renal Transplant Recipients Susceptible to BKV Infection*
42	16:00-16:30	**Xiaoying Wang**, *Turing patterns in a predator-prey model with seasonality*
44	16:30-17:00	**Yan Wang**, *Persistence and extinction of population in reaction-diffusion-advection model with strong Allee effect growth*
20	17:00-17:30	**Rebecca Everett**, *Parameter estimation for modeling intermittent androgen suppression therapy in prostate cancer patients*
21	17:30-18:00	**Yaqin Feng**, *Steady states of lattice population models with immigration*

Wednesday May 2nd (cont.)

PARALLEL TALKS

Page	Room: CB2 206	Session Chair: Veronica Ciocanel
19	15:30-16:00	**Chris Cosner**, *Ideal free dispersal in time periodic environments*
18	16:00-16:30	**Veronica Ciocanel**, *Modeling microtubule-based transport in the frog oocyte*
24	16:30-17:00	**Evan Haskell**, *Predator-mediated coexistence with attractive or repulsive prey-taxis*
15	17:00-17:30	**Fan Bai**, *Probability of a major infection in a stochastic within-host model for viral infection with multiple latent and infectious stages*
27	17:30-18:00	**Sophia Jang**, *Dynamics of tumor-CD4$^+$-cytokine-host cells interactions with treatments*

PARALLEL TALKS

	Room: CB2 207	Session Chair: Yixiang Wu
32	15:30-16:00	**Rongsong Liu**, *Age-dependent intraspecific competition in pre-adult life stages and its effects on adult population dynamics*
17	16:00-16:30	**Cameron Browne**, *Models of dynamic virus and immune response networks*
47	16:30-17:00	**Yanyu Xiao**, *Mathematical modeling for machine tool vibration*
46	17:00-17:30	**Yixiang Wu**, *Spatial spread of epidemic diseases in geographical settings: seasonal influenza epidemics in Puerto Rico*
38	17:30-18:00	**Junping Shi**, *Effect of spatial average on the spatial-temporal pattern formation of reaction-diffusion systems*

	18:15-19:30	WELCOME RECEPTION / POSTER SESSION
52		**Barrett Brister**, *Multistabillity of synchronized clusters in networks of phase oscillators*
52		**Ilia Ilmer**, *Two-species competition with directed diffusion and harvesting*
53		**Benjamin Letson**, *Invariant angular manifolds in the Goodwin oscillator*
53		**Jonathan Tyler**, *Revisiting a synthetic intracellular regulatory network that is sufficient for oscillations*

Thursday May 3rd

	8:00-9:00	Registration desk opens (CB2 204)

PLENARY TALK

Page	Room: CB2 201	Session Chair: Yuanwei Qi
11	9:00-10:00	**Bard Ermentrout**, *Follow your nose: the mathematics of olfactory navigation*
	10:00-10:30	COFFEE BREAK

PARALLEL TALKS

	Room: CB2 201	Session Chair: Pei Liu
36	10:30-11:00	**Jonathan Rubin**, *Getting our feet wet: rivers in planar flows and neuronal models*
31	11:00-11:30	**Pei Liu**, *on-Isothermal electrokinetics: energetic variational approach*
37	11:30-12:00	**Chunhua Shan**, *Complex dynamics and bifurcations in a toxin-dependent aquatic population model*
26	12:00-12:30	**Trachette Jackson**, *Multiscale modeling accurately predicts in-vivo response of combined IL-6 blockade and traditional chemotherapy in stem cell driven cancers*

PARALLEL TALKS

	Room: CB2 206	Session Chair: Yiyuan Wang
28	10:30-11:00	**Winfried Just**, *Why are biological systems so messy, and how can mathematicians cope?*
27	11:00-11:30	**Harsh Jain**, *Data and identifiability in a model of cancer treatment*
44	11:30-12:00	**Yiyuan Wang**, *Dynamical models for estimating the population sizes for Culex mosquitoes using the weekly trap counts*
22	12:00-12:30	**Paula Grajdeanu**, *Mathematical biologist, data scientist, or both?*

Thursday May 3rd (cont.)

PARALLEL TALKS

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29	10:30-11:00	**Bingtuan Li**, *Multiple invasion speeds in a two-species integro-difference competition model*
43	11:00-11:30	**Xueying Wang**, *Stochastic models of Bovine Babesiosis with juvenile cattle*
45	11:30-12:00	**Jonathan Welker**, *A new immuno-epidemiological model of Visceral Leishmaniasis in dogs*
38	12:00-12:30	**Wenxian Shen**, *Front propagation dynamics in chemotaxis models with Logistics source on R^N*
	12:30-14:00	LUNCH BREAK

PLENARY TALK

Room: CB2 201 | Session Chair: Maia Martcheva

11	14:00-15:00	**Robert Gatenby**, *Evolution-based models in cancer biology and treatment*
	15:00-15:30	COFFEE BREAK

PARALLEL TALKS

Room: CB2 201 | Session Chair: Qingyan Shi

13	15:30-16:00	**Shangbing Ai**, *Traveling wave solutions for predator-prey systems*
47	16:00-16:30	**Xiaoxia Xie**, *Nonlocal differential equations and convergence to the classical differential equations*
39	16:30-17:00	**Qingyan Shi**, *Hopf bifurcation in a reaction?diffusion equation with distributed delay and Dirichlet boundary condition*
46	17:00-17:30	**Qiliang Wu**, *Quenched flow in symmetric multi-component FCH*
19	17:30-18:00	**Heiko Enderling**, *Harnessing tumor-immune interactions to trigger systemic effects after radiotherapy*

Thursday May 3rd (cont.)

PARALLEL TALKS

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16	15:30-16:00	**Igor Belykh**, *Foot force models of crowd dynamics on a wobbly bridge*
48	16:00-16:30	**Xiao Yu**, *Dynamics of populations with individual variation in dispersal on bounded domains*
25	16:30-17:00	**Xi Huo**, *Dynamical behaviors of an antimicrobial deescalation model*
34	17:00-17:30	**Calistus Ngonghala**, *Ecology meets economic growth: a general dynamic model framework*
39	17:30-18:00	**Partha Srinivasan**, *Distributions of proteins – models and experiments*

PARALLEL TALKS

Room: CB2 207 | Session Chair: Chengcheng Huang

32	15:30-16:00	**Maia Martcheva**, *Backward bifurcation and oscillations in a nested immuno-eco-epidemiological model*
28	16:00-16:30	**Yu Jin**, *The spread of the invasive alga Codium fragile driven by long-distance dispersal of buoyant propagules*
24	16:30-17:00	**Chengcheng Huang**, *Circuit models of low dimensional cortical variability*
22	17:00-17:30	**Hayriye Gulbudak**, *Heterogeneous viral strategies promote co-existence in virus-microbe systems*
14	17:30-18:00	**Julien Arino**, *Role of community structure and household size on the propagation of tuberculosis*
	18:30-19:00	CASH BAR (Cape Florida Room, 2nd floor, Student Union)
	19:00-21:00	BANQUET (Cape Florida Room, 2nd floor, Student Union)

Friday May 4th

	8:00-9:00	Registration desk opens (CB2 204)

PLENARY TALK

Page	Room: CB2 201	Session Chair: Shigui Ruan
12	9:00-10:00	**Jianhong Wu**, *Complexity of co-feeding transmission dynamics*
	10:00-10:30	COFFEE BREAK

PARALLEL TALKS

	Room: CB2 201	Session Chair: Evan Milliken
20	10:30-11:00	**Guihong Fan**, *Global Hopf bifurcation in a compartmental model with delays for tick population*
40	11:00-11:30	**Biao Tang**, *Optimal dengue vaccination to mitigate Zika cases*
34	11:30-12:00	**Evan Milliken**, *The role of outcome preferences in optimizing heterogenous disease control strategies*
50	12:00-12:30	**Jia Zhao**, *Computational modeling of Cytokinesis of Eukaryotes driven by F-actin enriched contractile ring"*

PARALLEL TALKS

	Room: CB2 206	Session Chair: Xianghong Zhang
13	10:30-11:00	**Azmy Ackleh**, *Changes in population dynamics resulting from evolutionary response to an environmental disturbance*
23	11:00-11:30	**Leonid Hanin**, *Metastasis suppression by the primary tumor: a natural law*
50	11:30-12:00	**Xue Zhang**, *Understanding the impact of diapause and co-feeding on tick-borne disease spread*
49	12:00-12:30	**Xianghong Zhang**, *Models to assess the effects of Wolbachia-carrying mosquito augmentations and mating competition on the control of dengue disease*

Friday May 4th (cont.)

PARALLEL TALKS

Page | Room: CB2 207 | Session Chair: Jing Li

33	10:30-11:00	**Connell McCluskey**, *Disease models that include immigration*
30	11:00-11:30	**Jing Li**, *Species interactions modify the spread of vector-borne pathogens independent of transmission mode*
41	11:30-12:00	**Necibe Tuncer**, *Structural and practical identifiability analysis of Zika epidemiological models*
36	12:00-12:30	**Shigui Ruan**, *On an advection-reaction-diffusion competition system with double free boundaries modeling invasion and competition of Aedes Albopictus and Aedes Aegypti mosquitoes*
	12:30-14:00	LUNCH BREAK

PLENARY TALK

Room: CB2 201 | Session Chair: Andrew Nevai

10	14:00-15:00	**Qiang Du**, *Nonlocal models of diffusion, their computation and applications*
	15:00-15:30	COFFEE BREAK

PARALLEL TALKS

Room: CB2 201 | Session Chair: Xiaoming Zheng

43	15:30-16:00	**Xuefeng Wang**, *Using effective boundary conditions to model fast diffusion on a road in a large field*
51	16:00-16:30	**Xiaoming Zheng**, *A mathematical model of angiogenesis and tumor growth: analysis and application in anti-angiogenesis therapy*
18	16:30-17:00	**Robert Stephen Cantrell**, *A PDE model of intraguild predation with cross-diffusion*

Friday May 4th (cont.)

PARALLEL TALKS

Page	Room: CB2 206	Session Chair: Richard Schugart
31	15:30-16:00	**Wei Lin**, *Causation network constructions*
37	16:00-16:30	**Richard Schugart**, *Using a mathematical model with individual patient data to quantify differences between patients with diabetic foot ulcers*
21	16:30-17:00	**Zhaosheng Feng**, *Approximate analysis to degenerate reaction-diffusion systems*

PARALLEL TALKS

	Room: CB2 207	Session Chair: Qiuyi Su
35	15:30-16:00	**Eric Numfor**, *Management strategies in a malaria model combining human and transmission-blocking vaccines*
40	16:00-16:30	**Qiuyi Su**, *Periodic solutions of abstract semilinear equations with applications to biological models*
17	16:30-17:00	**Elena Braverman**, *On state and cycle stabilization for discrete models of population dynamics*
	17:00-17:30	Panel Session (CB2 201)
	17:30-18:00	Discussions & Concluding Remarks (CB2 201)

Abstracts of Plenary Talks

May 2, 14:00-15:00, Room 201

Gradient dynamics: motion near a manifold of quasi-equilibria

Peter Bates
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This concerns general gradient-like dynamical systems in Banach space with the property that there is a manifold along which solutions move slowly compared to attraction in the transverse direction. Conditions are given on the energy (or, more generally, Lyapunov functional) that ensure solutions starting near the manifold stay near for a long time or even forever. Applications are given with the vector Allen-Cahn and Cahn-Morral systems.

Joint work with G. Fusco and G. Karali.

May 4, 14:00-15:00, Room 201

Nonlocal models of diffusion, their computation and applications

Qiang Du
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Nonlocality is ubiquitous in nature. We present some nonlocal models that can be used to study many problems ranging from flying patterns of bumblebees to propagating paths of cracks in materials. We discuss some recent results on the development of a common mathematical framework for the analysis and computation of these nonlocal models.

