Parallel Session

Epidemiology IX

ADVANCES IN MULTISCALE MODELING OF INFECTIOUS DISEASES

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Keywords: Infectious Diseases, Multiscale Modeling, Within-host, Between-host.

Infectious diseases are persistent threats to humankind - killing annually 16 million people worldwide. The spread of pathogens between infectious and susceptible hosts remains a central vexation for science as it involves several complex and dynamic processes. The link between the infection dynamics within an infected host and the susceptible population-level transmission is widely acknowledged. However, several technical aspects of the interface of within- and between-host scales are still in their infancy.

In this talk, we present a formal mathematical derivation of the reproductive number between host (R_0^B) as an increasing function of the reproductive number within host (R_0^W). Numerical analyses reveal that a Michaelis-Menten form of virus-dependent transmission is more likely to recapitulate the behavior between the two scales than a form directly proportional to the pathogen [1].

On a computational level, a static network model that embodies age-specific human contact patterns in Europe and a within-host viral infection model can be coupled to forecast "what if" scenarios. Considering Ebola virus infection as an example [2], multiscale simulation results uncovered that in order to have a protective antibody level, vaccination would need to be delivered from one week to four months before the exposure to Ebola virus. Correspondingly, mass vaccination programs also reduced the reproductive number (R_0) to below one. Notably, compared to a non-intervention scenario, a low vaccination coverage of 33% could reduce the number of infected cases by 10- to 100-fold.

The use of multiscale modeling is a promising tool to provide early and data-driven evaluations of vaccination strategies — possibly also for newly emerging or re-emerging pathogens. More collaborative efforts are needed to attract the attention of experimentalists and clinicians in determining key within-host parameters to model disease transmission.

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References

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AN EXACT APPROACH TO CALIBRATING INFECTIOUS DISEASE MODELS TO SURVEILLANCE DATA

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Keyword: Model calibration.

When mathematical models of infectious diseases are used to inform health policy, an important first step is often to calibrate a model to disease surveillance data. While frequently overlooked, the calibration process is nontrivial at best and can be inefficient, poorly communicated and a major hurdle to the overall reproducibility of modeling results.

In this work [1], we describe a general approach to calibrating infectious disease models to surveillance data. The technique is able to match surveillance data to high accuracy in a very efficient manner as it is based on the Newton-Raphson method for solving nonlinear systems. To demonstrate its robustness, we use the calibration technique on multiple models for the interacting dynamics of HIV and HSV-2

References

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ECONOMICAL EPIDEMIC MODEL AS CONTROLLED SWITCHED SYSTEM

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Keywords: Impulsive control, Basic reproduction number.

In this contribution, we consider a SIR model controlled by impulsive vaccination and isolation control. Considering the problem of determining optimal impulsive controls to minimize the total outbreak size over the course of the epidemic and using necessary condition of optimality, our goal is to draw conclusions about the effect of the shortage of the drug treatment on the management of strategies of control policy. In other words, we are going to answer two questions: What if the treatment runs out during the epidemic? If this happens, could we still control the spread of the disease?

References

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MATHEMATICS OF A SEX-STRUCTURED MODEL FOR TRICHOMONIASIS TRANSMISSION DYNAMICS

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Keywords: Trichomoniasis, Equilibrium points, Stability, Bifurcation bifurcation.

A deterministic model for the transmission dynamics of Trichomoniasis disease in a population is designed and analysed. The model is shown to exhibit the phenomenon of backward bifurcation, where a stable disease-free equilibrium co-exists with a stable endemic equilibrium when the basic reproduction number $\mathcal{R}_0$ is less than one. It is further shown that the backward bifurcation dynamics is caused by the re-infection of individuals who recovered from the disease. In the absence of backward bifurcation, the global asymptotic stability of the disease-free equilibrium is shown whenever $\mathcal{R}_0 < 1$. Moreover, under special case of the model, the existence of globally asymptotically stable endemic equilibrium is confirmed when $\mathcal{R}_0 > 1$. Sensitivity analysis of the model, using the parameters relevant to the transmission dynamics of the Trichomoniasis disease, is given. Numerical experiments are given to support the theoretical analysis of the model.

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