Special Session 70: Modeling and Dynamics of Infectious Diseases

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The mini-symposium will focus on the modeling and dynamics analysis of mathematical models for the spread of emerging and re-emerging infectious diseases in populations. An essential feature of the mini-symposium is the emphasis on the use of state-of-the-art techniques, theories and new applications associated with the use of dynamical systems in modeling the spread of diseases in populations. Current modeling and mathematical challenges will also be discussed.

Effects of mixed Plasmodium malariae and Plasmodium falciparum infections on the dynamics of malaria

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Malaria continues to threaten the lives of people around the world, despite being preventable and treatable. Different factors affect the epidemiology and clinical outcomes of malaria. To investigate importance of mixed species infection, in the spread of malaria, a deterministic model for dual infection of Plasmodium malariae and Plasmodium falciparum is presented. Qualitative analysis of the model is performed. In addition to the disease free equilibrium, we show that there exists a boundary equilibrium corresponding to each species. The isolation reproductive number of each species is computed as well as the reproductive number of the full model. Conditions for global stability of the disease free equilibrium as well as local stability of the boundary equilibria are derived. The model has an interior equilibrium which exists if at least one of the isolation reproductive numbers is greater than unity. Among the interesting dynamical behaviors of the model, the phenomenon of backward bifurcation where stable boundary equilibrium coexists with a stable interior equilibrium, for a certain range of the associated invasion reproductive number less than unity is observed. Results from analysis of the model show that, when cross-immunity between the two species is weak, there is a high probability of coexistence of the two species and when cross-immunity is strong, competitive exclusion is high. Further, an increase in the reproductive number of species i increases the stability of its boundary equilibrium and its ability to invade an equilibrium of species j.

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Mathematical Analysis of Chikunguya Model with Time Delay

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Chikungunya is a viral disease that is spread by mosquitoes. The disease, which resembles dengue fever, is spread in Africa, Asia and the Indian subcontinent (and also, in recent decades, in Europe and the Americas). It accounted for over 1.25 million cases in India alone in 2006. In this talk, a deteministic model with time delay (accounting for the incubation period of the disease) is used to study the transmission dynamics of the disease in a given population. Qualitative analyses of the model reveals the presence of the phenomenon of backward bifurcation in the model when the associated reproduction number (R_0) is less than unity (this, in turn, makes disease control difficult). Simulations are carried out to investigate the effect of time delay on the disease dynamics (and control). Analysis of the model shows that, the disease will persist, whenever $R_0 1$. Furthermore, an increase in the length of incubation period, increases the chikungunya burden in the community if a certain threshold quantities, denoted by Δ_b and Δ_v are positive.

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Backward bifurcations in disease transmission models

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Backward bifurcation, a phenomenon where a stable disease-free equilibrium typically co-exists with a stable endemic equilibrium, is known to have important consequences on the effective control or persistence of the disease in a population. The talk discusses some common and new causes of backward bifurcation in Kermack-McKendrick-type deterministic compartmental models for the spread of some emerging and re-emerging diseases.

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The effects of pre-existing immunity on infectious diseases

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Immune system memory (or immunity) is gained as a result of primary infection or vaccination, and boosted by later vaccinations or secondary infections. The components of immune system memory (i.e. T-cells, antibodies) are primed to react and fight quickly against future infections of strains related to those previous experienced by an individual. The existence of immunity, thus affects an individual, but will also affect the spread of a disease in a population by reducing transmission. The extent of the effects of immunity on an individual, and how this translates to the population are not well understood. In this talk we will focus on different aspects of immunity with different diseases as case studies.

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Modeling recent outbreaks of Dengue Fever in Pakistan

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Dengue is a mosquito borne disease prevalent in the tropical and subtropical countries including Pakistan. We evaluate Pakistans dengue situation and compare this to epidemic trends in different countries including Brazil. We propose a deterministic mathematical model incorporating the transmission of different strains of dengue and identify a number of factors related to the spread of the disease in Pakistan. Using data from recent outbreaks we determine the value of basic reproductive number and compare it with the basic reproductive number evaluated in different countries including Brazil.

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Mathematical insights in evaluating effectiveness of interventions for HIV prevention

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Mathematical models have been used to simulate HIV transmission and to study the use of chemoprophylaxis among men-who-have sex with men. Often a single intervention outcomes based on cumulative number or fractions of infections prevented, on reduction in HIV prevalence or incidence have been used to evaluate the effectiveness of PrEP interventions. These indicators express a wide variation over time and often disagree in their forecast on the success of the intervention. We develop a deterministic mathematical model of HIV transmission to evaluate the public-health impact of oral PrEP interventions, compare PrEP effectiveness with respect to different evaluation methods and analyze its dynamics over time. We compare four traditional evaluation methods including relative reduction in HIV prevalence and incidence which are considered to avoid the ambiguity associated with commonly used indicators based on the absolute number of prevented infections. We consider two additional methods which estimate the burden of the epidemic to the public-health system. We also investigate the short term and long term behavior of these indicators and the effects of key parameters on the expected benefits from PrEP use.

