Parallel Session

Epidemiology VII

DIRECT TRANSMISSION MODELS FOR INDIRECTLY TRANSMITTED ENVIRONMENTAL PATHOGENS

Lee Benson

lee.benson@bioss.ac.uk

Department of Computing Science & Mathematics, University of Stirling. Biomathematics & Statistics Scotland (BioSS). Scotland's Rural College (SRUC)

Keywords: Aquaculture, Epidemiology, Bayesian inference, Markov chain Monte Carlo, Latent residuals.

Compartmental epidemiological models are used extensively in human and land-based animal disease systems, with the disease transmission process often characterised as resulting from a series of discrete contacts between individuals – so called 'direct transmission' models. Typically, such models assume the mass-action principle acts across the whole population or within subcomponents. In such models the force of infection on susceptible individuals is proportional to the absolute or relative size of the infectious sub-population. However, in some disease systems, especially those found in aquaculture, the 'environmental pathogen load' can play a significant role in infection spread, e.g. white spot syndrome virus in *penaeus monodon* (Asian tiger shrimp). In such scenarios proportionality between the force of infection and the number, or proportion, of infectious individuals cannot be assumed to hold, a priori.

However, in practice modelling environmental pathogen load alongside host population disease dynamics is problematic. This is because although disease data on aquaculture systems often records time of death and/or the onset of symptoms of the focal host species, it typically does not include extensive measurement of environmental pathogen load over time. Nevertheless, from these data Bayesian Markov chain Monte Carlo methods (MCMC) can be used to infer direct transmission model parameters plus host latent infection and recovery times and associated 'residuals'. We therefore investigate when direct transmission models are sufficient to capture disease dynamics governed by the environmental pathogen load.

Embedded within an MCMC algorithm, the method of latent residuals is investigated as a means of detecting departures from the direct transmission assumption, including the quantity of data required. Considering a range of scenarios, we identify a subclass of host-pathogen systems in which environmental pathogen population dynamics are sufficiently 'fast' relative to host population dynamics that we are able to model the system without needing to account for the pathogen load.

Acknowledgements: Prof Glenn Marion, BioSS. Dr Andy Hoyle, Computing Science & Mathematics, University of Stirling. Dr Darren Green, Aquaculture, University of Stirling. Dr Ross Davidson, SRUC & BioSS. Prof Mike Hutchings, SRUC.

Parallel Session

Epidemiology VII

COEXISTENCE IN A SEASONAL EPIDEMIC MODEL

Viggo Andreasen

viggo@ruc.dk

Department of Science, Roskilde Univ., DK-4000 Roskilde, Denmark

Joint work with Greg Dwyer (Department of Biology, Univ. of Chicago).

Keywords: Epidemic model, Coexistence, Multiple timescales, Invasion condition.

Pathogens of insects often infect only larvae, and in many species, there is only one larval period per year, so that there is only one pathogen epidemic per year. If incubation times differ between pathogen strains, two or more strains may co-exist in the same host population, in contrast to the situation in stable environments [1].

To understand this coexistence, we assume that the host population size is regulated independently of the disease, so that the population at the beginning of each epidemic is of size N. During the epidemic, susceptible hosts S are infected by either of two strains I and Y, according to an SIR-type epidemic model;

$$\begin{aligned} \dot{S} &= -\beta_I SI - \beta_Y SY \\ \dot{I} &= \beta_I SI - \nu_I I \\ \dot{Y} &= \beta_Y SY - \nu_Y Y, \end{aligned}$$

where we have omitted the size of the immune class R. We determine the outcome of the seasonal epidemic by the burn-out approximation, corresponding to the assumption that the epidemic burns out prior to the end of the larval season [2].

The initial number of hosts infected with strain I in the subsequent season $(n+1)$ is proportional to the total production of pathogen I during season n, where the proportionality constant W_I describes the probability that the virus survives the winter, such that

$$I^{n+1}(0) = W_I \int_{=}^{\infty} \nu_I I(t)\, dt$$

with a similar expression for $Y^{n+1}(0)$, while $S^{n+1}(0) = N - I^{n+1}(0) - Y^{n+1}$. For the case in which inter-generational survival is small, so that $W_I \ll 1$, we use multiple time scales and the theory for singular regular points to derive an approximate condition for when strain Y can establish if strain I is at endemic equilibrium. We then show that mutual invasion is possible for a wide range of parameter values. This result helps explain the high levels of polymorphism documented in some insect pathogens, and in pathogens in general [3], without the need to include fitness tradeoffs.

Acknowledgements: Our work was funded by National Institutes of Health grant R01GM96655.

References

[1] HJ. Bremermann, HR Thieme. (1989). *A competitive exclusion principle for pathogen virulence*. J. Math. Biol. 27, 179–190.

