

---

**Mathematical Epidemiology**  
**Épidémiologie mathématique**

(Org: **Fred Brauer** (University of British Columbia) and/et **Carlos Castillo-Chavez** (Arizona State University))

---

---

**JULIEN ARINO**, University of Manitoba

*Modelling global aspects in vaccination*

Public health systems typically operate at a variety of scales, from local to national authorities. Vaccination policies, for instance, are typically set at the regional or national level. Supranational entities such as the WHO facilitate information flow between member nations but have no regulative role. With the globalization and generalization of travel, it is however becoming increasingly important to consider public health systems on a larger scale than that of nations. In the case of vaccination, it is indeed frequent now that an individual vaccinated (or not) in one jurisdiction will spend a substantial amount of time in other jurisdictions, where vaccination schedules or practices might be very different. I will present a metapopulation framework useful to consider such issues. I will present examples at the international level.

---

**GERARDO CHOWELL**, Georgia State University

*Forecasting epidemic trajectories using simple dynamic models*

Mathematical models provide a quantitative framework with which scientists can assess hypotheses on the potential underlying mechanisms that explain patterns in observed data at different spatial and temporal scales, generate estimates of key kinetic parameters, assess the impact of interventions, optimize the impact of control strategies, and generate forecasts. We review and illustrate a simple data assimilation framework for calibrating mathematical models based on ordinary differential equation models using time series data describing the temporal progression of case counts relating, for instance, to population growth or infectious disease transmission dynamics. We will present results from recent forecasting efforts in the context of emerging infectious diseases using phenomenological and mechanistic models. In the process, we also discuss issues related to parameter identifiability, uncertainty quantification and propagation.

---

**BALTAZAR ESPINOZA**, Arizona State University

*Consequences of short-term mobility across heterogeneous risk environments: the 2014 West African Ebola outbreak*

The role of highly vulnerable regions as the weakest link in a health system is studied on the context of the 2014 West African Ebola Virus Disease (EVD) outbreak. The West African EVD-affected region is envisioned as a polarized area that allows us to study the EVD dynamics exhibited in the zone divided by cordons sanitaires. By using the Lagrangian modeling approach, the local and global dynamics of EVD are assessed in order to study the potential benefits of mobility management as a control measure.

---

**ZHILAN FENG**, Purdue University

*Implications for infectious disease models of heterogeneous mixing on control thresholds*

Mixing among sub-populations, as well as heterogeneity in characteristics affecting their reproduction numbers, must be considered when evaluating public health interventions to prevent or control infectious disease outbreaks. In this talk, we model preferential within- and proportional among-group contacts in compartmental models of disease transmission and derive results for the overall effective reproduction number ( $R_v$ ) assuming different levels of vaccination in the sub-populations. Specifically, we unpack the dependency of  $R_v$  on the fractions of contacts reserved for individuals within one's own subgroup and show that  $R_v$  increases as this fraction increases in a given sub-population. These considerations lead to our proposing the gradient of  $R_v$  with respect to subgroup vaccination fractions as a measure by which to evaluate interventions. Another significant result is that for general mixing schemes, both  $R_0$  and  $R_v$  are bounded below and above by their corresponding expressions when mixing is proportionate and isolated, respectively. This work is based on (1) Glasser et al., Lancet Infectious

Diseases (2016) [http://dx.doi.org/10.1016/S1473-3099\(16\)00004-9](http://dx.doi.org/10.1016/S1473-3099(16)00004-9), (2) Feng et al., J. Theor. Biol. 386 (2015) 177–187, and (3) Poghotanyan et al. J. Math. Biol. (2018) <https://doi.org/10.1007/s00285-018-1216-z>.

---

**ABBA GUMEL**, Arizona State University

*Mathematical Assessment of the Role of Insecticide Resistance on Malaria Dynamics*

The widespread use of indoors residual spraying (IRS) and insecticides-treated bednets (ITNs) has led to a dramatic reduction of malaria burden in endemic areas (with most of the gains attributed to the use of bednets). Unfortunately, such usage has also resulted in the challenging problem associated with the evolution of insecticide resistance in the mosquito population in those areas. Thus, it is imperative to design malaria control strategies, based on using these (insecticides-based) interventions, that reduce malaria burden while effectively managing insecticide resistance in the mosquito population. This talk is based on using a new mathematical model for assessing the population-level impact of wide-scale use of currently-available bednets on the evolution of insecticide resistance and malaria burden in an endemic setting.

