



Editorial: Mathematical modeling of infectious disease dynamics

Constantinos I Siettos

To cite this article: Constantinos I Siettos (2016) Editorial: Mathematical modeling of infectious disease dynamics, *Virulence*, 7:2, 119-120, DOI: [10.1080/21505594.2016.1150402](https://doi.org/10.1080/21505594.2016.1150402)

To link to this article: <https://doi.org/10.1080/21505594.2016.1150402>



© 2016 Taylor and Francis Group, LLC



Published online: 23 Mar 2016.



Submit your article to this journal [↗](#)



Article views: 819



View related articles [↗](#)



View Crossmark data [↗](#)



Citing articles: 1 View citing articles [↗](#)

Editorial: Mathematical modeling of infectious disease dynamics

Constantinos I Siettos*

School of Applied Mathematics and Physical Sciences; National Technical University of Athens; Athens, Greece

Introduction to the Special Issue

Epidemics have left their own stamp on mankind's history. Civilizations have been wiped off the face of the earth through the ages. The list is long: biblical pharaonic plagues which hit Ancient Egypt in the middle of the Bronze Age around 1715 B.C.; the “λοιμός” in Athens from 430 to 425 B.C., which set the end of the Periclean golden era; the Black Death Bubonic Plague (1348–1353), which is estimated to have killed over 25 million people in Europe alone; and the pandemic influenza of 1918–1919 which swept through the globe, leaving a death-toll of around 40 million people.¹

In recent decades, emerging and re-emerging epidemics such as AIDS, measles (one of the leading causes of death among children), malaria, and tuberculosis have killed millions of people each year. Today, the UNAIDS reports around 37 million people living with HIV. WHO reported around 440,000 deaths from malaria in 2015; related deaths from measles were about 314 every day, while 1.5 million died from tuberculosis. Recently, the Ebola epidemic devastated populations in the countries of West Africa, leaving more than 11,300 dead.²

In this battle, mathematical models and systems theory enhance our arsenal in combating epidemics. Recent advances in different fields ranging from computational science and engineering to biology and epidemiology have advanced our knowledge, such as the molecular structure of a variety of viruses and the impact of network transmission properties on the

evolution of epidemics. State-of-the-art computational techniques allow the efficient extraction of important information from very large databases (VLDB) including publicly available biological, transportation, sociological, demographical, and epidemiological data, which in turn help us construct more realistic models that shed light into the mechanisms that govern the outbreaks.¹ Thus, public authorities take a closer look at the use of mathematical models to make decisions and to design efficient interventions.

Brief review of Contributions

This special issue of *Virulence* contains eight high-quality contributions from researchers worldwide that discuss a variety of topics ranging from agent-based modeling of infectious diseases to immunology, to hospital-acquired infections to social diseases such as obesity. All papers underwent a very careful peer review process and were revised to accommodate comments made by the reviewers. Special attention was given to the selection of the appropriate reviewers. The choice was based on strict academic criteria including expertise in the field and international reputation. In what follows, I briefly review the published contributions in alphabetical order with respect to the name of the first author.

Aquino and Nunes³ investigated influenza A phylogeny dynamics's rapid mutation of the pathogen in a host population with a weak immune response. Their model is a detailed stochastic individualistic one within

the allowing for reinfection by the same strain. Each viral strain was characterized by several epitopes and infected persons developed only one type of antibody. Protection from the infection was analogous to the number of matching antibodies for the antigenic sites of the strain. The results underpin the importance of developing multiscale models that can approximate the dynamics ranging from the response of the immune system to phenotypic heterogeneity (host-pathogen interactions) to the contact transmission network (host-host interactions) for understanding the epidemic evolution of fast-mutating viruses such as influenza A.

Demongot *et al.*⁴ developed a social network model based on preferential attachment to investigate the dynamics of obesity. Under this framework obesity is regarded as a contagious “social” disease where individuals tend to create social links and behave similarly to other individuals with analogous attributes and habits while they cut links with individuals with different habits. Thus, obesity propagates as a disease from individual to individual through the social network. Their model is based on demographic data considering age and sex distributions, mortality, fertility, composition of families, as well as socio-economic factors.

Doan *et al.*⁵ investigated the efficacy of infection control interventions against *Acinetobacter baumannii*, a common factor of hospital-related infections. In particular, the authors propose a compartmental model to evaluate “what if” scenarios of different interventions for reducing *A. baumannii* prevalence in intensive care units (ICUs). The model describes the

*Correspondence to Constantinos I Siettos; Email ksiet@mail.ntua.gr

Submitted: 01/31/2016; Accepted: 01/31/2016

<http://dx.doi.org/10.1080/21505594.2016.1150402>

probabilities of infection and colonization with or without antibiotic exposure from free-living bacteria in the environment. Sensitivity analysis to changes in the model parameters is also provided. Their study addresses important insights for designing effective infection control interventions, emphasis of the importance of effective antimicrobial stewardship programs to reduce the duration of antibiotic therapy, as well as environmental cleaning.

