SMB MEPI - PDEE Subgroup Virtual Mini-Conference Schedule and Abstracts

Link to Schedule on Conference Website

Sunday, Feb 26

11:45 - 12:00 PM	*Zoom Room Open*
12:00 - 12:10 PM	*Opening Remarks*
12:10 - 1:00 PM	*Plenary Talk:* Folashade Agusto, University of Kansas
1:00 - 1:20 PM	Tarun Mohan, Texas Christian University
1:20 - 1:40 PM	Devdatta Adhikary, Indian Statistical Institute
1:40 - 2:00 PM	Jaewook Joo, Case Western Reserve University
2:00 - 2:10 PM	*Break*
2:10 - 2:30 PM	Omar Saucedo, Virginia Tech
2:30 - 2:50 PM	Rodolfo Guadalupe Blanco Rodriguez, University of Idaho
2:50 - 3:10 PM	Seoyun Choe, University of Central Florida
3:10 - 3:20 PM	*Break*
3:20 - 4:00 PM	*Panel: Collaborations between PDEE-EPI*

Monday, Feb 27

11:45 - 12:00 PM	*Zoom Room Open*
12:00 - 12:50 PM	*Plenary Talk:* Benito Chen-Charpentier, University of Texas, Arlington
12:50 - 1:10 PM	Michael Cortez, Florida State University
1:10 - 1:30 PM	Claus Kadelka, Iowa State University
1:30 - 1:50 PM	Jing Jiao, Florida State University
1:50 - 2:00 PM	*Break*
2:00 - 2:20 PM	Rebecca Tyson, University of British Columbia Okanagan
2:20 - 2:40 PM	Susmita Sadhu, Georgia College & State University
2:40 - 3:00 PM	Lale Asik, University of the Incarnate Word
3:00 - 3:10 PM	*Break*
3:10 - 4:00 PM	*Working Group Session I*

Tuesday, Feb 28

11:45 - 12:00 PM	*Zoom Room Open*
12:00 - 12:50 PM	*Plenary Talk:* Brandon Ogbunu, Yale University
12:50 - 1:10 PM	John Jungck, University of Delaware
1:10 - 1:30 PM	Pablo Cárdenas, Massachusetts Institute of Technology
1:30 - 1:50 PM	Yoav Ram, Tel Aviv University
1:50 - 2:00 PM	*Break*
2:00 - 2:20 PM	Woldegebriel Assefa Woldegerima, York University
2:20 - 2:40 PM	Benjamin Levy, Fitchburg State University and NOAA
2:40 - 3:00 PM	Chapin S. Korosec, York University
3:00 - 3:10 PM	*Break*
3:10 - 4:00 PM	*Working Group Session II*
4:00 - 4:10 PM	*Closing Remarks*

Sunday, February 26th

Sunday, February 26th
12:10 - 1:00 PM EST
Plenary Talk

Double whammy:
Exploring the effects of prescribed fire and rising temperature on tick-borne diseases

Folashade Agusto
University of Kansas

Abstract

In recent times tick ranges have been expanding due in part to rising temperatures as consequence of climate change, thereby increasing the risks and prevalence of tick-borne illnesses across the country. Thus, it is vital to find practical ways of managing tick populations. Prescribed fires are common form of land management practices; it is time and cost-efficient when applied across large amounts of land. In this seminar, I investigate the effects of prescribed fire intensity and the duration between burns on the prevalence of tick-borne illnesses as temperature rises. Using stage-structured tick-host models with impulsive differential equations our results indicate that prescribed fire intensity has a larger impact in reducing disease prevalence than frequency between burns. Exploring the use of prescribed burns in preventing the establishment of ticks in new areas shows that fewer burns are ineffective at preventing their establishment because ticks can recover relatively quickly following a burn but frequent, long-term prescribed burns can slow and possibly prevent their establishment.

Sunday, February 26th
1:00 - 1:20 PM EST

How easy is it for a virus to escape multiple vaccines?