---

**MICHAEL LI**, University of Alberta

*Mathematical Modeling of HIV/SIV Infection in Brain*

Understanding HIV-1 replication and latency in different reservoirs is an ongoing challenge in the care of patients with HIV/AIDS. We used mathematical models to quantify the progression and predict the viral dynamics of HIV-1 and SIV infection within the brain during effective combination antiretroviral therapy (cART), and discuss the effects of the "shock-and-kill" strategy for eliminating latent reservoirs.

---

**CESAR MONTALVO**, Arizona State University

*The regressive burden of Water Related infections on income disparity*

Approximately 2.1 billion people in our planet (29

---

**VICTOR MORENO**, Arizona State University

*Understanding the Impact of Social Factors on the Transmission Dynamics of Infectious Diseases*

The possible impacts of environmental dependent risk on disease dynamics within a Lagrangian modeling perspective; where the identity (defined by place of residency) of individuals is preserved throughout the epidemic process are explored. Scenarios include the dynamics of Zika virus and Tuberculosis in two highly distinct idealized environments defined by a parameter that models highly distinctive levels of risk. The underlying assumption is that these two communities are intimately connected due to economics with the impact of various patterns of mobility being incorporated via the use of residency times. Hence, the impact of mobility within these two highly distinct risk environments on the dynamics and control of these diseases is systematically explored. It is found that collaboration and mobility, under some circumstances, can reduce the overall disease burden. In addition, the impact of individuals who refuse to be vaccinated is explored. MMR vaccination and birth rate data from the State of California are used to determine the impact of the anti-vaccine movement on the dynamics of growth of the anti-vaccine sub-population. Results suggest that under realistic California social dynamics scenarios, it is not possible to revert the influence of anti-vaccine contagion.

---

**ANUJ MUBAYI**, Arizona State University

*Modeling, Estimation, and Uncertainty: Challenges associated with Transmission Dynamics and Control of Leishmaniasis*

Neglected tropical diseases (NTD) disproportionately affect more than one billion the world's poorest populations. Leishmaniasis is one of the NTDs, which is the second largest parasite killer ( 50,000 deaths annually, 2010). Mathematical modeling of Leishmaniasis transmission can play a central role in optimizing the utility of limited resources of a region in the presence of scarce and uncertain reported data. The talk will highlight that, when developing models for NTDs use, we need to pay

careful attention to the intrinsic assumptions and ecological conditions embedded within modeling frameworks and how such assumptions have been beneficial to understanding of transmission dynamics of Leishmaniasis and eventual elimination. The study will also review the role of stochastic factors affecting the level of underreporting of the disease incidence.

---

**KYONGAH NAH**, York

*Assessing Systemic and Non-systemic Transmission Risk of Tick-borne Encephalitis*

Estimating the tick-borne encephalitis (TBE) infection risk under substantial uncertainties of the vector abundance, environmental condition and human-tick interaction is important for evidence-informed public health intervention strategies. Estimating this risk is computationally challenging since the data we observe, i.e., the human incidence of TBE, is only the final outcome of the tick-host transmission and tick-human contact processes. The challenge also increases since the complex TBE virus transmission cycle involves the non-systemic route of transmission between co-feeding ticks. Here, we describe the hidden Markov transition process, using a novel TBE transmission-human case reporting cascade model that couples the susceptible-infected compartmental model describing the TBE virus transmission dynamics among ticks, animal hosts and humans, with the stochastic observation process of human TBE reporting given infection. This is based on a joint work with K. Nah, F. Magpantay, A. Bede-Fazekas, G. Röst, A. Trájer, X. Wu and X. Zhang.

---

**JOAN PONCE**, Purdue University

*Dynamics of a childhood disease model with quarantine*

Epidemiological models with exponentially distributed disease stages, although simpler to analyze, have been shown to have limitations in many cases. The model results can be improved by considering more realistic distributions. In this talk, I will present a model with gamma distributions for the exposed and infectious stages to study the impact of isolation on sustained oscillations observed in many childhood diseases. This model is an extension of the model considered in Feng and Thieme (Math Biosc. 1994), in which exponential distributions are assumed for disease stages and it is shown that the threshold value for isolation to generate sustained oscillations is very long for most childhood diseases. By analyzing the stability of the endemic equilibrium and the threshold for Hopf bifurcation of our model, we show that the minimum value for the isolation period required for Hopf bifurcation can be reduced significantly so that the model can be more applicable to many childhood diseases.