[2] G. Dwyer et al. (2000).*Pathogen-driven Outbreaks in forest defoliators revisited: building models from experimental data*. Am. Nat. 156, 105-120

[3] A. E. Fleming-Davies et al. (2015). Effects of host heterogeneity on pathogen diversity and evolution. Ecol. Lett. 18, 1252-1261.

Parallel Session

Epidemiology VII

VIRULENCE MANAGEMENT IN A MODEL WITH EXPLICIT WITHIN- AND BETWEEN-HOST DYNAMICS

Ruili Fan

ruili.fan@helsinki.fi

Department of Mathematics and Statistics, University of Helsinki, FIN-00014, Finland

Joint work with Stefan A.H. Geritz (University of Helsinki), Mats Gyllenberg (University of Helsinki).

Keywords: Virulence management, Within-host, Between-host.

We formulate a SIR model with explicit within-host and between-host dynamics. We use this model to study optimal virulence management for three types of low-tech interventions: (1) "soup-and-soap" (to reduce mortality by providing basic care), (2) quarantine (to reduce transmission by isolating infected individuals), or (3) both treatments at the same time. We consider six different object functions: (1) the incidence of the disease, (2) the disease induced death rate at population level, (3) the probability that a newborn susceptible eventually dies of disease, (4) the population density of infected, (5) the population density of susceptibles, and (6) the population fraction of susceptibles. We seek to separately minimize the first four object functions, and to maximize the last two, as functions of the fraction of the infected that are eligible for treatment and the per capita cost of a treatment. Given a fixed financial budget, there is a trade-off between the fraction of the infected population to be treated and the cost per treated individual. The results show that the optimal management strategies depend on the kind of intervention, on the particular choice of object function as well as on the budget level.

Parallel Session

Epidemiology VII

AN IN-HOST PERSPECTIVE ON DISEASE DYNAMICS

Maria Vittoria Barbarossa

barbarossa@uni-heidelberg.de

Heidelberg University, Institute of Applied Mathematics, Heidelberg, Germany

Keywords: Immuno-epidemiology, Physiological structure, Delay equations, Basic Reproduction Number.

After a disease outbreak, recovered individuals constitute a large immune population, however their immunity is waning in the long term and they may become susceptible again. At the same time, the host's immune system can be boosted by repeated exposure to the pathogen, which is linked to the density of infected individuals present in the population. This prolongs the length of the host's immunity. Such an interplay of within host and population level dynamics poses significant challenges in rigorous mathematical modeling of immuno-epidemiology. In this talk we present a new modeling approach for disease dynamics, monitoring the immune status of individuals and including both waning immunity and immune system boosting. A coupled system of ordinary and partial differential equations allows to investigate the temporal evolution of the distribution of immunities in a population, showing that different immune boosting mechanisms lead to very different stationary distributions of the immunity at the endemic steady state.

Acknowledgements:MVB is supported by the European Social Fund and by the Ministry of Science, Research and Arts Baden-Württemberg.

References

[1] MV Barbarossa, G Röst. (2015). *Immuno-epidemiology of a population structured by immune status: a mathematical study of waning immunity and immune system boosting*, J. Math. Biol., 71(6), pp. 1737–1770.

[2] MV Barbarossa, M Polner, G Röst. (2017). *Stability switches induced by immune system boosting in an SIRS model with discrete and distributed delays*, SIAM J. Appl. Math., 77(3), pp. 903–925.

[3] MV Barbarossa, M Polner, G Röst. (2018). *Temporal evolution of immunity distributions in a population with waning and boosting*, BiorXiv 253476.

Parallel Session

Epidemiology VII

CULLING CAUSES COMPENSATORY POPULATION GROWTH DUE TO RELEASE FROM DISEASE-INDUCED MORTALITY

Eleanor Tanner

ent1@hw.ac.uk

Department of Mathematics and the Maxwell Institute for Mathematical Sciences, Heriot-Watt University, Edinburgh, EH14 4AS, UK

Joint work with Andy White (Heriot-Watt University), Peter Lurz, Christian Gortázar (SaBio-IREC), Iratxe Díez-Delgado (SaBio-IREC), Mike Boots (UC Berkeley).

Keywords: Culling, Compensatory growth, Wild boar, Disease management.

It is well known that culling and harvesting can reduce population abundance and that some of the mortality induced by culling can be offset through compensatory growth. However, culling is also a widespread strategy in wildlife disease management and the potential impacts of culling on infection dynamics are poorly understood. We develop a general mathematical model of infectious disease to investigate the magnitude of compensatory growth in systems that harbour virulent parasites. We use classical compartmental SI, SIR and SIRS mathematical models and consider density dependent, frequency dependent and free-living transmission to identify the interplay between culling and disease induced mortality. We show that culling can lead to a reduction in the force of infection and therefore compensatory growth due to a population level release from disease-induced mortality. In the absence of long-lasting immunity this phenomenon accounts for the majority of the compensatory growth response in systems that harbour virulent parasites. These effects are illustrated by a case-study of harvesting wild boar populations in Central Spain suffering endemic tuberculosis.