Tarun Mohan
Texas Christian University

Abstract

When the novel coronavirus, SARS-CoV-2, was first detected in late 2019, and began its rapid spread around the world, scientists raced to develop vaccines against the virus. Within about a year of the first detection, a number of different vaccines were approved for emergency use. Unfortunately, the recent omicron variant has shown that SARS-CoV-2 has the potential to evade some of the immune protection provided by vaccines. Some researchers have speculated that having multiple vaccines with different mechanisms, potentially eliciting different arrays of antibodies and T cells, would make it harder for the virus to mutate enough to evade all the vaccines, even if some of the vaccines became less effective against certain strains. Here, we use mathematical modeling to investigate whether having multiple vaccines decreases the likelihood of having a virus that can fully evade all vaccines. Using a series of models with an increasing number of vaccines, we find that vaccination rates need to be higher in order for a virus to escape all vaccines as the number of vaccines increases. These models provide a basis for studying the dynamics of an epidemic in the presence of multiple vaccines.

Sunday, February 26th
1:20 - 1:40 PM EST

A novel index for identifying priority species: An illustration through plankton data of the Bay of Bengal

Devdatta Adhikary
Indian Statistical Institute

Abstract

Biodiversity around us is under threat due to pollution and other human activities. The functional linkage among species stabilizes an ecosystem's equilibrium. Therefore, the loss of any species from any ecosystem hampers other ecosystems' stability. So, the conservation of biodiversity, as well as the ecosystem, is the need of the hour. One of the basic steps to save the biodiversity of any ecosystem is the bio-monitoring of a habitat. Therefore, species prioritization is an inevitable aspect in determining which species should be selected for making conservation policies. Existing literature reveals that threatened or endangered species are mainly selected as priority species. Even the established priority indices also target the threatened or endangered species. But the importance of the species that are abundant and persistent in an ecosystem is undeniable, as they have an enormous impact on the food web of the ecosystem. The conventional method of priority species identification for any habitat is solely based on abundance data. But only abundance data fails to capture the effect of seasonal fluctuations and repeated appearance of species in the ecosystem. So, we propose a modified measure of fitness to overcome the above lacunae. Our new measure can capture the dynamics of both dominating and migratory or weather-specific species fitness functions. The performance of the proposed method is analyzed through primary field data on plankton abundance from Digha, West Bengal - Talsari, Odisha region, Bay of Bengal. Only the abundance-based method identified 45 top-priority species in, which many important species were missing. However, using this modified metric, we are able to identify 51 plankton species as top-priority species from 131 species for the Bay of Bengal ecosystem. Hence, the modified metric has global application in identifying the top priority fitted species for any ecoregion from terrestrial to aquatic environments. Since, we are confident that this present study along with the extensive literature reviews and news searches can enlighten the research arena of conservation biology.

Sunday, February 26th
1:40 - 2:00 PM EST

Accelerating population extinction through temporal modulation counter-diabatic control

Jaewook Joo
Case Western Reserve University

Abstract

Stochastic fluctuations are ubiquitous in natural and man-made systems. Those fluctuations can give rise to dramatic, unexpected, oftentimes catastrophic, and dynamical consequences such as a sudden population collapse to extinction. Those events are very rare and never happen on a realistic time scale. We are keenly interested in speeding up such a fluctuation-induced rare event of population extinction. We consider a stochastic Verhulst population growth model in a time-modulated environment and use WKB large deviation theory for the analysis. In the absence of time-modulated perturbation the stochastic system transits from a metastable state to an extinction state along the fluctuation-induced optimal path. This optimal path is a heteroclinic orbit connecting a stable manifold of the extinction state with an unstable manifold of the fluctuation-induced state. As the time-modulated perturbation is turned on, the extinction accelerates, but such an acceleration is only limited to a small amplitude temporal modulation beyond which the optimal path is disconnected, making the fluctuation-induced extinction implausible. We use the counter-diabatic control to enforce the connectedness of the optimal path during large amplitude temporal modulation of the environment, achieving the maximally accelerating the population extinction.

Sunday, February 26th
2:10 - 2:30 PM EST

Impact of Data Structure, Availability, and Noise Distribution on Practical and Structural Identifiability of an SEIR Model

Omar Saucedo
Virginia Tech

Abstract

With the increasing practice of using biological data to assess and parameterize theoretical models, it is vital to understand the conditions under which we can reliably recover model parameters from available data. This is particularly important in the context of epidemiological data, which may gradually become available alongside an emerging outbreak. In this work, we consider an SEIR compartmental model, with three unknown parameters. Our goal is to assess how different methods and resolutions of data collection determine parameter identifiability. We consider the impact of the frequency and duration of observations, for different types of observed data (prevalence, incidence, and cumulative incidence). We utilize both Monte Carlo simulations and correlation matrices to assess parameter identifiability under these conditions.