May 3, 9:00-10:00, Room 201

Follow your nose: the mathematics of olfactory navigation

Bard Ermentrout
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Olfaction (the sense of smell) is the oldest of our sensory modalities and has been used for millions of years for animals to find mates, find food, avoid predators, etc. In a large multi-investigator collaboration, we have begun to try to understand the algorithms animals use to navigate complex odor landscapes. I will describe several simple algorithms that use spatial and temporal information about the odor to locate its source. Underlying these simple algorithms is some interesting nonlinear dynamics. I will discuss the continuous dynamics of binaral search where the organism uses the concentration differences between two sensors to steer toward the source. Depending on the odor environment, various types of complex dynamics emerge including stable fixed points, periodic orbits, torii, and chaos. I will also show that even this simple algorithm works quite well in real odor plumes. Secondly, I will describe a discrete algorithm where the animal samples the concentrations at different time points and uses this comparison to determine the heading. By reducing this algorithm to its very simplest form, we are able to also analyze the underlying dynamics. Finally, we will show the role of "noise" on improving the algorithms and how it can be leveraged as a search strategy.

May 3, 14:00-15:00, Room 201

Evolution-based models in cancer biology and treatment

Robert Gatenby
Moffitt Cancer Center
robert.gatenby@moffitt.org

A number of successful systemic therapies are available for treatment of disseminated cancers. However, tumor response to these treatments is almost invariably transient and therapy fails due to emergence of resistant populations. The latter reflects the temporal and spatial heterogeneity of the tumor microenvironment as well as the evolutionary capacity of cancer phenotypes to adapt to therapeutic perturbations. Interestingly, although cancers are highly dynamic systems, cancer therapy is typically administered according to a fixed, linear protocol. Treatment is changed only when the tumor progresses but successful tumor adaptation begins immediately upon administration of the first dose. Applying evolutionary models to cancer therapy demonstrate the potential advantage of using more dynamic, strategic approaches that focus not just on the initial cytotoxic effects of treatment but also on the evolved mechanisms of cancer cell resistance and the associated phenotypic costs. The goal of evolutionary therapy is

to prevent or exploit emergence of adaptive tumor strategies. Examples of this approach include adaptive therapy and double bind therapy. The former continuously alters therapy to maintain a stable tumor volume using a persistent population of therapy-sensitive cells to suppress proliferation of resistant phenotypes. The latter uses the cytotoxic effects of an initial therapy to promote phenotypic adaptations that are then exploited using follow-on treatment. In preclinical models, application of adaptive therapy permits indefinite tumor control with a single cytotoxic drug. Ongoing clinical trials using cancer treatment protocols based on mathematical models of evolutionary principles will be presented.

May 2, 9:00-10:00, Room 201

Interaction of Diffusion and Spatial Heterogeneity in Ecology

Wei-Ming Ni
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In this lecture I wish to report some recent advances in understanding, both mathematically and experimentally, the effect of diffusion in ecology; in particular, how interaction between diffusion and spatial heterogeneity affects carrying capacity which plays a central role in competition of species.

May 4, 9:00-10:00, Room 201

Complexity of co-feeding transmission dynamics

Jianhong Wu
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Co-feeding has been recognized as an important transmission route in vector-borne diseases but little has been done in terms of modeling and analyzing the co-feeding transmission dynamics. Here we develop a mathematical model for the vector-host population dynamics where the distribution pattern of vector-over-host is governed by the vector attachment and host grooming behaviors. We show this coupled system of vector-within-host distribution and vector-host interaction exhibits bi-stability and nonlinear oscillation in a very natural way.

Abstracts of Parallel Talks

May 4, 10:30-11:00, Room 206

Changes in population dynamics resulting from evolutionary response to an environmental disturbance

Azmy Ackleh
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Prolonged exposure to a disturbance such as a toxicant has the potential to result in rapid evolution of toxicant resistance in many short-lived species. This evolution may allow a population to persist at higher levels of the toxicant than is possible without evolution. Here we apply evolutionary game theory to Leslie matrix models to obtain Darwinian equations that couple population and evolutionary dynamics. We consider two cases for which evolution of toxicant resistance may have important dynamic consequences. In the first case, we examine how persistence outcomes for surrogate species may change when one species is able to persist by evolving toxicant resistance while another is not. In the second case, we consider how evolution of toxicant resistance may impact both predator and prey when a prey species evolves in response to a toxicant but the predator does not due to different time scales.

May 3, 15:30-16:00, Room 201

Traveling wave solutions for predator-prey systems

Shangbing Ai
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In this talk a recent result on the existence of traveling wave solutions for a general class of predator-prey systems with delays will be presented. The proof of this theorem will be outlined, and its applications to concrete systems will be discussed.

May 3, 17:30-18:00, Room 207

Role of community structure and household size on the propagation of tuberculosis

Julien Arino
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Some northern communities in Canada experience high tuberculosis incidence rates, comparable with those in much poorer countries and much higher than in the rest of Canada. There are a variety of factors at play. We focus here on the relative roles of crowding and social structure, by considering a discrete time model with transmission within and between households.

This is joint work with Ryan Sherbo.

May 2, 11:00-11:30, Room 201

Existence and stability of traveling pulse solutions of the FitzHugh-Nagumo equation

Gianni Arioli
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One of the parameters of the FitzHugh-Nagumo model is the ratio epsilon of two time scales, which takes values between 0.001 and 0.1 in typical simulations of nerve axons. Based on the existence of a (singular) limit at epsilon = 0, it has been shown that the FitzHugh-Nagumo equation admits a stable traveling pulse solution for sufficiently small epsilon > 0. Here we prove the existence of such a solution for epsilon = 0.01, both for circular axons and axons of infinite length. This is in many ways a completely different mathematical problem. In particular, it is non-perturbative and requires new types of estimates. Some of these estimates are verified with the aid of a computer. The methods developed in this paper should apply to many other problems involving homoclinic orbits, including the FitzHugh-Nagumo equation for a wide range of other parameter values.

May 2, 17:00-17:30, Room 206

Probability of a Major Infection in a Stochastic Within-Host Model for Viral Infection with Multiple Latent and Infectious Stages

Fan Bai
Texas Tech University
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Establishment or spread of a viral infection within healthy individuals depends on the amount of exposure to a viral source, either through virus particles or through cells that have been infected. In this investigation, we assume that a potential infection has reached the site of the healthy target cells. We apply stochastic in-host models and multitype branching processes to investigate the dynamics of a viral infection of target cells that has multiple latent and infectious stages. Our goal is to investigate the probability of a major infection after exposure to either virus particles or to cells in the latent or infectious stages. It is shown that the probability of a major infection is more likely if the virus has gained entry into the target cells or reached the latter stages of the latent infection. However, in some cases, the probability of a major infection is less likely if the introduced infected cells have reached the final stage. The theoretical estimates from the branching process are shown to have good agreement with numerical simulations of the full stochastic model.

May 2, 15:30-16:00, Room 201

Optimal Control of Immunosuppressants in Renal Transplant Recipients Susceptible to BKV Infection

H. Thomas Banks
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Individuals undergoing kidney transplants are put on a lifetime supply of immunosuppression to prevent an allograft rejection. However suboptimal immunosuppressive therapy can put a renal transplant recipient at a risk of infection. The key to a successful transplant is finding the optimal balance between over-suppression and under-suppression of the immune response, a task which is difficult to achieve in renal transplant patients due to the narrow therapeutic index of immunosuppressants. We use a mathmatical model to design a feedback control loop using Receding Horizon Control (RHC) methodology. Since data is not available for all model states we use NonLinear Kalman Filtering (specifically Extended Kalman Filter (EKF)) to estimate the states which do not have data available. Combining RHC and EKF we design an adaptive treatment plan which predicts the optimal immunosuppression therapy for renal transplant recipients.

May 3, 15:30-16:00, Room 206

Foot force models of crowd dynamics on a wobbly bridge

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Modern pedestrian and suspension bridges are designed using industry standard packages, yet disastrous resonant vibrations are observed, necessitating multimillion dollar repairs. Recent examples include pedestrian-induced vibrations during the opening of the Solfrino Bridge in Paris in 1999 and the increased bouncing of the Squibb Park Bridge in Brooklyn in 2014. The most prominent example of an unstable lively bridge is the London Millennium Bridge, which started wobbling as a result of pedestrian-bridge interactions. Pedestrian phase locking due to footstep phase adjustment is suspected to be the main cause of its large lateral vibrations; however, its role in the initiation of wobbling was debated. We develop foot force models of pedestrians response to bridge motion and detailed, yet analytically tractable, models of crowd phase locking. We use biomechanically inspired models of crowd lateral movement to investigate to what degree pedestrian synchrony must be present for a bridge to wobble significantly and what is a critical crowd size. Our results can be used as a safety guideline for designing pedestrian bridges or limiting the maximum occupancy of an existing bridge. The pedestrian models can be used as crash test dummies when numerically probing a specific bridge design. This is particularly important because the U.S. code for designing pedestrian bridges does not contain explicit guidelines that account for the collective pedestrian behavior.

May 2, 10:30-11:00, Room 207

Early estimates of epidemic final size

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Early in a disease outbreak it is important to be able to estimate the final size of an epidemic in order to assess needs for treatment and to be able to compare the effects of different treatment approaches. However, it is common for epidemics, especially of diseases considered dangerous, to grow much more slowly than expected. We suggest that by assuming behavioral changes in the face of an epidemic and heterogeneity of mixing in the population it is possible to obtain reasonable early estimates.

May 4, 16:30-17:00, Room 207

On state and cycle stabilization for discrete models of population dynamics

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For some values of parameters, simple discrete models (Ricker, logistic, Beverton-Holt) exhibit chaotic behavior. If a constant positive perturbation is introduced, some of the maps experience break of chaos, and a series of period halving bifurcations can eventually give birth to a stable two-cycle. However, similar stable cycles can be observed in discrete equations, once maps are modified to describe contest, not scramble, competition.

Recently, several methods were developed to stabilize otherwise unstable difference equations (proportional feedback, prediction based and target oriented controls). This is illustrated with vector modified applied to LPA (larvae-pupae-adults) model describing the flour beetle Tribolium castaneum life stages.

May 2, 16:00-16:30, Room 207

Models of dynamic virus and immune response networks

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The dynamics of virus and immune response within a host can be viewed as a complex and evolving ecological system. For example, during HIV infection, an array of CTL immune response populations recognize specific epitopes (viral proteins) presented on the surface of infected cells to effectively mediate their killing. However HIV can rapidly evolve resistance to CTL attack at different epitopes, inducing a dynamic network of viral and immune response variants. We consider models for the network of virus and immune response populations. Our analysis provides insights on viral immune escape from multiple epitopes. In the "binary allele" setting, we prove that if the viral fitness costs for gaining resistance to each of n epitopes are equal and multiplicative, then the system of 2^n virus strains converges to a "perfectly nested network" with less than $n+1$ persistent virus strains. Overall, our results suggest that immunodominance is the most important factor determining viral escape pathway of HIV against multiple CTL populations. To conclude, I briefly discuss ongoing collaborative work to connect the models with intra-host SIV/immune response data.

May 4, 16:30-17:00, Room 201

A PDE model of intraguild predation with cross-diffusion

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This talk concerns a quasilinear parabolic system modeling an intraguild predation community in a focal habitat in R^n for $n \geq 2$. In this system the intraguild prey employs a fitness-based dispersal strategy whereby the intraguild prey moves away from a locale when predation risk is high enough to render the locale undesirable for resource acquisition. The system modifies an earlier model considered by Ryan and Cantrell by adding an element of mutual interference among predators to the functional response terms in the model, thereby switching from Holling two forms to Beddington-DeAngelis forms. We show that the resulting system can be realized as a semi-dynamical system with a global attractor for any $n \geq 2$. In contrast, the original model was restricted to two dimensional spatial habitats. The permanence of the intraguild prey then follows as in Ryan and Cantrell by means of the Acyclicity Theorem of Persistence Theory.

