Wine, Ebola and Terrorism

MIT Lincoln Laboratory

March 16 2015

Many populations exhibit an early exponential rise in number, reach a peak and then fall back to low levels. This is true of the population of yeast spores in a bottle of fermenting wine, the number of people infected with the Ebola virus during an outbreak, and the number of recruits to a terrorist organization. These populations are often modeled with Lotka-Volterra [5] or Verhulst equations [1]

$$\frac{dP}{dt} = rP\left(1 - \frac{P}{K}\right)$$

where P is the population size, r is the rate of growth and K is called the carrying capacity. Diseases are often modeled using the SIR model (Susceptible, Infected, Removed) [4],

$$\frac{dS}{dt} = -\beta SI$$
$$\frac{dI}{dt} = \beta SI - \gamma I$$
$$\frac{dR}{dt} = \gamma I$$

where β is the rate that infected individuals cause infections in the susceptible population and γ is rate of removal. An individual is removed from the SIR model population by either recovering and no longer being susceptible, or dying. The ratio $r_0 = \frac{\beta}{\gamma}$ is the basic reproductive rate and describes the number of susceptible individuals infected by a person who already has the disease. Diseases do not rapidly evolve so r_0 remains relatively constant, yet from one outbreak to the next the course of the disease may be very different. The first case of Ebola occured in South Sudan in 1976 and killed 151 of the 284 people infected. During the current outbreak in West Africa (as of 15 February 2015) 23539 cases had been reported resulting in 9541 deaths in the three countries of Guinea, Liberia and Sierra Leone. If r_0 remains constant, what might account for the vastly different outcomes of the progression of the disease between 1976 and the present? One possible explanation is greatly improved communication through cell phones and the internet which allowed for better information to be quickly transmitted, but also permitted competing false information [7, 6]. The rumors that quickly spread contradicted best medical practice and may have led

to increased exposure in the population. Changes in travel patterns may also have contributed to the rapid spread of the disease [2].

For this project, the focus will be on discovering the underlying causes of the spread of Ebola beyond the simple SIR model. We will expand the basic model with plausible variants and then validate the improved model with the best available data. Several organizations have developed powerful tools to help build models, e.g. Insight Maker (https://insightmaker.com/) and Vensim (http://vensim.com/) [3]. These modeling tools are often open source or freely downloadable, and do not require experience in programming as the models are graphical in nature. Participants should have a good mathematics / statistics background and the ability to think creatively about how to expand the model to include secondary factors that may influence the outcome, but do not need any expertise in a programming language.

References

- Peter A. Abrams, The evolution of predator-prey interactions: Theory and evidence, Annual Review of Ecology and Systematics 31 (2000), 79–105.
- [2] Marta C. Gonzalez Ruben Juanes Christos Nicolaides, Luis Cueto-Felgueroso, A metric of influential spreading during contagion dynamics through the air transportation network, PLoS ONE 7 (2012), 1-10.
- [3] R. G. Dudley and C. S. Soderquist., A simple example of how system dynamics modeling can clarify, and improve discussion and modification, of model structure, Presentation at the Annual Meeting of the American Fishery Society, 1999.
- [4] Leah Edelstein-Keshet, Mathematical models in biology, SIAM, 1988.
- [5] Alfred J. Lotka, Analytical note on certain rhythmic relations in organic systems, Proceedings of the National Academy of Sciences of the United States of America. 1920;6(7):410-415. 6 (1920), 410-415.
- [6] Dennis Mollison, Dependence of epidemic and population velocities on basic parameters, Math Biosciences 107 (1991), 255-287.
- James M. Wilson Shannon M. Fast, Marta C. Gonzalez and Natasha Markuzon, Modelling the propagation of social response during a disease outbreak, J. R. Soc. Interface 12: 20141105 (2014).