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A data driven spatiotemporal rabies model for skunk and bat interaction in Northeast Texas

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We formulate a simple partial differential equation model in an effort to qualitatively reproduce the spread dynamics and spatial pattern of rabies in northeast Texas region with overlapping reservoir species (skunks and bats). Most existing models ignore reservoir species or model them with patchy models by ordinary differential equations. In our model, we incorporate interspecies rabies infection in addition to rabid population random movement. We apply this model to the confirmed case data from northeast Texas with most parameter values obtained or computed from existing literatures. Results of simulations using both the skunk only model and our skunk and bat model demonstrate that the model with overlapping reservoir species more accurately reproduces the progression of rabies spread in northeast Texas.

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The impact of an imperfect vaccine and Pap cytology screening on the transmission dynamics of Human Papillomavirus and Cervical Cancer.

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The talk will address the problem of the transmission dynamics of human papillomavirus in a population. A new sex-structured model, which takes into account the associated multiple cervical intraepithelial neoplasia stages, will be used to assess the combined impact of Pap cytology screening and a vaccine on the disease dynamics and the associated dysplasia. Rigorous qualitative analysis will be presented. Simulation results, using a realistic set of parameter values, will also be discussed.

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Avian influenza: Modeling and implications for control

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At present H5N1 avian influenza is a zoonotic disease where the transmission to humans occurs from infected domestic birds. Since 2003 more than 500 people have been infected and nearly 60% of them have died. If the H5N1 virus becomes efficiently human-to-human transmittable, a pandemic will occur with potentially high mortality. A mathematical model of avian influenza which involves human influenza is introduced to better understand the complex epidemiology of avian influenza and the emergence of a pandemic strain. The model is parameterized based on demographic and epidemiological data on birds and humans. The differential equation system faithfully projects the cumulative number of H5N1 human cases and captures the dynamics of the yearly cases. The model is used to rank the efficacy of the current control measures used to prevent the emergence of a pandemic strain. We find that culling without repopulation and vaccination are the two most efficient control measures each giving 22% decrease in the number of H5N1 infected humans for each 1% change in the parameters. Control measures applied to humans, however, such as wearing protective gear, are not very efficient, giving less than 1% decrease in the number of H5N1 infected humans for each 1% change in the parameters. Furthermore, we find that should a pandemic strain emerge, it will invade, possibly displacing the human influenza virus in circulation at that time. Moreover, higher prevalence levels of human influenza will obstruct the invasion capabilities of the pandemic H5N1 strain. This effect is not very pronounced, as we find that 1% increase in human influenza prevalence will decrease the invasion capabilities of the pandemic strain with 0.006%.

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General comments on the construction of mathematical models for predator-prey interactions

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Mathematical modeling of interacting populations can provide valuable insights into important features of these systems. Further, when combined with relevant mathematical analysis and data, these models may also give rise to a fundamental understanding of the nature of the systems. However, a critical issue is that a priori no universal set of rules exist for the (unique) construction of the models. In spite of this weakness, broad classes of mathematical models predict essentially the same general features for the properties of their solutions. The main purpose of this presentation is to discuss these issues and, in particular, examine the role of dynamic consistency in the modeling construction process. Explicit examples will be given to illustrate some consequences of the application of dynamic consistency.

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Evaluation of diagnostic test for Lymphatic Filariasis in Papua New Guinea using a mathematical model

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Lymphatic Filariasis (LF) is a debilitating disease endemic in developing countries around the world. The prevalence of the LF in Papua New Guinea (PNG) is considered the highest in the world. The most feasible strategy for LF in many countries has been Mass Drug Administration (MDA). But in PNG previous rounds of MDA have not been successful in eliminating LF. Optimal use of such control programs depends on our ability to accurately identify infected individuals after populations have received multiple rounds of MDA. Available diagnostic tests have low and varying efficacy and the value of the tests for monitoring the end points of transmission has not been validated. This study provides a framework to test the value of new and existing diagnostic/monitoring tools as prevalence decreases because of MDW. In our model analysis, we use data

from the ongoing and previous MDA field trials as well as literature review from the PNG. The talk will show results of the impact on the elimination of Lymphatic Filariasis of the errors in five available tests that include microfilarial level-, antigen- and antibody-based tests.

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Understanding cholera transmission and interventions using mathematical models

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Cholera, a preventable disease, remains a global cause of morbidity and mortality, capable of causing periodic epidemic disease. Implementation of appropriate disease control efforts, including vaccination, requires an understanding of transmission dynamics, which may be best quantified by mathematical models. We explore the utility of mathematical models in understanding transmission dynamics of cholera, and in assessing the magnitude of interventions necessary to control epidemic disease

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Mathematical models of binge drinking, heroin epidemics, anorexia and bulimia

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We introduce some mathematical models in the Dynamics of Infectious Diseases. In particular, we first develop a two-stage (four component) model for youths with serious drinking problems and their treatment. The youths with alcohol problems are split into two classes, namely those who admit to having a problem and those who do not. We study the stability of the equilibria of a model of heroin epidemics. Then, we propose a mathematical model to study the evolution of the number of anorexic and bulimic people, by analyzing the effect of an education coefficient and the possible influence of media on the spread of eating disorders. Finally, epidemic models with evolution are considered where the diffusion of individuals is influenced by intraspecific competition pressures and are weakly affected by different classes. The nonlinear stability of some of the models is proved by introducing new Liapunov functions in the PDEs model.