May 2, 16:00-16:30, Room 206

Modeling microtubule-based transport in the frog oocyte

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In the development of egg cells into embryos, spatial differentiation is essential in determining the role of the new embryo cells in the growing organism. This spatial patterning often relies on asymmetric accumulations and transport of proteins in the oocyte. In the frog, messenger RNA (mRNA) dynamically switches between diffusion and active transport states in its journey to the periphery of the egg cell, where it accumulates and creates a spatial axis of development. Using dynamical systems modeling and analysis, we investigate the transport of mRNA and its dependence on cytoskeleton structures in the oocytes consisting of microtubules. Numerical studies using model microtubule structures allow us to predict that an anchoring mechanism at the cell periphery may be key in healthy development. I will also briefly discuss other active transport processes such as neurofilament transport along axons.

May 2, 15:30-16:00, Room 206

Ideal free dispersal in time periodic environments

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Determining which movement strategies are evolutionarily stable is a significant problem in evolutionary spatial ecology. In environments that vary in space but not in time, a variety of models predict that the movement strategies which are evolutionarily stable are those that can produce an ideal free equilibrium distribution of the populations using them. In that case a population has an ideal free distribution if all individuals have the same fitness and there is no net movement of individuals. This requires the population to exactly match the global distribution of resources. In reaction-diffusion-advection models and their nonlocal analogues, that can be achieved with strategies that use only local information. In this talk I will present results that extend the concept of an ideal free distribution to environments where the total amount of resources remains constant but their spatial distribution varies periodically in time. It appears that in the time periodic case achieving the analogue of an ideal free distribution using local movement by advection and diffusion requires the use of information that is nonlocal in space and time, such as spatial memory. Ideal free dispersal turns out to be evolutionarily stable in the time periodic setting.

May 3, 17:30-18:00, Room 201

Harnessing tumor-immune interactions to trigger systemic effects after radiotherapy

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Tumors grow within a host tissue that both facilitates progression by supplying nutrients and growth factors, and inhibits it through physical constraints and immune surveillance. Transformed cancer cells are confronted with this innate and adaptive immune surveillance, and tumors that develop to become clinically apparent have evolved to evade the immune system. Focal therapy activates an anti-tumor immune response that can propagate through the circulatory system. The systemic distribution of activated T cells is dependent on the anatomic distribution of metastatic sites, the tumor volume of each metastasis, the site of activation, and the local therapy protocol of immune activation. We have developed a mathematical model to decipher the complex local and systemic tumor-immune interactions. Tumor growth follows expansion of proliferating cells with site-dependent growth rate modulated by the predation of cytotoxic T cells. Cytotoxic T cells are recruited in response to tumor burden and proliferate in tissues in the presence of tumor-associated antigens. Local tumor-immune interaction dynamics are

combined with a spatially distributed model of blood flow through the system of large arteries in the human anatomy to simulate the blood flow fractions into each critical organ. The model shows metastatic tumors are highly interdependent, and changes in the nonlinear tumor-immune interactions in one tumor can perturb the systemic antitumor immune response, potentially facilitating spontaneous regression or aggressive outgrowth in distant sites. The clinical outcome of therapeutic intervention then depends on therapy-induced changes in tumor-immune dynamics both locally and systemically, as well as patient-specific initial conditions of the global disease.

May 2, 17:00-17:30, Room 201

Parameter Estimation for Modeling Intermittent Androgen Suppression Therapy in Prostate Cancer Patients

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Advanced prostate cancer is often treated by androgen suppression therapy, since prostate cells depend on androgens for proliferation and survival. To improve the patients' quality of life and possibly delay the development of resistance, intermittent androgen suppression (IAS) therapy can be given rather than continuous therapy. We consider a mathematical model of IAS therapy involving tumor cells, androgen, and the biomarker prostate-specific antigen and investigate parameter estimation with clinical data. Specifically, we implement iterative weighted least squares inverse problems to investigate the patient-specific parameters that can be confidently estimated.

May 4, 10:30-11:00, Room 201

Global Hopf Bifurcation in a Compartmental Model With Delays for Tick Population

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Transmitted by ticks, Lyme disease is an emerging infectious disease which can cause severe problems for human health. The reproduction and development of ticks are closely related to the environmental factors, in particularly the daily temperature. In this paper, we adopt and propose a three-stage population model for ticks with three delays to reflect the impact of average daily temperature on the different developmental stages. We present some local dynamics of the model through local analysis of equilibria which are related to the short term behavior of the population, but we are more interested in the average temperature in each of the development stages on the distribution of the ticks population. Based on the local analysis of Hopf bifurcation, we will focus

more on the global existence and dynamics of the Hopf bifurcation. Our results show that the tick population can persist in a region in a oscillatory manner if the sum of three developmental delays fall into the critical time delay interval. To illustrate our theoretical results on global Hopf bifurcation, we will present bifurcation diagrams using delays as bifurcation parameters as well as associate solutions of the model.

May 2, 17:30-18:00, Room 201

Steady states of lattice population models with immigration

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We consider the time evolution of the lattice subcritical Galton-Watson model with immigration. We prove Carleman type estimation for the cumulants in the simple case (binary splitting) and show the existence of a steady state. We also present the formula of the limiting distribution in a particular solvable case.

May 4, 16:30-17:00, Room 206

Approximate Analysis to Degenerate Reaction-Diffusion Systems

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In this talk, we study the case that some species migrate from densely populated areas into sparsely populated areas to avoid crowding, and investigate a more general reaction-diffusion system by considering density-dependent dispersion as a regulatory mechanism of the cyclic changes. Here the probability that an animal moves from the point x_1 to x_2 depends on the density at x_1. Under certain conditions, we apply the higher terms in the Taylor series and the center manifold method to obtain the local behavior around a non-hyperbolic point of codimension one in the phase plane, and use the Lie symmetry reduction method and Abel Operator to explore approximate wave solutions. Numerical simulation and biological explanation are presented.

May 3, 12:00-12:30, Room 206

Mathematical Biologist, Data Scientist, or both?

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Mathematical Biology and Data Science can be used to address practically any and all aspects of biology and medicine, from basic understanding of biological and physiological processes, to disease and therapy modeling. Whether to improve our understanding of the environment or to cure disease and maintain health, these two quantitative disciplines are now helping biosciences and healthcare move forward. However, mathematical modeling – in particular through deterministic models, and data science – in particular through machine learning, offer different approaches. In this talk I will discuss my experiences with both these approaches, pros and cons, differences and similarities, and what developments could be most helpful and most accessible to accelerate discovery science, empowering communities, and improve health and well being.

May 3, 17:00-17:30, Room 207

Heterogeneous Viral Strategies Promote Coexistence in Virus-Microbe Systems

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Viruses of microbes, including bacterial viruses (phage), archaeal viruses, and eukaryotic viruses, can influence the fate of individual microbes and entire populations. Here, we model distinct modes of virus-host interactions and study their impact on the abundance and diversity of both viruses and their microbial hosts. We consider two distinct viral populations infecting the same microbial population via two different strategies: lytic and chronic. A lytic strategy corresponds to viruses that exclusively infect and lyse their hosts to release new virions. A chronic strategy corresponds to viruses that infect hosts and then continually release new viruses via a budding process without cell lysis. The chronic virus can also be passed on to daughter cells during cell division. The long-term association of virus and microbe in the chronic mode drives differences in selective pressures with respect to the lytic mode. We utilize invasion analysis of the corresponding nonlinear differential equation model to study the ecology and evolution of heterogenous viral strategies. We first investigate stability of equilibria, and characterize oscillatory and bistable dynamics in some parameter regions. Then, we derive fitness quantities for both virus types and investigate conditions for competitive exclusion and coexistence. In so doing we find unexpected results, including a regime in which the chronic virus requires the lytic virus for survival and invasion.

May 2, 10:30-11:00, Room 206

Nonlinear elastic vessel wall models for studying pulmonary hypertension in cardiovascular networks

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Pulmonary hypertension (PH) is a rare but deadly cardiovascular disease. As PH advances, the relative composition of vessel wall constituents is altered. The ensuing wall stiffening increases blood pressure which, in turn, can induce further vessel wall remodeling. Yet, the precise manner in which these interactions occur is not well understood. Nonlinear hyperelastic continuum models, capturing structural remodeling of vessel walls in the presence of pulmonary hypertension (PH) are presented. These continuum models are developed in the context of 1D fluid-structure models of pulmonary cardiovascular networks. A Holzapfel-Gasser-Ogden (HGO)-type hyperelastic constitutive law for combined bending, inflation, extension and torsion of a nonlinear elastic tube is employed. Specifically, nonlinear relations between blood pressure and vessel wall cross-sectional area that reflect structural alterations with advancing PH are formulated and evaluated.

May 4, 11:00-11:30, Room 206

Metastasis Suppression by the Primary Tumor: A Natural Law

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We study metastatic cancer progression through an extremely general individual-patient mathematical model that is rooted in the contemporary understanding of the underlying biomedical processes yet is essentially free of specific biological assumptions of mechanistic nature. The model accounts for primary tumor growth and resection, shedding of metastases off the primary tumor and their selection, dormancy and growth in a given secondary site. However, functional parameters descriptive of these processes are assumed to be essentially arbitrary. In spite of such generality, the model allows for computing the distribution of site-specific sizes of detectable metastases in closed form. Under the assumption of exponential growth of metastases before and after primary tumor resection, we showed that, regardless of other model parameters and for every set of site-specific volumes of detected metastases, the model-based likelihood-maximizing scenario is always the same: complete suppression of metastatic growth before primary tumor resection followed by an abrupt growth acceleration after surgery. This scenario is commonly observed in clinical practice and is supported by a wealth of experimental and clinical studies conducted over the last 110 years. Furthermore, several biological mechanisms have been identified that could bring about suppression of metastasis by the primary tumor and accelerated vascularization and growth of metastases after primary tumor resection.

May 2, 16:30-17:00, Room 206

Predator-mediated coexistence with attractive or repulsive prey-taxis

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The interactions of species can have significant impact on species distribution and diversity. For example, resource competition between two species can lead to a competitive exclusion where one species is driven to extinction. However, it is known that the presence of a predator can mediate a coexistence between the competing species and the predator. How this predator mediated coexistence is influenced by other predator mediated effects is less studied. Here we compare the impact on predator mediated coexistence when the predator is either attracted or repulsed by one of the prey choices. We study a system of three populations involving two competing species with a common predator. All three populations are mobile via diffusion within a bounded spatial domain Ω, but the predator's movement is influenced by one prey's gradient representing either prey-taxis (attraction) or a prey defense mechanism that is repulsive to the predator which we term repulsive prey-taxis. We prove existence of positive solutions, and investigate pattern formation through a Turing style bifurcation analysis and numerical simulation.

This work is in collaboration with Jonathan Bell (University of Maryland Baltimore County).

May 3, 16:30-17:00, Room 207

Circuit models of low dimensional cortical variability

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Neuronal variability is a reflection of recurrent circuitry and cellular physiology, and its modulation is a reliable signature of cognitive and processing state. A pervasive yet puzzling feature of cortical circuits is that despite their complex wiring, population-wide shared spiking variability is low dimensional with all neurons fluctuating en masse. Previous model cortical networks are at loss to explain this variability, and rather produce either uncorrelated activity, high dimensional correlations, or pathologically network behavior. We show that if the spatial and temporal scales of inhibitory coupling match known physiology then model spiking neurons naturally generate low dimensional shared variability that captures in vivo population recordings along the visual pathway. Further, top-down modulation of inhibitory neurons provides a parsimonious mechanism for how attention modulates population-wide variability both within and between neuronal areas, in agreement with our experimental results. Our theory provides a critical and previously missing mechanistic link between cortical circuit structure and realistic population-wide shared neuronal variability.