Kattis *et al.*⁶ proposes a systematic data mining-assisted framework to extract dominant collective statistics from detailed epidemic data. The paradigm is an individualistic Susceptible-Infected-Susceptible (SIS) model evolving on adaptively changing networks. The cornerstone of the proposed methodology is the equation-free multiscale modeling approach that can be exploited to extract “on demand” useful macroscopic information from detailed individualistic simulations/data. This allows a reduction of the high-dimensional phase-space of epidemic dynamics to a low-dimensional space that it is spanned by a few macroscopic observables. Bridging the equation-free approach with Diffusion Maps (DMAPS), a state-of-the-art learning manifold technique, allows for the determination of the macroscopic variables which shape the evolution of the emergent epidemic dynamics.

Kiskowski and Chowell⁷ developed a detailed agent-based model to approximate the dynamics of the Ebola virus epidemic that has ravaged West Africa populations in the past two years. Particular emphasis was given on the impact of the spatial constrained contact social structures in the disease transmission dynamics. The proposed model incorporates a network of households and communities of different sizes. Simulation results showed good agreement with the

epidemic growth observed in Guinea, Liberia, and Sierra Leone. Their analysis underscores the importance of the spatial contact structure in the spread and control of the epidemic.

Reppas *et al.*⁸ studied the interplay between the dynamics of vascularized tumor growth and the associated immune system response in favor of the design of effective immunomodulatory therapies. Their analysis is based on a mean-field model that approximates the dynamics of vascularized tumor growth as well as the corresponding effector cell recruitment dynamics. The authors performed a systematic analysis of a model behavior with the aid of bifurcation theory. By doing so, they determine the parameter regimes that allow an immune-mediated tumor control in terms of an external modification of effector cell dynamics.

Sardar *et al.*⁹ developed a two strain model to approximate the dynamics of dengue infection. Since 2002, dengue is an endemic disease in southeast Asia ravaging the population with two seasonal peaks per year. The authors exploit the model to estimate the reproduction numbers in two provinces of Sri Lanka during the period 2013–14. Seasonality was incorporated in terms of periodicity in the mosquito biting rate. Using univariate sensitivity analysis, the authors also assessed the sensitivity of the reproduction number with respect to various model parameters such as the transmission probability and the mosquito mortality rate.

Scott *et al.*¹⁰ provide a review on mathematical modeling approaches to hepatitis C virus (HCV) epidemic. The virus was first isolated in 1989 and since then HCV transmission dynamics has been the topic of many modeling studies. The review focuses on the transmission between people who inject drugs (PWID), detailing

assumptions, the way through which the virus is transmitted, as well as the limitations of the modeling approaches. The review ranges from mean field Susceptible-Infected-Recovered (SIR) models with treatment policies to individualistic networked models. Future challenges pinpointing the need for developing strain models are also given.

Concluding Remarks

Modern epidemiological research integrates advances from interdisciplinary fields ranging from molecular biology and epidemiology to sociology and applied mathematics, facilitating the design and implementation of better public health strategies to combat epidemics. However, it should be stressed that the usefulness of mathematical models should not be overestimated. Despite huge technological progress and concentrated wealth, breakdowns and cuttings in public health infrastructure continue to cause rapid impoverishment in developing countries, and remain the major reason of epidemic spread.

Acknowledgments

I gratefully acknowledge the authors for their generous contributions, the referees for taking the time and effort to review the papers in detail, the Editor-in-Chief of *Virulence* Professor Eleftherios Mylonakis for inviting me as a guest editor of this special issue, and also the editorial team of Taylor and Francis: Adam Weiss, Zachary Ayres, and David Penyak, for their help and patience throughout the process.

References

1. Siettos CI, Russo L. Mathematical modeling of infectious disease dynamics: a review. *Virulence* 2013; 4(4):295-306.
2. CDC - National Center for Health Statistics - Homepage. <http://www.cdc.gov/vhf/ebola/outbreaks/2014-west-africa/case-counts.html>
3. Aquino T, Nunes A. Host immunity and pathogen diversity: a computational study. *Virulence* 2016; 7(2):121-128.
4. Demongeot J, Hansen O, Taramasco C. Discrete dynamics of contagious social diseases: Example of obesity. *Virulence* 2016; 7(2):129-140.
5. Doan TN, Kong DCM, Marshall C, Kirkpatrick CMJ, McBryde ES. Modeling the impact of interventions against *Acinetobacter baumannii* transmission in intensive care units. *Virulence* 2016; 7(2):141-152.
6. Kattis AA, Holiday A, Stoica A-A, Kevrekidis IG. Modeling epidemics on adaptively evolving networks: a data-mining perspective. *Virulence* 2016; 7(2):153-162.
7. Kiskowski M, Chowell G. Modeling household and community transmission of Ebola virus disease: epidemic growth, spatial dynamics and insights for epidemic control. *Virulence* 2016; 7(2):163-173.
8. Reppas AI, Alfonso JCL, Hatzikirou H. In silico tumor control induced via alternating immunostimulating and immunosuppressive phases. *Virulence* 2016; 7(2):174-86.
9. Sardar T, Sasmal S, Chattopadhyay J. Estimating dengue type reproduction numbers for two provinces of Sri Lanka during the period 2013–14. *Virulence* 2016; 7(2):187-200.
10. Scott N, Hellard M, McBryde ES. Modeling hepatitis C virus transmission in developed settings: assumptions, limitations and future challenges. *Virulence* 2016; 7(2):201-208.