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Impact of enhanced Malaria control on the competition between Plasmodium falciparum and Plasmodium vivax

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The primary focus of malaria research and control has been on P. falciparum, the most severe of the four Plasmodium species causing human disease. However, there is substantial overlap between the spatial distributions of Plasmodium falciparum and Plasmodium vivax. We developed a mathematical model describing the dynamics of P. vivax and P. falciparum in the human and mosquito populations and fit the model to clinical case data to understand how improving malaria interventions affects the competition between the two species. We addressed the uncertainty in parameter estimates by performing a parametric bootstrapping procedure. This procedure predicted that P. vivax outcompeting P. falciparum is the most likely outcome based on our model. Moreover, the predictions of our model are counter to what one expects based on the case data alone. Although the proportion of cases due to falciparum has been increasing, the fit of our model to the data reveals that this observation is insufficient to draw conclusions about the long-term competitive outcome of the two species.

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Robust uniform persistence and competitive exclusion in a nonautonomous multistrain SIR epidemic model with disease-induced mortality

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We consider a nonautonomous version of the (autonomous) SIR epidemic model of Ackleh and Allen (2003), for competition of n infection strains in a host population, and focus on new questions, mainly regarding robust uniform persistence of the total population, as well as of the susceptible and infected subpopulations. We show that the first two forms of persistence depend entirely on the rate at which the population grows from the extinction state, respectively the rate at which the disease is vertically transmitted to offspring. We also discuss the competitive exclusion among the n infection strains, namely when a single infection strain survives and all the others go extinct. Numerical simulations are also presented, to account for the situations not covered by the analytical results. The approach we developed here is general enough to apply to other nonautonomous epidemic models.

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Global dynamics of cholera models with differential infectivity

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Cholera is a bacterial disease that can be transmitted to humans directly by person-to-person contact or indirectly via the environment (mainly contaminated water). An ordinary differential equation model for cholera dynamics is formulated that includes these two transmission pathways with nonlinear incidence, as well as stages of infection and infectivity states of the pathogen. Lyapunov functions and a graph-theoretic approach are used in the model analysis to show that a basic reproduction number gives a sharp threshold determining whether cholera dies out or becomes endemic. A further model that includes temporary immunity using distributed delays is formulated. Numerical simulations show that oscillatory solutions may occur for parameter values taken from the literature on cholera data, and differential infectivity may alter the existence, amplitude and period of oscillations in cholera prevalence.

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Heterogeneity in the infectiousness of humans in the dynamics of malaria transmission and control.

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The purpose of this talk is to present an exploration and a discussion of a heterogeneous population-level model for assessing the role of gametocyte density on malaria transmission dynamics. Studies have shown a positive correlation between gametocyte density and infectiousness to mosquito, but this correlation was qualified as loose. In fact, other studies have shown that there are low or non-infectious high-density gametocyte carriers and highly infectious low-density gametocyte carriers with infectious individuals having undetected levels of gametocytes. Hence, the successful transmission of parasites to a mosquito is more about "quality" than quantity and infectiousness cannot be simply explained by parasite density. Therefore, we seek to present and analyze a model that takes into account the heterogeneous nature of infectious humans based on "quality" and quantity which may seriously impact the contact rates and infectiousness of humans to mosquitoes and the overall dynamics of malaria transmission

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Mathematical analysis of a virus dynamics model with general incidence rate and cure rate

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The rate of infection in many virus dynamics models is assumed to be bilinear in the virus and uninfected target cells. In this paper, the dynamical behavior of a virus dynamics model with general incidence rate and cure rate is studied. Global dynamics of the model is established. We prove that the virus is cleared and the disease dies out if the basic reproduction number $R_0 = 1$ while the virus persists in the host and the infection becomes endemic if $R_0 > 1$.

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The impact of temperature on the establishment of Lyme disease Tick Vector Ixodes Scapularis

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A stage-structured periodic deterministic model was formulated to simulate the impact of temperature on the tick (Ixodes scapularis) survival and seasonality at Long Point, Ontario, Canada. 7 season-based model coefficients were parameterized using Fourier series analysis by fitting temperature and tick data. We derived the basic reproduction number for the tick population, R_0, as the number of new female adult ticks produced by an index female adult tick when there are no density dependent constraints acting anywhere in the life cycle of the tick population. We confirmed that, both mathematically and numerically, the tick would go to extinction when $R_0 < 1$, and found the successful tick invasion and persistence when $R_0 > 1$. A minimum degree-days threshold in a one year to identify whether the tick population extinct or not was found. A global sensitivity analysis based on popular Latin Hypercube Sampling (LHS) sampling method was performed which demonstrated the mean monthly temperature in June, July, August would more significantly sensitive to establish the tick population. Therefore temperature would significantly influence the risk of tick establishment in a habitat.

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