May 2, 11:30-12:00, Room 206

Modeling the Effect of Antibiotic Exposure on the Transmission of Methicillin-resistant *Staphylococcus aureus* in Hospitals with Environmental Contamination

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Both deterministic and stochastic mathematical models are developed to explore the roles that antibiotic exposure and environmental contamination play in the transmission dynamics of nosocomial infections in hospitals. Uncolonized patients without or with antibiotic exposure, colonized patients without or with antibiotic exposure, uncontaminated and contaminated health-care workers, and free-living Methicillin-resistant *Staphylococcus aureus* (MRSA) are included in the models. Under the assumption that there is no admission of the colonized patients, the basic reproduction number R_0 is calculated. We prove that when $R_0 < 1$, the infection-free equilibrium is globally asymptotically stable; when $R_0 > 1$, the infection is uniformly persistent. Numerical simulations show that environmental cleaning is the most important intervention. Increasing the stay of colonized patients with antibiotic exposure in hospital will increase the prevalence of MRSA, which implies to treat patients with antibiotic exposure as efficiently and quickly as possible. Screening and isolating colonized patients at admission, and improving compliance with hand hygiene are also important control strategies.

May 3, 16:30-17:00, Room 206

Dynamical behaviors of an antimicrobial deescalation model

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Antimicrobial deescalation is a widely recommended antibiotic treatment strategy, but its advantages and trade offs in the reduction of antibiotic usage, control of ICU infections, and impact on bacteria resistance development were not well understood. We develop and analyze mathematical models to compare deescalation with the conventional drug use strategy, and conclude that deescalation is superior or inferior than the conventional drug strategy in different aspects and under different biological conditions. This talk will summarize the most recent mathematical analysis results.

May 3, 12:00-12:30, Room 201

Multiscale Modeling Accurately Predicts In-vivo Response of Combined IL-6 Blockade and Traditional Chemotherapy in Stem Cell Driven Cancers

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It is well known that growth and survival of cancer stem cells (CSCs) is highly influenced by tumor microenvironmental factors and molecular signaling, initiated by cytokines and growth factors. IL-6 is a key regulator of a number of cellular processes including proliferation, survival, differentiation, migration and invasion and it is also commonly overexpressed in many cancers. Recent evidence shows that IL-6 is not only secreted by tumor cells, but is produced at even higher levels by endothelial cells (ECs). Research shows that high intratumoral levels of IL-6 enhance the survival, self-renewal and tumor initiation potential of cancer stem cells. These studies of the impact of IL-6 on CSCs provide strong motivation for the development of anti-IL-6 therapies for the targeted treatment of stem cell driven cancers.

In this talk, a multi-scale mathematical model that operates at the intracellular, molecular, and tissue level is developed in order to investigate the impacts of endothelial cell-secreted IL-6 signaling on the crosstalk between tumor cells and ECs during tumor growth. This endothelial cell tumor cell (EC-TC) model is used to study the therapeutic impact of Tocilizumab (TCZ), a competitive IL-6R inhibitor, on tumor growth and cancer stem cell (CSC) fraction, alone and in combination with the traditional chemotherapeutic agent, Cisplatin. The approach used here is novel in that it includes full receptor occupancy dynamics between endothelial-cell produced IL-6, IL-6R, and TCZ. Validation is achieved by directly comparing model predictions to data generated by a series of in-vivo experiments. This multiscale approach provided excellent predictive agreement with the decrease in tumor volumes, as well as a decrease in CSC fraction.

Targeting key regulators of the cancer stem cell phenotype to overcome their critical influence on tumor growth is a promising new strategy for cancer treatment. This predictive modeling framework can serve to rapidly evaluate dosing strategies for IL-6 pathway modulation, as well as providing the basis for proposing combination treatments with IL-6 blockade and cytotoxic or other targeted therapies.

May 3, 11:00-11:30, Room 206

Data and identifiability in a model of cancer treatment

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Mathematical modeling has a long history in the field of cancer therapeutics, and there is increasing recognition that it can help uncover the mechanisms that underlie tumor response to treatment. However, making quantitative predictions with such models often requires parameter estimation from data, raising questions of parameter identifiability and estimability. Even in the case of structural (theoretical) identifiability, imperfect data and the resulting practical unidentifiability of model parameters can make it difficult to infer the desired information, and in some cases, to yield biologically correct inferences and predictions. Here, we examine parameter identifiability and estimability using a case study of two compartmental, ordinary differential equation models of cancer treatment with drugs that are cell cycle-specific (taxol) as well as non-specific (oxaliplatin). We proceed through model building, structural identifiability analysis, parameter estimation, practical identifiability analysis and its biological implications, as well as alternative data collection protocols and experimental designs that render the model identifiable. Despite the models being structurally identifiable, we show that without consideration of practical identifiability, incorrect incorrect biological predictions can result. We illustrate the usefulness of estimating practically identifiable combinations in generating biologically meaningful insights. These results highlight the importance of understanding the underlying mechanisms rather than purely using parsimony or information criteria/goodness-of-fit to decide model selection questions.

May 2, 17:30-18:00, Room 206

Dynamics of tumor-CD4$^+$-cytokine-host cells interactions with treatments

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Mathematical models of interactions between tumor cells, CD4$^+$ T cells, cytokines, and host cells are proposed to investigate the role of CD4$^+$ on tumor regression. Our results suggest that host cells along with the mechanism of production of CD4$^+$ T cells play important roles in driving tumor dynamics. Cancer cells can be eradicated if the tumor has a small growth rate and is also not competitive. Treatments by either CD4$^+$, cytokines, or a combination of the two are applied to study their effectiveness. It is concluded that doses of treatments along with the tumor size are critical in determining the fate of the tumor. Tumor cells can be eliminated completely if doses of treatments by cytokine are large. The treatments are in general more effective if the tumor size is smaller. Bistability is observed in all of the models with or without the treatment strategies indicating that there is a window of opportunity for clearing off the tumor cells.

May 3, 16:00-16:30, Room 207

The spread of the invasive alga Codium fragile driven by long-distance dispersal of buoyant propagules

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The secondary spread of an invasive species after initial establishment is a major factor in determining its distribution and impacts. In this study we constructed an individual-based model for the spread of the invasive green alga Codium fragile ssp. fragile along a straight coastline, in order to understand the factors governing spreading speed. Codium can spread locally through non-buoyant propagules, while long-distance dispersal depends on the wind-driven dispersal of buoyant fragments. Since fragment buoyancy is determined by light conditions, we first modelled the buoyancy of fragments, yielding a dispersal time dependent on light conditions. We then used this dispersal time, along with empirical wind speeds and directions to calculate a dispersal kernel for fragments. Finally, we incorporated this dispersal kernel into a population growth model including survival rate and fragmentation rate, to calculate a population spreading speed. We obtained the spreading speeds under current environmental conditions along the east coast of Canada and also conducted a sensitivity analysis to investigate the potential influence of environmental shifts associated with climate change on the spread of Codium.

May 3, 10:30-11:00, Room 206

Why are biological systems so messy, and how can mathematicians cope?

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This talk will describe some past and some ongoing work of the presenter and a number of his collaborators on approximating continuous-time models of biological systems with simpler discrete-time systems and on studying the latter. Several open problems that in the presenter's opinion may lead to methodological advances will be outlined.

May 2, 11:30-12:00, Room 201

Mathematical modeling about the synergy and antagonism of combination drugs with immune checkpoint inhibitors and anti-tumor drug

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There has been much progress in recent years in developing checkpoint inhibitors, primarily PD-1 antibodies and PD-L1 antibodies. However, because of lack of tumor-infiltrating effector T cells, many patients in clinical trials do not respond to checkpoint inhibitor treatment. It was recently suggested that the combination of an immune checkpoint inhibitor and another anti-tumor drug, such as a cancer vaccine or BRAF inhibitor, may function synergistically to induce more effective antitumor immune responses. In this work, we considered the combination therapies of cancer with a checkpoint inhibitor and a cancer vaccine (or BRAF/MEK inhibitor) using mathematical models. Cancer vaccine activates dendritic cells so that they induce more T cells to infiltrate the tumor. BRAF kinase, is a key part of MAPK pathway of cancer cell proliferation. BRAF-targeted therapy induces significant responses in the majority of patients. We use mathematical models with systems of partial differential equations to explore the efficacy of the two drugs and compare the simulations with data from mouse experiments. The synergy map of combinations of an anti-PD-1 and a cancer vaccine shows that for optimal efficacy under MTD constraint, the level of dosage of anti-PD-1 should be related to the level of dosage of cancer vaccine as indicated by the optimal dose curve in the map. In contrast, the efficacy map of combination of an anti-PD-1 and a BRAF/MEK inhibitor shows that at large doses the drugs may become antigonistic: an increase in one of the drugs may actually result in an increase in the tumor volume.

May 3, 10:30-11:00, Room 207

Multiple Invasion Speeds in a Two-Species Integro-Difference Competition Model

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We discuss an integro-difference competition model for the case that two species consecutively invade a habitat. We show that if a species spreads into a traveling wave of its rival, or if two species expand their spatial ranges in both directions, in a direction where open space is available, the species with larger invasion speed can always establish a wave moving into open space with its own speed. We demonstrate that when one species is stronger in competition, under appropriate conditions, the speeds at which the boundaries between two species move can be analytically determined. In general there are multiple invasion speeds in the model. It is

possible for a species to develop two separate waves propagating with different invasion speeds. It is also possible for each species to establish a single wave spreading with distinct speeds in both directions.

May 4, 11:00-11:30, Room 207

Species interactions modify the spread of vector-borne pathogens independent of transmission mode

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Many pathogens are transmitted by vectors, and transmission studies traditionally focus on vector-host-pathogen interactions. However, vectors are animals that forage in complex food webs, where they interact with individuals of many species across multiple trophic levels. These interactions might indirectly affect pathogens by altering vector fitness, movement, or feeding duration, although these effects might differ for pathogens with different transmission modes. Interactions that affect vector-host encounter rates, for example, might most strongly affect non-persistent pathogens, which attach to vector mouthparts and are transmitted quickly; interactions that affect vector feeding duration might most strongly affect persistent pathogens that require long feeding bouts for transmission. Here we addressed these knowledge gaps by developing a model to explore the impacts of interactions such as competition, predation, and mutualism on the spread of pathogens with either non-persistent or persistent transmission modes. Interactions that affected aspects of vector movement and feeding behavior (feeding duration, vector-host encounter rates) substantially altered rates of pathogen spread, whereas interactions affecting vector fitness (births, deaths) had relatively small effects. These effects of species interactions were largely independent of transmission mode, except when interactions affected vector-host encounter rates, where effects were strongest for non-persistent pathogens. Our results suggest that including species interactions and food web structure in pathogen transmission models could greatly enhance our understanding of disease ecology.

May 2, 11:00-11:30, Room 206

Stage-structured Discrete-time Models for Interacting Wild and Sterile Mosquitoes and their Global Dynamics

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To study the impact of the sterile insect technique on the disease transmission, we formulate stage-structured discrete-time models for the interactive dynamics of the wild and sterile mosquitoes using Beverton-Holt-type of survivability, based on difference equations. We incorporate different strategies for releasing sterile mosquitoes, and investigate the model dynamics. Numerical simulations are also provided to demonstrate dynamical features of the models.

May 4, 15:30-16:00, Room 206

Causation network constructions

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Existing methods on causation detection often misidentify indirect causal influences as direct ones, due to the effect of causation transitivity. We solve this problem by developing a method based on an articulated integration of three basic tools from nonlinear dynamics and statistics. We demonstrate our method by using data from a number of real-world systems. As direct causal links are key to the fundamental dynamical underpinnings of a variety of complex systems, our PCM method will become a useful tool to unlock from data the inner mechanisms of real systems in diverse disciplines.

May 3, 11:00-11:30, Room 201

on-Isothermal Electrokinetics: Energetic Variational Approach

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A number of ion channels are observed to be sensitive to the temperature changes. These temperature-gated ion channels can detect the temperature thus regulate the internal homoeostasis and disease-related processes such as the thermal adaptation and the fever response. In order to understand how the temperature affects the ion channel mechanics, we develop a Poisson–Netnst–Planck–Fourier (PNPF) system through the energetic variational approach. With given form of the free energy functional and the entropy production, we achieve the mechanical equations and a temperature equation, which satisfy the laws of thermodynamics automatically. From the energy point of view, we also develop the numeric scheme which satisfy the discrete energy dissipation.