Sunday, February 26th
2:30 - 2:50 PM EST

Stochastic network modeling to quantify co-evolution during interventions

Rodolfo Guadalupe Blanco Rodriguez
University of Idaho

Abstract

We learn with COVID-19 that public health measures can vary widely from country to country, with some implementing intensely restrictive lockdowns while others focusing on mass vaccination campaigns with obvious logistical constraints. However, new variants are evolving in different countries with the potential to escape the effects of current vaccines. To explore scenarios for lockdowns, evolution, and vaccination, we develop a stochastic computational framework to simulate the transmission of two variants in different network models. We implemented three types of stochastic networks: small-world Watts-Strogatz, completely random Erdős-Renyi, and scale-free Barabási Albert. We averaged 100 stochastic simulations with 1 million nodes for each case study. Our results show that the networks with highly connected nodes reach higher outbreak peaks than networks with a Poisson degree distribution, and the peak values are independent of the infectivity of the variant. We also found that confinement for 15-day intervals can reduce the peak number of infected nodes, especially in small-world networks. Finally, we tested the duration of a vaccination campaign and found that a 20-day vaccination campaign yields better results than a 10-day campaign. Vaccination coverage for at least 50% of the population is necessary to have low cases of infection, even considering more infectious variants of the virus.

Sunday, February 26th
2:50 - 3:10 PM EST

The impact of travel restriction on patterns of disease dynamics for multi-patch models

Seoyun Choe
University of Central Florida

Abstract

After bigging the COVID-19 pandemic around the world, travel restriction policies internationally and domestically have been important issues. In mathematical epidemiology, there are several modelings about the impact of varying residence times or travel restrictions, such as lockdowns on the infectious disease dynamics in a heterogeneous environment. We set two kinds of multi-patch models: Lagrangian and Euler models. For the Euler model, we explored how the travel frequency (restriction) affects the pattern of disease dynamics for a multi-patch model. For the Lagrangian model, we proved that the basic reproduction number is monotonically decreasing with respect to the travel restriction factor. Also, we derived the final size relation by using the weighted geometric mean. Numerical simulations illustrate that the final size of the outbreak depends on the travel restriction measure as well as the transmissibility. Moreover, we investigated patch-specific optimal treatment strategies.

Monday, February 27th

Plenary Talk
Monday, February 27th
12:00 - 12:50 PM EST

Sensitivity analysis and its application to models in Mathematical Biology

Benito Chen-Charpentier
University of Texas, Arlington

Abstract

Most models in Mathematical Biology depend on parameters. Usually these parameters are not easy to determine accurately. The also have variations due to the variability in the biological objects been studied. It is very important to determine how much the solutions of a mathematical model of a biological process change due to changes in the values of the parameters. One way is to use sensitivity analysis. That is to estimate how much the solution varies with changes in the parameters. There are two types of sensitivity analysis: local and global. In this talk we explore the use of local and different versions of global sensitivity analysis as applied to a model in Mathematical Biology.

Monday, February 27th
12:50 - 1:10 PM EST

Comparing the effects of host species richness of different metrics of disease

Michael Cortez
Florida State University

Abstract

Biodiversity is changing across the globe, with species extirpation in some regions and invasions in others. The loss or gain of a host species from a community can increase or decrease the risk of infection and levels of disease in a community. In this talk, I use an SIR-type multi-host model with an environmentally transmitted pathogen to explore how host additions/removals affect disease levels. I use local sensitivity analysis to make predictions about how the effects of the gain or loss of a host species depends on host competence (i.e., the ability of each host species to transmit the pathogen), interspecific interactions between host species (e.g., the strength of interspecific host competition), and the metric of disease (e.g., the number or proportion of infected individuals and the basic reproduction number, R0). My results help explain how community context shapes host species richness-disease relationships and it yields insight about when and why metrics respond differently to changes in host species richness.