---

**KARYN SUTTON**, Institute for Disease Modeling

*The Role of Social Distance and Infectiousness in TB Transmission*

TB transmission is determined by an individual's infectiousness throughout the course of disease, combined with the exposure of other susceptible individuals. The course of an individual's infectiousness remains poorly understood and is likely highly variable. Efforts to accelerate the worldwide decline in TB, the deadliest infectious disease today, are primarily focused on early diagnosis and effective chemotherapies. Mathematical models, which are used to inform intervention strategies, largely assume uniform infectiousness and equal exposure between susceptible and infected individuals, neglecting the potentially powerful effects of social structure. We extend an individual-based TB transmission model to incorporate social distance, with differential exposure rates representing social structures (e.g., peers or familial relationships) or physical distances (e.g., household members, neighbors, community members). We discuss differences in transmission under multiple hypothesized social structures and assumed courses of individual infectiousness. We discuss the compensating changes needed in TB transmission parameters to reproduce the same prevalence trends, along with implications for further recommended intervention strategies.

---

**SHERRY TOWERS**, Arizona State University

*Assessing the average length of infection chains in an outbreak of infectious disease*

Analytical expressions for the reproduction number,  $R_0$ , and final size have been obtained in the past for an extremely wide variety of mathematical models for infectious disease spread. However, what has so far not been studied is the average number

of infections that descend down the chains of infection begun by each of the individuals infected in an outbreak (we refer to this quantity as the “average number of descendant infections” per infectious individual, or ANDI). ANDI includes not only the number of people that an individual directly contacts and infects, but also the number of people that those go on to infect, and so on until the chain of infection dies out.

Quantification of ANDI has relevance to the anti-vaccine debate; if the individual probability of hospitalisation with a disease is  $p$ , the probability that at least one individual down an average chain of infection is hospitalised is  $1 - (1 - p)^{\text{ANDI}}$ .

Here we examine a Susceptible, Infected, Recovered (SIR) model, and obtain the analytic expression for ANDI. We compare the estimates of ANDI from this expression to those obtained using an Agent Base Monte Carlo formalism that keeps track of who infected whom. We find that ANDI is maximised for smaller  $R_0$ , and that ANDI in even relatively small populations can be literally dozens of individuals.

While the model examined in this initial analysis is simple, the analytic and computational formalisms we have developed can be expanded to a wide variety of models for disease spread.

---

**PAULINE VAN DEN DRIESSCHE**, University of Victoria

*Disease extinction versus persistence in discrete-time epidemic models*

Discrete-time infectious disease models are formulated in populations that are asymptotically constant, for example, governed by the Beverton-Holt equation. The basic reproduction number  $\mathcal{R}_0$  is calculated, and is shown to act as a threshold. If  $\mathcal{R}_0 < 1$ , then the disease-free equilibrium is proved to be globally asymptotically stable; whereas if  $\mathcal{R}_0 > 1$ , then the disease persists. Results are applied to specific discrete-time epidemic models that are formulated as SEIR infections (e.g., chickenpox) and anthrax in animals. [This is joint work with Abdul-Aziz Yakubu]

---

**JAMES WATMOUGH**, University of New Brunswick

*A mathematical analysis of an integrated approach to malaria control targeting vector-host interactions*

Many methods are available for the control of vector-borne diseases. Bed-nets can prevent encounters between vector and host; a vector’s search for suitable hosts can be manipulated using both attracting and repelling odorants; artificial feeders can be used to provide vectors with alternate sources for blood-meals, thus reducing contacts with hosts. We present an analysis of a mathematical model for malaria transmission designed to examine the effectiveness of an integrated disease-control approach where mosquito nets, protective odorants and mosquito feeders are used. Each intervention has a threshold coverage above which it will be effective for control when used alone. Not surprisingly, most interventions are more effective in combination. One exception is the combination of bed-nets with repellents, which can lead to increased prevalence at low coverage rates.

---

**PING YAN**, Public Health Agency of Canada

*Discussions on generation intervals and beyond*

The concept of generation intervals have been around in the literature for a long time (since the 1940s), and the terminology has been sometimes interchangeably called as serial intervals or transmission intervals, although their definitions are different both conceptually and quantitatively. In addition to the ambiguity in terminology, the matter is more complex because some of these intervals are based observations; some are not observable but they conceptually capture the transmission between infector-infectee pairs; whereas some are “intrinsic” (Champredon and Dushoff, 2015) within a single infected individual that directly link to important transmission parameters such as the reproduction number. This presentation is a discussion on these distinctions with a renewed perspective in the relationships between the transmission rate at the level of individuals and the intrinsic growth rate at the population level, and a discussion on the distribution of the (intrinsic) generation intervals and its relationship with the equilibrium conditions.