May 2, 15:30-16:00, Room 207

Age-dependent intraspecific competition in pre-adult life stages and its effects on adult population dynamics

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Intra-specific competition in insect and amphibian species is often experienced in completely different ways in their distinct life stages. Competition among larvae is important because it can impact on adult traits that affect disease transmission, yet mathematical models usually ignore larval competition. We present two mathematical models of larval competition in the form of delay differential equations for the adult population derived from age structured models that include larval competition. Results on boundedness and persistence are also proved.

May 3, 15:30-16:00, Room 207

Backward bifurcation and oscillations in a nested immuno-eco-epidemiological model

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This talk introduces a novel partial differential equation immuno-eco-epidemiological model of competition in which one species is affected by a disease while another can compete with it directly and by lowering the first species' immune response to the infection, a mode of competition termed stress-induced competition. When the disease is chronic, and the within-host dynamics are rapid, we reduce the partial differential equation model (PDE) to a three-dimensional ordinary differential equation (ODE) model. The ODE model exhibits backward bifurcation and sustained oscillations caused by the stress-induced competition. The ODE model, although not a special case of the PDE model, is useful for detecting backward bifurcation and oscillations in the PDE model. Backward bifurcation related to stress-induced competition allows the second species to persist for values of its invasion number below one. Furthermore, stress-induced competition leads to destabilization of the coexistence equilibrium and sustained oscillations in the PDE model. We suggest that complex systems such as this one may be studied by appropriately designed simple ODE models.

May 4, 10:30-11:00, Room 207

Disease models that include immigration

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Many disease transmission models exhibit a threshold behaviour based on the basic reproduction number $\mathcal{R}_0$.

- If $\mathcal{R}_0 < 1$, then there is a globally asymptotically stable disease-free equilibrium.
- If $\mathcal{R}_0 > 1$, then there is a globally asymptotically stable endemic equilibrium.

This phenomenon has been shown for many individual models that are based on ODEs as well as systems with delay, age-structure and diffusion. A necessary condition for this phenomenon to occur is that there is a disease-free equilibrium.

If a system includes immigration of infected individuals, however, then there is no disease-free equilibrium, and therefore the basic reproduction number $\mathcal{R}_0$ does not exist. Let the level of immigration of infected individuals be given by W. Then $W = 0$ is the case where there is only immigration of healthy people. It is interesting to consider what happens as W increases from 0. Two issues arise.

- How does the size of $\mathcal{R}_0$ affect the movement of the disease-free equilibrium as W increases from 0? Does it move into the positive orthant or away from the positive orthant?
- How is the calculation that the endemic equilibrium was globally asymptotically stable affected by W increasing from 0?

Models that include immigration of infected individuals are important due to the high level of contact between different cities, regions and countries in today's world. Local eradication of a disease does not lead to permanent eradication due to modern levels of travel.

May 4, 11:30-12:00, Room 201

The role of outcome preferences in optimizing heterogenous disease control strategies

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As infectious diseases spread, they do not observe city, state, regional or national boundaries. As such, underlying susceptible population has a patchy structure which suggests metapopulation approach to epidemic modeling. When the patches of the metapopulation are managed by different public health authorities, it is natural to consider heterogeneous disease control strategies. For deterministic models, the basic reproduction number, R_0, is typically a sharp threshold separating the extinction or persistence of the disease. When comparing two control strategies, the one which minimizes R_0 is optimal. Stochastic epidemic models are necessary to account for inherent randomness in the initial phase of an outbreak. In this case, the disease is considered persistent if the probability of extinction is less than 1. This probability is often approximated by branching process techniques. It has been shown that R_0 is also a threshold in the case of branching processes. The probability of extinction is a hitting probability. In this talk, a case is made to consider other hitting probabilities to measure the effectiveness of control strategies when outcome preferences are biased by public health authorities. A technique to approximate these probabilities is presented. Results are compared to standard techniques.

May 3, 17:00-17:30, Room 206

Ecology meets economic growth: a general dynamic model framework

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Understanding why some human populations remain extremely poor despite current development trends around the world remains a mystery to the natural, social and mathematical sciences. The poor rely on their immediate natural environment for subsistence and suffer from high burdens of infectious diseases. We present a general framework for modeling the ecology of poverty and disease, focusing on infectious diseases and renewable resources. Interactions between these ecological drivers of poverty and economics create reinforcing feedbacks resulting in three possible development regimes: 1) globally stable wealthy/healthy development, 2) globally stable unwealthy/unhealthy development, and 3) bistability. We show that the proportion of parameters leading to poverty is larger than that resulting in healthy/wealthy development; bistability consistently emerges as a general property of generalized disease-economic systems and that the systems under consideration are most sensitive to human disease parameters. The framework highlights feedbacks, processes and parameters that are important to measure in future studies of development, to identify effective and sustainable pathways out of poverty.

May 4, 15:30-16:00, Room 207

Management Strategies in a Malaria Model Combining Human and Transmission-blocking Vaccines

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The concurrent use of multiple strategies has been recommended as an effective strategy to reduce malaria and its burden. In this talk, I will present a new mathematical model studying control strategies of malaria transmission, where the control is a combination of human and transmission-blocking vaccines, and larvacide (vector control). The existence of backward bifurcation is established analytically in the absence of vaccination, and numerically in the presence of vaccination. Optimal strategies using vaccination and vector control are investigated to gain qualitative understanding on how different combinations of these controls should be used to reduce disease prevalence in a malaria endemic setting. Our results suggest that the combination of the two vaccination controls integrated with vector control has the highest impact on reducing the number of infected humans and mosquitoes in the population.

May 2, 10:30-11:00, Room 201

Transport of intermediate filaments in cells

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Intermediate filament proteins assemble in filaments that organize in networks, which are in charge of important cell functions such as the regulation of the mechanical integrity of cells. The intracellular transport of intermediate filaments has been recently identified as a key process for the network dynamics. Their regulated interactions with motor proteins and structural linkers result in different modes of motility for filaments in cells. To elucidate the spatio-temporal distribution of intermediate filaments in cells, mathematical models are developed to investigate the contributions of different types of transport. A first model for the motion of single filaments driven by antagonistic motor proteins is proposed.

May 4, 12:00-12:30, Room 207

On an Advection-reaction-diffusion Competition System with Double Free Boundaries Modeling Invasion and Competition of *Aedes Albopictus* and *Aedes Aegypti* Mosquitoes

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Based on the invasion of the *Aedes albopictus* mosquitoes and the competition between *Ae. albopictus* and *Ae. aegypti* mosquitoes in the United States, we consider an advection-reaction-diffusion competition system with two free boundaries consisting of an invasive species (*Ae. albopictus*) with density u and a local species (*Ae. aegypti*) with density v in which u invades the environment with leftward front $x = g(t)$ and rightward front $x = h(t)$. In the case that the competition between the two species is strong-weak and species v wins over species u, the solution (u, v) converges uniformly to the semi-positive equilibrium $(0, 1)$, while the two fronts satisfy that $\lim_{t\to\infty}(g(t), h(t)) = (g_\infty, h_\infty) \subset \mathbb{R}$. In the case that the competition between the two species is weak, we show that when the advection coefficients are less than fixed thresholds there are two scenarios for the long time behavior of solutions: (i) when the initial habitat $h_0 < \pi\big(\sqrt{4-\nu_1^2}\big)^{-1}$ and the initial value of u is sufficiently small, the solution (u, v) converges uniformly to the semi-positive equilibrium $(0, 1)$ with the two fronts $(g_\infty, h_\infty) \subset \mathbb{R}$; (ii) when the initial habitat $h_0 \geq \pi\big(\sqrt{4-\nu_1^2}\big)^{-1}$, the solution (u, v) converges locally uniformly to the interior equilibrium with the two fronts $(g_\infty, h_\infty) = \mathbb{R}$. In addition, we propose an upper bound and a lower bound for the asymptotic spreading speeds of the leftward and rightward fronts. Numerical simulations are also provided to confirm our theoretical results.

Based on a joint paper with Canrong Tian.

May 3, 10:30-11:00, Room 201

Getting our feet wet: rivers in planar flows and neuronal models

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In this work, we introduce a new change of coordinates, local orthogonal rectification or LOR, that can be applied at any selected curve in the phase space of a dynamical system. LOR, based on the Frenet frame, yields a coordinate system, the LOR frame, which allows us to rigorously study curves that are nearly-invariant in the flow. We use the LOR approach to derive a novel definition for rivers, long-recognized but poorly understood trajectories that locally attract other orbits yet need not be related to invariant manifolds or other familiar phase space structures; to identify rivers within several example neuronal systems; and to analyze how these structures impact model neuronal dynamics.

May 4, 16:00-16:30, Room 206

Using a Mathematical Model with Individual Patient Data to Quantify Differences Between Patients with Diabetic Foot Ulcers

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In this work, we quantify differences in healing responses between type-II diabetic patients with foot ulcers. This work builds off of our previous publication (Krishna et al., B Math Biol, 2015), where we formulate a mathematical model to describe healing responses using averaged time-course data from another study (Muller et al., Diabet Med, 2008). In Mullers work, they collect data from 16 patients with type-II diabetes. In addition to recording wound areas, Muller also measures levels of matrix metalloproteinases and their inhibitors at Weeks 0, 1, 2, 4, 8, and 12, collected from wound fluid. The patients are divided into two groups categorized as good healers and poor healers dependent upon the healing response at the four-week point. In our previous work, we use the average data to calibrate our mathematical model and quantify differences between the two groups. In our current work, we have calibrated our mathematical model for each individual patient and have quantified differences between these patients. In this presentation, we will discuss how our model has identified differences across patients using a variety of techniques.

May 3, 11:30-12:00, Room 201

Complex dynamics and bifurcations in a toxin-dependent aquatic population model

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The study of effects of environmental toxins on ecosystems is of great interest from both environmental and conservation points of view. In this talk, I will present the complex dynamics and bifurcations of a toxin-dependent aquatic population model. The analytical and numerical results show that both the environmental toxin level and the depuration capability of the population significantly affect the population persistence. The model exhibits a multifarious array of dynamics. While low levels of external toxin allow population persistence and high levels of toxin lead to an extirpation, intermediate toxin concentrations can produce very rich dynamics, such as transient oscillations, hysteresis, heteroclinic orbits, and a codimension-two bifurcation. In particular, a regime of bistability exists where the population is doomed to extinction or survival, depending on initial state of the system. As a practical implication of our study, the toxic effects of methylmercury on rainbow trout are scrutinized.

May 3, 12:00-12:30, Room 207

Front Propagation Dynamics in Chemotaxis Models with Logistics Source on R^N

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This talk is concerned with front propagation dynamics in chemotaxis models with logistic source on R^N. I will first identify the circumstances under which positive classical solutions of such chemotaxis models exist globally, which is fundamental for the study of front propagation dynamics as well as many other dynamical features. Next, I will discuss the asymptotic stability of nonzero constant equilibria of such chemotaxis models, which also plays a role in the study of front propagation dynamics. I will then consider the spreading properties of positive solutions with compactly supported or front-like initial functions. Throughout the talk, special attention will be given to the combined effect of the chemotaxis sensitivity and the logistic damping on the above dynamical issues.

This talk is based on my joint works with Rachidi Salako.

May 2, 17:30-18:00, Room 207

Effect of spatial average on the spatial-temporal pattern formation of reaction-diffusion systems

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Some quantities in the reaction-diffusion models from cellular biology or ecology depend on the spatial average of density functions instead of local density functions. We show that such nonlocal spatial average can induce instability of constant steady state, which is different from classical Turing instability. In particular, for systems of two equations containing spatial averages, spatially non-homogeneous time-periodic orbits could occur through bifurcations from the constant steady state. Examples from a nonlocal predator-prey model and a pollen tube tip model will be used to demonstrate such bifurcations.

This is a joint work with Qingyan Shi and Yongli Song.