Monday, February 27th
1:10 - 1:30 PM EST

Heterogeneities in contact patterns matter: how to account for them

Claus Kadelka
Iowa State University

Abstract

Contact networks are heterogeneous. People with similar characteristics are more likely to interact, a phenomenon called assortative mixing or homophily. Empirical age-stratified social contact matrices have been derived by extensive survey work and used in many recent COVID-19 models. We lack however similar empirical studies that provide social contact matrices for a population stratified by attributes beyond age, such as gender, sexual orientation, or ethnicity.

In this talk, I use ethnic homophily and the problem of identifying optimal strategies to allocate limited COVID-19 vaccines as an example to show that accounting for heterogeneities with respect to attributes beyond age can have a profound effect on model dynamics and predictions. I describe a new method, which uses linear algebra and non-linear optimization, to expand a given contact matrix to populations stratified by binary attributes with a known level of homophily. I conclude by briefly describing more complicated extensions. This new method enables any modeler to account for the presence of homophily with respect to binary attributes in contact patterns, ultimately yielding more accurate predictive models.

Monday, February 27th
1:30 - 1:50 PM EST

How within-host priority effects between specialist and generalist pathogens affect disease risk

Jing Jiao
Florida State University

Abstract

When an individual host is co-infected by multiple pathogens, pathogen fitness within a host can depend on the order of infection. Previous studies demonstrated that this priority effect at the individual level can scale up to influence population-level disease dynamics. Here, we use epidemiological models to explore how priority effects mediate the interactions between generalist and specialist pathogens. We analyzed a two-host-two-pathogen SI-type model where the pathogens are environmentally transmitted (e.g., spore-based transmission), (ii) the focal host can be infected by a specialist pathogen and a generalist pathogen, and (iii) an alternative host can only be infected by the generalist pathogen. Our analysis focuses on how changes in the density of the alternative host alters the focal host's risk of infection by the specialist pathogen (measured as the density of infectious propagules), and how those effects are mediated by within-host priority effects. A motivating example is how the establishment of an invasive host affects a native host's risk of being infected by a specialist pathogen when both the native and invasive hosts can be infected by a generalist pathogen.

We found that: (i) In the absence of priority effects, there is a positive (negative) relationship between alternative host density and the focal host's risk of being infected by a specialist pathogen when the specialist pathogen has higher (lower) fitness in co-infected hosts. (ii) In the presence of a priority effect wherein the specialist pathogen has higher fitness when it is the second to infect a focal host, there is a positive relationship between alternative host density and the risk of being infected by the specialist pathogen. (iii) In the presence of a priority effect wherein the specialist has lower fitness when it is the second to infect a focal host, there is a negative relationship between alternative host density and the risk of being infected by a specialist pathogen. (iv) The priority effect, combined with pathogen infectivity, could lead to unimodal relationships between alternative host and the risk of being infected

by the specialist pathogen. These findings help provide a better understanding of how priority effect via coinfection affect disease risk in multiple-host-multiple-pathogen systems and highlights the importance of coinfection in disease control.

Monday, February 27th
2:00 - 2:20 PM EST

Climate variability and P-Tipping: A new route to extinction for predator-prey cycles

Rebecca Tyson
University of British Columbia Okanagan

Abstract

Cyclic systems can exhibit highly unexpected behaviour when subject to external forcing.¬† Many predator-prey systems are inherently cyclic, and subject to climatic forcing that can also be dominated by certain frequencies.¬† This coupling between two cyclic systems could mean that the observed predator-prey dynamics may be more dependent on the characteristics of the climatic time series than is generally suspected.¬† In particular, we ask if the predicted changes to the colour and frequency of environmental noise could put cyclic predator-prey systems at risk.¬† We investigate this question by studying two paradigmatic predator-prey models, the Rosenzweig-MacArthur and Leslie-Gower-May models, both with an Allee effect, under climatic forcing modeled via changes in prey productivity (prey growth and carrying capacity).

