Demographic and Environmental Variability on Population and Disease Extinction

Many biotic and abiotic factors impact the survival and productivity of plant and animal populations. Population densities vary over time as a result of changes in rainfall, temperature, food availability, presence of pathogens, pests, competitors or predators [3, 5, 6, 7]. In this project, we formulate predator-prey, competition, and epidemic models to study the impact of both demographic and environmental variability on population or disease extinction. Ordinary differential equations (ODE) are used as a framework to construct stochastic models, continuous-time Markov chains (CTMC) that account for the variability. In the stochastic models, the random variables governing the populations or epidemics are nonnegative and discrete-valued and changes in populations depend on probabilities. For example, the dynamics of an ODE model and sample paths of a CTMC model of an epidemic model and predator-prey model are graphed in Fig. 1. In the predator-prey model, the variability in the sample paths of the populations may result in prey or predator extinction but the ODE solution exhibits periodic oscillations of prey and predator with no extinction. In the susceptible-infectious-recovered (SIR) model, the sample paths either result in a major outbreak from spread of the infection to many others or a few cases with rapid disease extinction but the ODE solution exhibits a major outbreak with many infectious cases. Whether population or disease extinction occur in the stochastic models can be addressed through application of generating functions and branching processes [1, 2]. In the case of the stochastic SIR model, the answer is found by computing the ratio of the transmission rate, β, to the recovery rate, γ, the well-known basic reproduction number, $\mathcal{R}_0 = \beta/\gamma$, defined as the number of secondary infections caused by one infectious individual introduced into an entirely susceptible population. For the ODE SIR epidemic model, there are many new infectious cases if $\mathcal{R}_0 > 1$. But this is not necessarily the case for the stochastic SIR epidemic model. In particular, an estimate for the probability of disease extinction in the stochastic epidemic model is

$$\mathbb{P}_{ext} = \begin{cases} \left(\dfrac{1}{\mathcal{R}_0}\right)^i, & \text{if } \mathcal{R}_0 > 1 \\ 1, & \text{if } \mathcal{R}_0 < 1 \end{cases}$$

where i is the number of infectious individuals at the beginning of the epidemic. The probability of a major outbreak is given by $\mathbb{P}_{outbreak} = 1 - \mathbb{P}_{ext}$ [8]. In more complex disease or population settings with predation or competition, the theory of branching process can be used to obtain estimates for extinction. These mathematical methods can also be applied to stochastic models with seasonal variability to investigate the effects of seasonal changes such as temperature, rainfall, or food availability on survival or extinction of populations [4]. **Timeline:** Week 1: Background information on biological models, mathematical and computational methods with problem solving, computer training in Maple/MatLab and discussions. Weeks 2-3: Identify epidemic or predator-prey or competition from the literature, where seasonality plays an important role in spread, contacts, transmission, births, or deaths. Identify specific biological questions to address on population or disease extinction and begin formulating models. Weeks 4-6: Analysis, development of computer code, and simulation of new stochastic epidemic, predator-prey or competition models. Weeks 7-8: Verify analytical and numerical results, summarize findings, interpret results, and write paper.

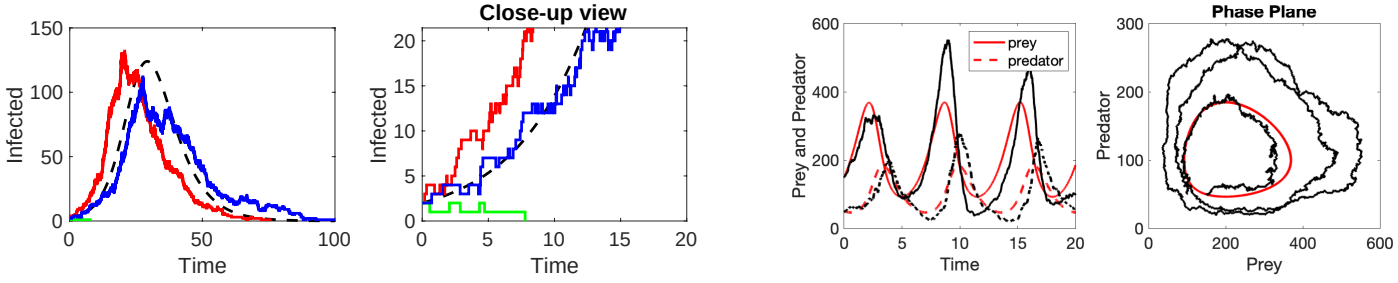

Figure 1: Two graphs on left: Infectious individuals at the initiation of an epidemic (3 CTMC sample paths [red,blue,green] with ODE solution [black dashed]). Two graphs on right: Predator-prey model (CTMC sample path [black] and ODE solution [red]).

References

[1] Linda JS Allen. A primer on stochastic epidemic models: Formulation, numerical simulation, and analysis. *Infectious Disease Modelling*, 2(2):128–142, 2017.

[2] Linda JS Allen, Sophia R Jang, and Lih-Ing Roeger. Predicting population extinction or disease outbreaks with stochastic models. *Letters in Biomathematics*, 4(1):1–22, 2017.

[3] Sonia Altizer, Andrew Dobson, Parviez Hosseini, Peter Hudson, Mercedes Pascual, and Pejman Rohani. Seasonality and the dynamics of infectious diseases. *Ecology Letters*, 9(4):467–484, 2006.

[4] Nicolas Bacaër and El Hadi Ait Dads. On the probability of extinction in a periodic environment. *Journal of Mathematical Biology*, 68(3):533–548, 2014.

[5] Robert D Bradley, J Delton Hanson, Brian R Amman, B Dnate'Baxter, Darin S Carroll, Nevin D Durish, Michelle L Haynie, Mariko Kageyama, Lisa K Longhofer, Francisca M Mendez-Harclerode, et al. Rapid recovery of rodent populations following severe drought. *The Southwestern Naturalist*, 51(1):87–93, 2006.

[6] Anthony Caravaggi, Maria Gatta, Marie-Claire Vallely, Kayleigh Hogg, Marianne Freeman, Erfan Fadaei, Jaimie TA Dick, W Ian Montgomery, Neil Reid, and David G Tosh. Seasonal and predator-prey effects on circadian activity of free-ranging mammals revealed by camera traps. *PeerJ*, 6:e5827, 2018.

[7] Stefan Halle and Nils Christian Stenseth. *Activity Patterns in Small Mammals: An Ecological Approach*, volume 141. Springer Science & Business Media, 2000.

[8] Peter Whittle. The outcome of a stochastic epidemic - a note on Bailey's paper. *Biometrika*, 42(1-2):116–122, 1955.