May 3, 16:30-17:00, Room 201

Hopf bifurcation in a reactiondiffusion equation with distributed delay and Dirichlet boundary condition

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The stability and Hopf bifurcation of the positive steady state to a general scalar reaction-diffusion equation with distributed delay and Dirichlet boundary condition are investigated in this paper. The time delay follows a Gamma distribution function. Through analyzing the corresponding eigenvalue problems, we rigorously show that Hopf bifurcations will occur when the shape parameter $n \geq 1$, and the steady state is always stable when $n = 0$. By computing normal form on the center manifold, the direction of Hopf bifurcation and the stability of the periodic orbits can also be determined under a general setting. Our results show that the number of critical values of delay for Hopf bifurcation is finite and increasing in n, which is significantly different from the discrete delay case, and the first Hopf bifurcation value is decreasing in n. Examples from population biology and numerical simulations are used to illustrate the theoretical results.

May 3, 17:30-18:00, Room 206

Distributions of Proteins – Models and Experiments

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Using quantitative data collected from adult rat aortic smooth muscle cell cultures *in vitro*, we compare the 2-stage model for protein synthesis, under the assumption that the time these proteins take to decay is significantly longer than their corresponding mRNA. Using the theoretical model, we are able to extract interesting biochemical parameters that may be verified using a different set of experiments, such as the number of proteins translated during an mRNA lifetime.

May 4, 16:00-16:30, Room 207

Periodic Solutions of Abstract Semilinear Equations with Applications to Biological Models

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We study the existence of periodic solutions to the abstract semilinear equation $du/dt = Au(t) + F(t, u(t))$, $t \geq 0$ and the abstract semilinear evolution equation $du/dt = A(t)u(t) + F(t, u(t))$, $t \geq 0$ in a Banach space X, where A is a linear operator on X (not necessarily densely defined) satisfying the Hille-Yoshida condition; $A(t)$ is a T-periodic linear operator on X (not necessarily densely defined) satisfying the hyperbolic conditions; and $F : [0, \infty) \times \mathrm{cl}(D(A)) \to X$ is continuous and T-periodic in t. Special cases when $F(t, u(t)) = f(t)$, where $f(t)$ is T-periodic in t, are also considered. Sufficient conditions on $A, A(t), f(t)$ and $F(t, u(t))$ are given to ensure the existence of T-periodic solutions in these equations. As applications, the main results are applied to establish the existence of periodic solutions in age-structured models with periodic harvesting and diffusive logistic equations with periodic coefficients.

May 4, 11:00-11:30, Room 201

Optimal dengue vaccination to mitigate Zika cases

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Zika and dengue viruses belong to the same Flavivirus family and are primarily transmitted by a common mosquito species Aedes aegypti. Zika outbreaks have commonly occurred in dengue-endemic areas, and cocirculation and coinfection of both viruses have been reported. As recent studies on immunological cross-reactivity have confirmed that convalescent plasma following dengue infection can enhance Zika infection, it is important to examine whether and how dengue vaccination in a large population may affect Zika infection dynamics due to antibody-dependent enhancement. In this study, we evaluate the impact of dengue vaccination on Zika infection dynamics through a mathematical coinfection dynamics model. We show that an appropriately designed and optimized dengue immunization program can not only help control the dengue spread but also, counter-intuitively, reduce Zika infections. We estimate that the optimal and critical dengue effective vaccine coverage rates in Mexico, Brazil, and French Polynesia to be (73.6%, 99%), (51.4%, 88.2%), and (68.2%, 97.6%), respectively.

This talk is based on the joint works with Xi Huo, Yanni Xiao, Shigui Ruan, and Jianhong Wu.

May 2, 11:30-12:00, Room 207

Disease spread on a dynamic multi-layer network: limit laws

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A fundamental feature of disease spread on networks is that disease affects the network structure. For example, infection may cause an individual to deactivate their regular community contacts (e.g. too ill to go to work or school), but concomitantly activate their contact with caregivers, either in home or hospital. This leads us to consider disease spread on a multi-layered network, where the structure of each layer evolves in tandem with the disease. Specifically, we give a law of large numbers for a stochastic disease process on a dynamic layered configuration model, resulting in a simple system of ODEs. Analysis of the resulting limiting ODEs illustrates how the structure of the different layers of the network, and the activation and deactivation of edges in the network, affects basic features of the outbreak such as the basic reproduction number and the final outbreak size. Among our results is a condition for when the correlation equations of Rand are asymptotically correct.

This is joint work with Karly Jacobsen, Mark Burch, and Grzegorz Rempala.

May 4, 11:30-12:00, Room 207

Structural and Practical Identifiability Analysis of Zika Epidemiological Models

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The Zika virus (ZIKV) epidemic has caused an ongoing threat to global health security and spurred new investigations of the virus. Use of epidemiological models for arbovirus diseases can be a powerful tool to assist in prevention and control of the emerging disease. In this article, we introduce six models of ZIKV, beginning with a general vector-borne model and gradually including different transmission routes of ZIKV. These epidemiological models use various combinations of disease transmission (vector and direct) and infectious classes (asymptomatic and pregnant), with addition to loss of immunity being included. The disease induced death rate is omitted from the models. We test the structural and practical identifiability of the models to find whether unknown model parameters can uniquely be determined. The models were fit to obtained time series data of cumulative incidences and pregnant infections from the Florida Department of Health Daily Zika Update Reports. The average relative estimation errors (ARE) were computed from the Monte Carlo simulations to further analyze the identifiability of the models. We show that direct transmission rates are not practically identifiable, however, fixed recovery rates improve identifiability overall. We found ARE low for each model (only slightly higher for those that account for a pregnant class), and help to confirm a reproduction number greater than one at the start of the Florida epidemic.

May 2, 12:00-12:30, Room 206

Modeling the Effects of Antibody Responses on HIV Dynamics under Drugs of Abuse

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Drugs of abuse, such as opiates, have been widely associated with enhancing HIV replication, accelerating disease progression and diminishing host-immune responses. In this talk, I will present a mathematical model to study the effects of antibody responses on HIV dynamics under drugs of abuse. The model is parameterized using data from simian immunodeficiency virus infection in morphine addicted macaques. Using our model, we evaluate how the presence of drugs of abuse alters antibody responses and how this alteration affects the key components of virus dynamics. Furthermore, we analyze how periodic intake of drugs of abuse have impact on the global stability properties of host-virus dynamical system.

May 2, 16:00-16:30, Room 201

Turing patterns in a predator-prey model with seasonality

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Many ecological systems show striking non-homogeneous population distributions. Diffusion-driven instabilities are commonly studied as mechanisms of pattern formation in many fields of biology but only rarely in ecology, in part because some of the conditions seem quite restrictive for ecological systems. Seasonal variation is ubiquitous in temperate ecosystems, yet its effect on pattern formation has not yet been explored. We formulate and analyze an impulsive reaction-diffusion system for a resource and its consumer in a two-season environment. While the resource grows throughout the 'summer' season, the consumer reproduces only once per year. We derive conditions for diffusion-driven instability in the system, and we show that pattern formation is possible with a ratio-dependent functional response or a Beddington-DeAngelis functional response. More importantly, we find that a low overwinter survival probability for the resource enhances the propensity for pattern formation: diffusion-driven instability occurs even when the diffusion rates of prey and predator are comparable (although not when they are equal).

May 4, 15:30-16:00, Room 201

Using effective boundary conditions to model fast diffusion on a road in a large field

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We consider a logistic diffusion equation on the plane consisting of two components, a straight "road" and a "field", in each of which the diffusion rate differs significantly. Compared to the size of the field, the width of the road is assumed to be small. Thus in this diffusion equation multiple scales appear in two places: the spatial variable and the diffusion parameter. Such an equation is not easy to solve numerically, and it is not easy to see the effects of the road. Recently, Berestycki, Roquejoffre and Rossi provide a model which is meant to resolve these issues. In this paper we first use the idea of effective boundary conditions (EBCs) to propose, rigorously, a different model: we study the limit of the solution of the original logistic equation as the width of the road approaches zero, obtaining a limiting model, in which the road now is the horizontal line with EBCs imposed on it. This effective problem has no multiple scales and hence should be easier to solve numerically. Moreover, to see the effects of the road, we further investigate the asymptotic propagation speed of the effective model, showing that the road indeed enhances the spreading speed along any direction within a certain angle with the road, provided that the diffusion rate on the road is of the order of the reciprocal of the width of the road.

May 3, 11:00-11:30, Room 207

Stochastic Models of Bovine Babesiosis with Juvenile Cattle

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Bovine Babesiosis is a tick borne parasitic disease, which renders more than 1.3 billion bovines at potential risk of being infected worldwide. This work is devoted to stochastic models of Bovine Babesiosis, with a focus on the disease extinction and outbreak and probability distribution of the infectious adult bovine and that of infectious ticks. The stochastic models are a system of continuous time Markov chains derived based on the dynamics of deterministic ordinary differential equation models, i.e., Model J in Saad-Roy et al. (BMB, 2015) and Model Aranda et al. (Math Methods Appl Sci, 2012) (which is a special case of Model J). The multitype branching process approximation is used to estimate the probability of disease extinction/outbreak. Unlike the deterministic dynamics that indicate the basic reproduction number R_0 serves a sharp disease threshold (i.e., if R_0 is less than or equal to the unity, the disease dies out; if R_0 is above the unity, the disease is uniformly persist and becomes established, in

Saad-Roy et al. (BMB, 2015)), our stochastic models indicate, more realistically, that there is always a positive probability that disease extinction within both cattle and tick populations.

This is joint work with Pauline van den Driessche.

May 2, 16:30-17:00, Room 201

Persistence and Extinction of Population in Reaction-Diffusion-Advection Model with Strong Allee Effect Growth

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A reaction-diffusion-advection equation with strong Allee effect growth rate is proposed to model a single species stream population in a unidirectional flow. Here random undirected movement of individuals in the environment is described by passive diffusion, and an advective term is used to describe the directed movement in a river caused by the flow. Under biologically reasonable boundary conditions, the existence of multiple positive steady states are shown when both the diffusion coefficient and the advection rate are small, which lead to different asymptotic behavior for different initial conditions. On the other hand, when the advection rate is large, the population becomes extinct regardless of initial condition under most boundary conditions.

May 3, 11:30-12:00, Room 206

Dynamical models for estimating the population sizes for Culex mosquitoes using the weekly trap counts

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The weekly Culex mosquito trap counts from a mosquito surveillance program are usually used to assess the mosquito abundance for determining the risk of WNV or other mosquitos borne diseases (MBD). However, these trap counts are sparse and not necessarily accurate for indicating exact numbers, density or population sizes of Culex mosquitoes in the trapping area. In this work, we define an "effective trapping zone" for a CDC light trap and incorporate the trapping mechanism of a trap and collecting procedure into a predictive dynamical population model for Culex mosquitoes. Also, the role of blood meal hosts and mosquito biting feeding preference will be incorporated into the model. Based on the weekly surveillance trap counts data and daily weather conditions from Peel region, Ontario, we predict total Culex mosquito population sizes as well as mosquito trap counts in the effective trapping zones.

This is a work supervised by Professor Huaiping Zhu and joint with Wendy Pons, Nicholas Ogden, Beate Sander.

May 3, 11:30-12:00, Room 207

A New Immuno-epidemiological Model of Visceral Leishmaniasis in Dogs

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Leishmaniasis is a neglected and emerging disease prevalent in Mediterranean and tropical climates. As such, the study and development of new models are of increasing importance. We present the progress on a new immuno-epidemiological model. The within-host system is based on the data collected by Courtenay et al. (2014), showing the movement and proliferation of the parasite, as well as the IgG response. The between-host system is a vector-host model structured by time-since-infection. We compute the basic reproduction number of the between-host model and provide the stability results of the disease-free and endemic equilibria. The model exhibits backward bifurcation and existence of multiple endemic equilibria when $R_0 < 1$.