Our analysis of these models uncovers a counterintuitive behaviour, which we call phase tipping or P-tipping, where tipping to extinction does occur under climate variability, but only from certain phases of the predator-prey cycle, and only when there is a sudden drop in productivity.¬† This tipping occurs even if the predator-prey cycle exists and is stable for all values of the climate parameters.¬† Intuitively, P-tipping arises because a fixed drop in prey resources has distinctively different effects when applied during the phases of the oscillations with the fastest growth and the fastest decline of prey. We confirm the relevance of P-tipping to real ecosystems by using parameter values consistent with the snowshoe hare and Canada lynx predator-prey system and climate in the boreal and deciduous-boreal forests of North America.

Monday, February 27th
2:20 - 2:40 PM EST

A novel mechanism for detecting an early warning signal of population collapse in a two-timescale predator-prey model.

Susmita Sadhu
Georgia College & State University

Abstract

In this talk, I will discuss a method of detecting an early warning signal of a sudden population collapse in a predator-prey model featuring two-timescales. The model under consideration studies the interaction between two species of predators competing for their common prey. In a parameter regime near *singular Hopf bifurcation*, the system exhibits bistability between a periodic attractor and a boundary equilibrium state. To determine whether a solution that starts in a vicinity of the coexistence equilibrium approaches the periodic attractor or the point attractor, we reduce the system to a suitable normal form, which is valid near the singular Hopf bifurcation, and study its geometric structure. A key component of our study includes an analysis of the transient dynamics, characterized by their rapid oscillations with a slow variation in amplitude. The analysis is then used to devise a method for identifying an early warning signal, significantly in advance, of a future crisis that could lead to extinction of one of the predators. This is a joint work with Dr. S.C. Thakur from Auburn University.

Monday, February 27th
2:40 - 3:00 PM EST

Reconciling contrasting effects of nitrogen on pathogen transmission and host immunity using stoichiometric models

Lale Asik
University of the Incarnate Word

Abstract

Hosts rely on the availability of nutrients for growth, as well as for their defense against pathogens. At the same time, changes in primary producer nutrition can alter the dynamics of pathogens that rely on their host for reproduction. Enhanced nutrient loads may thus promote faster pathogen transmission through increased pathogen reproduction, as well as through higher host biomass that stimulates density-dependent transmission. However, this effect may be reduced if hosts allocate a growth-limiting nutrient to pathogen defense. In canonical disease models, transmission is not a function of nutrient availability, while this is required to mechanistically understand their response to changes in the environment. Here, we explored the implications of nutrient-mediated pathogen infectivity and host immunity on infection outcomes using a stoichiometric disease model that explicitly integrates the contrasting dependencies of pathogen infectivity and host immunity on nitrogen (N). Our findings reveal dynamic shifts in host biomass build-up, pathogen prevalence, and force of infection, along N supply gradients with N-mediated host infectivity and immunity, compared to a model where the transmission rate was fixed. We show contrasting responses in pathogen performance with increasing N supply between N-mediated infectivity and N-mediated immunity, revealing an optimum for pathogen transmission at intermediate N supply. This is caused by N limitation of the pathogen at low N supply and by pathogen suppression via enhanced host immunity at high N supply. By integrating both nutrient-mediated pathogen infectivity and host immunity into a stoichiometric model, we provide a theoretical framework that is a first step in reconciling the contrasting role nutrients can have on host-pathogen dynamics.

Tuesday, February 28th

Plenary Talk
Tuesday, February 28th
12:00 - 12:50 PM EST

Proteins and Words, Needles and Birds: Disease dynamics in ecological and evolutionary context

Brandon Ogbunu
Yale University

Abstract

In this presentation, I offer presentations covering two areas of my research program: (i) the molecular evolution of disease and (ii) the ecology of disease transmission. Topics range from the evolution of antimicrobial resistance, to diseases spread in populations of people who inject drugs, and waterfowl as vectors for bacterial pathogens.

Tuesday, February 28th
12:50 - 1:10 PM EST

Phylogenetic Network Analysis of a COVID Superspreader Event

John R Jungck
University of Delaware

Abstract

Some epidemiological models account for super-spreaders, which are people or events that transmit COVID-19 to many people (Jha et al., 2020), resulting in clusters of infectious cases all originating from few starting points (Ndairou et al., 2020). We hypothesized that this super-spreading could be detected and visualized by sampling nucleotide sequences from a population and analyzing them phylogenetically to identify closely related sequences within a short time frame. We constructed a SPLITSTREE phylogenetic network analysis of COVID sequences from a Delaware population that were collected over a one week time frame when a new variant was being initially observed. Super-spreading provides a plausible explanation of the large number of identical sequences observed in our analysis, positing, for example, that many of the 90 infectious cases highlighted in one week could be traced back to an earlier case which may have been one recorded in a previous week.Within a genetically diverse dataset of 401 nucleotide sequences, large amounts of duplicate Spike sequences were present; it's highly likely based on the lengths – 3.8k bases –of these nucleotide sequences that identical sequences originated from the same or highly similar genetic sources. If this data were corroborated by patient data and contact tracing of identical sequences, it would confirm the likely hypothesis that super-spreading is visible at the nucleotide level of a dataset and can be identified using phylogenetic analysis.

Tuesday, February 28th
1:10 - 1:30 PM EST

Genomic models describe epidemiological determinants of pathogen evolution

Pablo Cárdenas
Massachusetts Institute of Technology

Abstract

Over the past century, mathematical models have become a powerful tool in epidemiology. Models provide a practical way to generate and test hypotheses about the spread of infections by comparing the results of simulations with real-world data. However, mass genomic sequencing of pathogens has provided an entirely new kind of epidemiological data. New modeling tools are therefore needed to be able to work with genomic information and allow us to ask questions about how the spread and evolution of pathogens influence each other. In light of this, we have developed Opqua (github.com/pablocarderam/opqua), a computational modeling framework that integrates pathogen epidemiology and genomic evolution. Unlike previous tools, Opqua allows pathogen evolutionary dynamics to affect the spread and epidemiology of the infection in real-time, according to user-defined, arbitrary fitness landscapes. Here, we use Opqua to study how epidemiological context and pathogen biology can shape evolution. We confirm that competition between pathogens can limit evolution across fitness valleys in high transmission environments and find that low transmission, host mobility, and complex pathogen life cycles facilitate reaching new adaptive peaks, thanks to population bottlenecks and decoupling of selective pressures. Using intra-host and population models with Opqua, we establish that the size of transmission bottlenecks and the temporal dynamics of intra-host growth can generate qualitatively distinct regimes of pathogen evolution. These regimes differ on whether they facilitate evolution going up or down fitness gradients. We show the effects of this by recapitulating real sequencing data in Opqua simulations of well-studied, localized SARS-CoV-2 outbreaks. All in all, we demonstrate that models intertwining epidemiology and evolution can help understand processes such as the emergence of novel pathogen genotypes with higher transmission or resistance to treatment. These results affirm the potential of genomic epidemiological modeling as a tool in infectious disease research.

Tuesday, February 28th
1:30 - 1:50 PM EST

Aneuploidy can be an evolutionary detour on the path to genetic adaptation

Yoav Ram
School of Zoology
Tel Aviv University

Abstract

Aneuploidy is common in eukaryotes, often leading to decreased growth and fitness. However, evidence from yeast and fungi, as well as human tumour cells, suggests that specific aneuploidies can be beneficial under stressful conditions and facilitate adaptation. Yona et al. (2012) have demonstrated in an evolutionary experiment with yeast that populations evolving under heat stress become aneuploid, only to later revert back to euploidy after genetic mutations have accumulated. It has therefore been suggested that aneuploidy serves as a 'stepping stone' on the path to adaptation.

To test this hypothesis, we developed an evolutionary model with both aneuploidy and mutation, and fit it to the results of the experiment using a Bayesian inference framework. We then predicted the genotype frequency dynamics during the experiment, demonstrating that the majority of the evolved euploid population likely did not descend from aneuploid cells, but rather directly from the euploid wild-type population. Surprisingly, these results agree with DNA sequencing results that show that mutant alleles common in aneuploid cells are uncommon in the evolved euploid population. Our model further predicts that if the experiment was repeated with smaller populations, then a larger fraction of the evolved population would descend from aneuploid cells.

Thus, we suggest that aneuploidy can be an inevitable evolutionary 'detour' rather than a 'stepping stone': it can delay, rather than facilitate, the adaptation of the population, and cells that become aneuploid may leave less descendants compared to cells that remain diploid.