May 2, 12:00-12:30, Room 207

Growth on Two Limiting Essential Resources in a Self-Cycling Fermentor

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A system of impulsive differential equations with state-dependent impulses is used to model the growth of a single population on two limiting essential resources in a self-cycling fermentor. The self-cycling fermentation process is a semi-batch process and the model is an example of a hybrid system. In this case, a well-stirred tank is partially drained, and subsequently refilled using fresh medium when the concentration of both resources falls below some prescribed threshold. We consider the process successful if the threshold for emptying and refilling the reactor can be reached indefinitely without interference by the operator. We prove that whenever the process is successful, the model predicts that the concentrations of the population and the resources converge to a positive periodic solution. We derive conditions for the successful operation of the process that are show to be initial condition dependent and prove that if these conditions are not satisfied, the the reactor fails after at most finitely many impulses. We show numerically that there is an optimal fraction of the medium drained from the tank at each impulse that maximizes the output of the process. Potential applications include water purification and biological waste remediation.

This is joint work with Ting-Hao Hsu and Tyler Meadows.

May 3, 17:00-17:30, Room 201

Quenched flow in symmetric multi-component FCH

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Multicomponent mixtures support bilayers with a diversity of lipid compositions. We study a two-component FCH model with a radial symmetric potential which admits a family of quasi-bilayers with various compositional ratios between amphiphile A and B. In the absence of pearling, the compositional and geometric evolution of quasi-bilayers decouples, in the sense that the former evolution takes place in a slow time scale when the normal velocity of interface is still zero. More specifically, the composition ratio satisfies a nonlocal equation accommodating rich dynamics. Depending on the competition between the phase separation and the quenching of the background, the composition ratio evolves into, (1) a homogeneous profile; (2) a phase separation profile where the bilayer consists of pure A regions and pure B regions; or, (3) a quenched profile in a co-dim two manifold. In last two cases, a rapid spatial variation of the composition promotes surface diffusion terms from lower orders. While the evolution of phase separation profiles mimics Allen-Cahn type coarse graining, novel dynamics emerges from rapid varying profiles in a neighborhood of the quenched manifold: compositional profiles stay nearby the quenching manifold and evolve into periodic profiles with large periods.

May 2, 17:00-17:30, Room 207

Spatial Spread of Epidemic Diseases in Geographical Settings: Seasonal Influenza Epidemics in Puerto Rico

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Deterministic models are developed for the spatial spread of epidemic diseases in geographical settings. The models are focused on outbreaks that arise from a small number of infected hosts imported into subregions of the geographical settings. The goal is to understand how spatial heterogeneity influences the transmission dynamics of the susceptible and infected populations. The models consist of systems of partial differential equations with diffusion terms describing the spatial spread of the underlying microbial infectious agents. The model is compared with real data from seasonal influenza epidemics in Puerto Rico.

May 2, 16:30-17:00, Room 207

Mathematical modeling for machine tool vibration

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We develop a delay differential equation that arises in the modelling of vibration (chatter) that can occur when machining a rotating workpiece using a lathe, in the case when the cut is made longitudinally. We explore conditions of eliminating chatter by spindle speed variation (SSV).

May 3, 16:00-16:30, Room 201

Nonlocal differential equations and convergence to the classical differential equations

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Nonlocal diffusions are ubiquitous in many fields of applied sciences, ranging from physics and material science, to biology, ecology and image processing. This talk is concerned with the relation between nonlocal evolution equations and their local counterpart, i.e., classical evolution equations.

More precisely, we will first consider the classical and nonlocal dispersal evolution equations, which are used to model the dynamics of diffusive systems in biology or ecology which exhibit random or local, and nonlocal internal interactions, respectively. We study the dynamics of such equations complemented with Dirichlet, Neumann, and periodic types of boundary condition in a unified way, and show that certain dynamics of classical dispersal evolution equations can be approximated by the dynamics of nonlocal dispersal evolution equations with the corresponding boundary condition and properly rescaled kernels.

Next, we consider the nonlocal and classical Allen-Cahn equations where the nonlocal kernel is sign-changing. In this case, we show that the approximations of nonlocal equations to its classical counterparts do not hold in general.

May 3, 16:00-16:30, Room 206

Dynamics of Populations with Individual Variation in Dispersal on Bounded Domains

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Most classical models for the movement of organisms assume that all individuals have the same patterns and rates of movement, but there is empirical evidence that movement rates and patterns may vary among individuals. One way to capture variation in dispersal is to allow individuals to switch between two distinct dispersal modes. We consider models for populations with logistic-type local population dynamics whose members can switch between two different nonzero rates of diffusion. The resulting reaction-diffusion systems can be cooperative at some population densities and competitive at others. We analyze the dynamics of such systems on bounded regions. The analytic methods include ideas and results from reaction-diffusion theory, semi-dynamical systems, and bifurcation/continuation theory.

This is a joint work with Drs. Steve Cantrell and Chris Cosner.

May 2, 11:00-11:30, Room 207

Multiple Attractors in the Simple Epidemic Model

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Multiple recurrent outbreak cycles have been commonly observed in infectious diseases such as measles and chicken pox. This complex outbreak dynamics in epidemics is rarely captured by deterministic models. In this paper, we investigate a simple 2-dimensional SI epidemic model and propose that the coexistence of multiple attractors attributes to the complex outbreak patterns. We first determine the parameter conditions for the existence of an isolated center, then properly perturb the model to have a generalized Hopf bifurcation, and obtain small-amplitude limit cycles surrounding the center. We further analytically prove that the maximum number of the coexisting limit cycles is three, and calculate a corresponding set of parameters. Simulation results demonstrate the case with the maximum coexisting attractors, which has one stable disease free equilibrium and two stable endemic periodic solutions separated by one unstable periodic solution. Therefore, different disease outcomes can be predicted by a single nonlinear deterministic model based on different initial data.

May 4, 12:00-12:30, Room 206

Models to assess the effects of Wolbachia-carrying mosquito augmentations and mating competition on the control of dengue disease

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The introduction of endosymbiont Wolbachia into laboratory-reared mosquito populations is an innovative new technology, which are then released to mix with natural populations to prevent the mosquito vectors from reproducing dengue virus or to suppress the density of natural mosquitoes and thus break the transmission cycle of dengue disease. Two stage-structured models are proposed to investigate the effects of non-identical sex ratio releases of Wolbachia-carrying mosquitoes and Wolbachia-carrying male releases with mating competition on the success of population replacement and suppression, respectively. Analysis of the existence, local stability and bifurcation analysis of the equilibria for the two models revealed the existence of forward bifurcation or backward bifurcation and multiple attractors, and their basins of attraction were numerically estimated. For the first model, the effects of mosquito augmentation for the model with imperfect and perfect transmission rates were obtained. Then three possible results for mosquito augmentation were summarized for different parameter regions. Further we explored an uncertainty and sensitivity analysis of solutions to estimate the effects of different parameter values on the success or failure of population replacement. For the second model, global dynamical properties of the system without and with male releases were explored by using Lyapunov function and theory of monotone operators, respectively. Then we defined the three levels of population suppression by parameter thresholds and computed their suppression rates, also discussed the control strategies for the success of population eradication. The success of population eradication will rely on assessing basic offspring number of natural mosquitoes, the selection of suitable Wolbachia strains and appropriate release amount of Wolbachia-carrying males. The results of this study will be helpful for public health authorities in designing proper strategies of mosquito augmentations for the control of dengue disease.

May 4, 11:30-12:00, Room 206

Understanding the impact of diapause and co-feeding on tick-borne disease spread

Xue Zhang
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We consider the dynamic vectorhostpathogen interaction motivated by tick-borne infections. We divide the vector population by the stage before and after the vectors contact with hosts when co-feeding transmission may take place, and we also consider the case where vector development involve two time lags due to normal development and diapause. We derive threshold conditions for disease persistence and for nonlinear oscillations in the vector population and in the diseased vector and host populations. Our analysis, using a mechanistic dynamic model, shows that diapause and co-feeding transmission may generate periodic and irregular oscillations even when seasonal variations of the environmental conditions are ignored.

This is based on joint work with Xiaotian Wu and Jianhong Wu.

May 4, 16:00-16:30, Room 201

Computational Modeling of Cytokinesis of Eukaryotes driven by F-actin Enriched Contractile Ring

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Cell Mitosis is a fundamental process in eukaryotic cell reproduction, during which parent cells nucleus first dissembles leading to DNA and chromosome replication, then chromosomes migrate to new locations within the parent cell to form offspring nuclei which triggers cytokinesis leading to the formation of two offspring cells eventually.

In this talk, we develop a full 3D multiphase hydrodynamic model to study the fundamental mitotic mechanism in cytokinesis, the final stage of mitosis. The model describes the cortical layer, a cytoplasmic layer next to the cell membrane rich in F-actins and myosins, as an active liquid crystal system and integrate the extra cellular matrix material and the nucleus into a multiphase complex fluid mixture. With the novel active matter model built in the system, our 3D simulations show very good qualitative agreement with the experimental obtained images. The hydrodynamical model together with the GPU-based numerical solver provides an effective tool for studying cell mitosis theoretically and computationally.

May 4, 16:00-16:30, Room 201

A mathematical model of angiogenesis and tumor growth: analysis and application in anti-angiogenesis therapy

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This talk presents a combined mathematical model of angiogenesis (new blood vessel growth) and tumor growth. The angiogenesis part assumes the capillary as a viscoelastic continuum whose stress depends on cell proliferation or death, and the tumor part is a Darcy's law model modeling the tumor mass as an incompressible fluid where the nutrient-dependent growth elicits volume change. We show the tumor tends to maximize the nutrient transfer by blood vessel co-option and the anti-angiogenesis treatment by using growth factor neutralizing antibody would regress the neovasculature and shrink the tumor size. However, the shrunk tumor mass could survive by feeding on mature blood vessels that resist the treatment.

May 2, 12:00-12:30, Room 201

Modeling the role of white-tailed deer in geographic spread of the black-legged tick Ixodes scapularis by a spatially nonlocal model

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Lyme disease is transmitted via blacklegged ticks, the spatial spread of which is believed to be primarily via transport on white-tailed deer. In this talk, I will present a mathematical model to describe the spatial spread of blacklegged ticks due to deer dispersal. The model turns out to be a system of differential equations with a spatially non-local term accounting for the phenomenon thata questing female adult tick that attaches to a deer at one location may later drop to the ground, fully fed, at another location. After justifying the well-posedness of the model and analyzing the stability of its steady states, we will explore the existence of traveling wave fronts connecting the extinction equilibrium with the positive equilibrium for the system. We derive an algebraic equation that determines a critical value c^* which turns out to be the minimum wave speed and the actual spread speed of the tick population. We then present some numerical simulation results to demonstrate the above results. We also explore the dependence of c^* on the dispersion rate of the white tailed deer, by which one may evaluate the role of the deer's dispersion in the geographical spread of the ticks.

This is a joint work with Stephen Gourley and Xiulan Lai et al.

Abstracts of Posters

The poster session is held on Wednesday May 2, 18:15-19:30.

Multistabillity of synchronized clusters in networks of phase oscillators

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Patterns of synchronized clusters are observed in many biological networks, ranging from neuronal populations to fireflies and ecological metacommunities. Despite significant interest, the emergence and hysteretic transitions between stable clusters in a network of identical oscillators have still not been fully understood. In particular, the celebrated Kuramoto model of identical phase oscillators is known to exhibit multiple spatiotemporal patterns, including co-existing clusters of synchrony and chimera states in which some oscillators form a synchronous cluster, while the others oscillate asynchronously. Rigorous analysis of the stability of clusters and chimeras in the finite-size Kuramoto model has proved to be challenging, and most existing results are numerical. In this talk, we contribute toward the rigorous understanding of the emergence of stable clusters in networks of identical Kuramoto oscillators with inertia. We derive the conditions under which patterns of synchrony stably co-exist and demonstrate how inertia affects the hysteretic transitions between the patterns. Our stability results also shed light on the emergence of transient and stable chimeras.