Tuesday, February 28th
2:00 - 2:20 PM EST

Mitigating Co-circulation of seasonal influenza and COVID-19 pandemic in the presence of vaccination: A mathematical modelling approach

Woldegebriel Assefa Woldegerima
York University

Abstract

The co-circulation of two respiratory infections with similar symptoms in a population can significantly overburden a healthcare system by slowing the testing and treatment. In this work, we proposed a mathematical epidemic model to investigate the co-circulation dynamics of COVID-19 and influenza, under different scenarios of influenza vaccine coverage, COVID-19 vaccine booster coverage and efficacy, and testing capacity.

We assessed the effects of the co-circulation of the two respiratory pathogens. We investigated the required minimal and optimal coverage of COVID-19 booster(3rd) and 4th dose, in conjunction with the influenza vaccine to avoid the coincidence of infection peaks for both diseases in a single season. We show that the testing delay brought on by the high number of influenza cases impacts the dynamics of influenza and COVID-19 transmission. The earlier the flu season's peak and the greater the number of infections with flu-like symptoms, the greater the risk of flu transmission, which slows down COVID-19 testing, resulting in the delay of complete isolation of COVID-19 patients who have not been isolated before the clinical presentation of symptoms and have been continuing their normal daily activities. Simulation results emphasize the importance of vaccine uptake for preventing infection, severe illness, and hospitalization at the individual level and for disease outbreaks control at the population level to avoid putting strain on already weak and overwhelmed healthcare systems.

Tuesday, February 28th
2:20 - 2:40 PM EST

Is climate change biasing fishery survey abundance indices? A simulation study starting point.

Benjamin Levy
Fitchburg State University and the National Oceanic and Atmospheric Administration

Abstract

NOAA Fishery stock assessment scientists use abundance estimates derived from stratified random bottom trawl data. To accurately represent true abundance, catches of a species must contain a low enough noise level to allow for a discernible pattern and all strata in which the population exists should be sampled. These assumptions might be violated given enough noise in the sampling process and/or climate change causing a population to move into previously uninhabited strata. Using the R package MixFishSim, we have developed data-driven spatial models for Yellowtail Flounder, Cod, and Haddock on Georges Bank to allow examination of these assumptions through simulation. Movement rates combine species-specific static habitat preferences with temperature tolerances. Habitat preferences were derived from niche models relating bottom trawl catches to environmental covariates. A repeating yearly temperature pattern produces repeating spatial biomass distributions in a given week, while a temperature gradient that increases on average over time results in spatial preferences that evolve throughout a given simulation. We examined several temperature scenarios and population trends to create simulated

spatial time series datasets for each species. We then conduct stratified random sampling on model output and compare abundance estimates using the stratified mean to those using a common model-based approach that allows inclusion of environmental covariates (VAST). Our focus is on the ability of contemporary indexing methods to track population trends under shifting spatial preferences. Results highlight conditions that can result in biased indexing estimates and demonstrate the value of including spatial covariates in abundance estimates.

Tuesday, February 28th
2:40 - 3:00 PM EST

Longitudinal immunological outcomes from three doses of COVID-19 vaccines in people living with HIV: antibodies, memory-B cells, cytokines, and a novel within-host immunological model

Chapin S. Korosec[1,2], Vitaliy Matveev[3], Mario Ostrowski[3], Jane M. Heffernan[1,2]

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[3]Department of Medicine, University of Toronto, Toronto, Ontario, Canada

Abstract

People living with HIV (PLWH) older than age 55 have an enhanced risk of complications from SARS-CoV-2 infection. It is further unclear whether multiple standard doses of COVID-19 vaccines elicit a durable immunity in this population or whether the vaccines can destabilize HIV reservoirs. In this talk I will discuss our unpublished work where we followed n = 91 PLWH aged 55+ and n = 23 age-matched HIV- individuals over a period of 48 weeks following COVID-19 dose one, capturing longitudinal immunological outcomes from two subsequent booster doses. I will introduce the longitudinal immunological findings for IgG, memory-B cells, and cytokines (IFNg and IL2). I will then motivate our novel within-host immunological model which couples these quantities together, and the findings of our fits to determine dose-dependent decay rates and half life values. Model fit findings, practical identifiability concerns, and biological implications of the within-host modelling approach will be discussed.