Two-species competition with directed diffusion and harvesting

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In the two-species competition model with logistic growth law and different dispersal strategies, the influence of harvesting (or culling) on the competition outcome is explored. Both species are harvested proportionally to their intrinsic growth, but the harvesting effort may be different for the two species. In the absence of harvesting, the two competitors may coexist, and one can apply such "small enough" harvesting rate that the state of coexistence is conserved. In the situation, when, in the absence of harvesting, one population may drive the other into extinction, by applying harvesting to the "successful" species one can guarantee survival to its competitor. We provide estimates on harvesting bounds that would lead to a desired outcome, be it competitive exclusion or coexistence. The analytical conclusions are supported by the numerical simulations and visualizations.

Invariant Angular Manifolds in the Goodwin Oscillator

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Using our new technique, Local Orthogonal Rectification (LOR), we build a moving normal frame to study the dynamics of the Goodwin oscillator near its limit cycle. The Goodwin oscillator is a standard model of genetic regulation and is widely used to model circadian rhythms. The LOR framework allows us to rectify the limit cycle while preserving and exposing the geometry of the nearby dynamics, particularly how trajectories rotate around the limit cycle. We identify novel two-dimensional, invariant manifolds, attendant to the limit cycle, which allow us to completely organize these angular dynamics. One of these manifolds serves as a separatrix for the angular dynamics and allows us to identify a region with high sensitivity to initial conditions, which may be useful in studying asymptotic phase sensitivity.

Revisiting a synthetic intracellular regulatory network that is sufficient for oscillations

Jonathan Tyler
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In 2000, Elowitz and Leibler introduced the repressilator a synthetic gene circuit with three genes that cyclically repress transcription of the next as well as a mathematical model describing it. In 2006, Muller et al. generalized the model for an arbitrary number of genes and analyzed the possible steady states, the stability of the steady states, and the possible asymptotic behavior. These previous models assume first-order transcription, translation, and degradation, with rates equivalent among genes, mRNAs, and proteins, respectively. This assumption, however, is not consistent with current biological knowledge. Accordingly, we propose a new repressilator model allowing for differing transcription, translation, and degradation terms. We show that, under conditions on these new functions, there is still a unique steady state when an odd number of genes are in the network. We also show that, with an odd number of genes, either the model converges to the steady state or to a periodic orbit. Finally, we compare fits of current repressilator data under the old and the new models. Fitting the data with the new model can lead to key insights into the dynamics of repression and degradation and help answer questions such as: How many repressors are necessary to inhibit transcription? How are certain proteins degraded?

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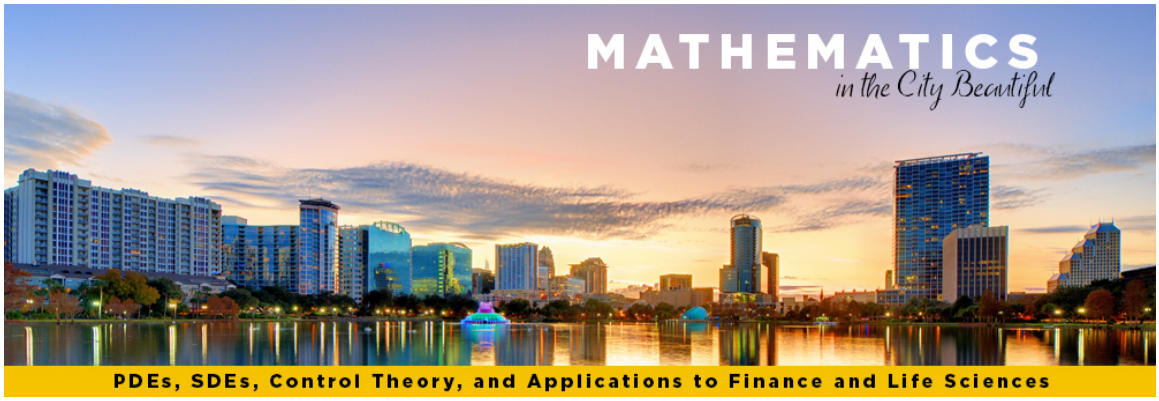

Mathematics in the City Beautiful:
PDEs, SDEs, Control Theory, and Applications to Finance and Life Sciences

The Conference "*Mathematics in the City Beautiful: PDEs, SDEs, Control Theory, and Applications to Finance and Life Sciences*" will be held at the University of Central Florida, on December 14-16, 2018, on the occasion of Professor Jiongmin Yong's 60th Birthday. More information regarding the MCB conference can be found on website

https://sciences.ucf.edu/math/mcb

CONFIRMED PLENARY SPEAKERS

- **Tyrone Duncan**, University of Kansas
- **Mary Ann Horn**, Case Western Reserve University
- **Suzanne Lenhart**, University of Tennessee
- **Jin Ma**, University of Southern California
- **George Yin**, Wayne State University
- **Xu Zhang**, Sichuan University, China

SCIENTIFIC COMMITTEE

- **William Hager**, University of Florida
- **Suzanne Lenhart**, University of Tennessee
- **Xin Li**, University of Central Florida
- **Eduardo Teixeira**, University of Central Florida
- **Xunyu Zhou**, Columbia University

The MCB conference will be co-organized by Suzanne Lenhart (Tennessee), Joseph Brennan, Andrew Nevai, Yuanwei Qi, Zhisheng Shuai, and Qiyu Sun (Central Florida), and be sponsored by the Department of Mathematics at the University of Central Florida. *We are currently seeking for external funding support and will update this later of the summer.*

University of Central Florida

The University of Central Florida, founded in 1963, is one of the fastest growing universities in the United States, and it is currently ranked the second largest university with a total enrollment over 66,000 students.

The university and its 13 colleges offer 216 degree programs from UCF's main campus, hospitality campus, health sciences campus, online and through its 12 regional locations. Regional campuses are located throughout Central Florida and include a fully accredited College of Medicine in the Medical City at Lake Nona. In addition to its impressive size and strength, UCF is ranked as a best-value university by *Kiplinger's*, as well as one of the nation's most affordable colleges by *Forbes*.

The university benefits from a diverse faculty and staff who create a welcoming environment and opportunities for students to grow, learn and succeed.

Department of Mathematics at the University of Central Florida

The Department of Mathematics at the University of Central Florida has 36 research faculty and 12 teaching faculty serving over 5,000 undergraduate and over 100 graduate students and invest heavily on research. Faculty members include invited speakers at the International Congress of Mathematics, the Marcus Wallenberg Prize winner, the ICTP Ramanujan Prize winner, Fellow of the Brazilian Academy of Sciences, Fellows of the American Mathematical Society, and a National Science Foundation CAREER awardee.

The UCF Mathematics Department has offered the PhD program in Mathematics since Fall 1993, with a Financial Mathematics track added in Fall 2017, the Master program in Mathematics since Spring 1971, with an Industrial Mathematics track added in Fall 2000 and a Financial Mathematics track added in Fall 2017, and the Graduate Certificates in Mathematical Sciences since Fall 2009. The current graduate program has 55 active PhD students, 13 active master students, and 38 active graduate certificate students. As of Fall 2017, the department has awarded 71 PhD degrees, 356 Master degrees, and 11 Mathematical Science Certificates.

The emphasis in the program is on contemporary areas of applied mathematics and traditional areas of core mathematics. A wide variety of graduate courses are offered to train students in mathematics and its application in a collegial, friendly environment with small classes and high student-faculty interaction. For appropriately trained students opportunities may exists to work under the Cooperative Education Program with local industries like *Lockheed Martin*, *NASA*, *Siemens*, and *Harris Corporation*.

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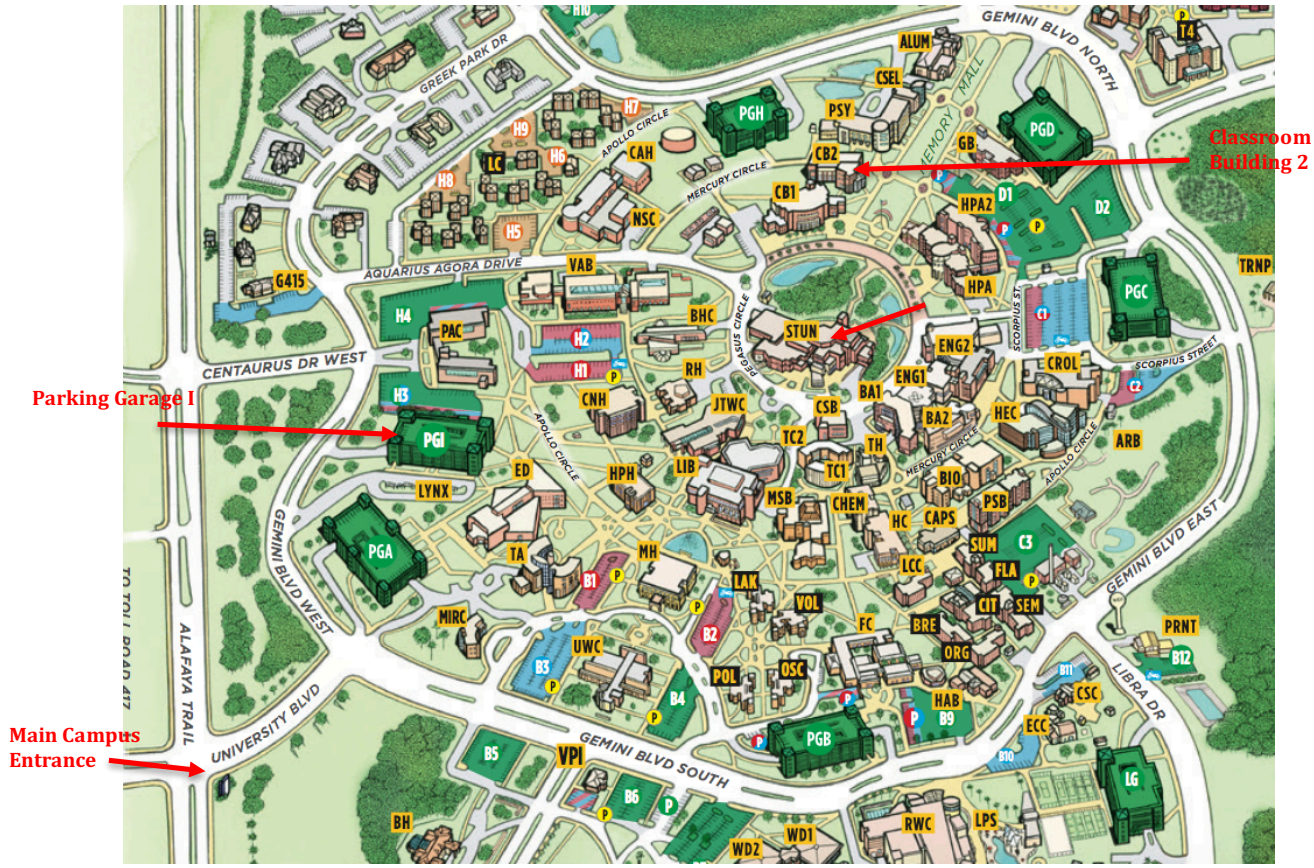
University of Central Florida Map



Guidance for FMB Conference Participants

CB2: Classroom Building 2
(*all conference talks, registration and welcome reception/poster session*)
PGI: Parking Garage I (*free conference parking permit available*)
MSB: Mathematical Sciences Building (*for your information o*nly)
STUN: Student Union (*conference banquet*)

Please notice that the hotel La Quinta Inns & Suites (11805 Research Pkwy, Orlando, FL 32826) locates the south of the campus.

To drive from the hotel to Parking Garage I, make a right turn to Research Pkwy West when leaving the hotel. Make a right turn at the first traffic light, Alafaya Trail North. Continuing Alafaya Trail North for 0.7 mile, make a right turn (the second traffic light) to University Blvd East, where is the main entrance to the campus. Make a left turn at the first light, Gemini Blvd West. Continuing Gemini Blvd West for 0.2 mile, Parking Garage I is on your right. We have obtained conference parking permit (free of charge) for conference participants to park in Parking Garage I.

Parking Garage I is 0.4 mile away from Classroom Building 2 (about an 8-minute walk).

Thank You!