Research Statement

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A little learning is a dangerous thing;/ Drink deep, or taste not the Pierian spring:/ There shallow draughts intoxicate the brain,/ And drinking largely sobers us again.

– Alexander Pope

General Topics: Mathematical Epidemiology. Delay Differential Equations (DDEs). Dynamical Systems. Numerical analysis. Stochastic Modeling.
MSC2010 Classifications: 34C60, 34K, 37M, 92D25, 92D30

Motivation & Background

My research falls squarely within the field of mathematical epidemiology – focused on modeling infectious disease at the population level where naturally-occurring delays are present in the system.

At its heart, the field of epidemiology is concerned with disease management; it seeks paths to mitigate the effect of a disease with the hope of eventually eradicating it. In order to do this, medical professionals have historically turned to observation. They would observe the cause of a disease, identify its mode of transmission, and then determine possible control measures. While such information is essential to a full understanding of illness, it also suffers from an inherent flaw; the observations must occur alongside a disease outbreak. In particular, this prevents epidemiologists from operating under the normal scientific method because it makes model validation through experimentation impractical and unethical. Consequentially, epidemiology has embraced mathematics to build accurate models of real life scenarios.

So-called *compartmental models* are the primary tool used in mathematical epidemiology. In such models, the population is partitioned into compartments according to the disease dynamics. Differential equations are then used to describe how the compartments interact – giving rise to a dynamical system. The best known compartmental model was derived by Kermack and McKendrick in 1927, where the entire population is divided into a susceptible class (S), an infectious class (I), and a removed class (R) [7]. Its framework can be found in Figure 1. This simple concept has proven to be very powerful because such a framework is

highly customizable; it can be adapted to fit almost any given disease and population. For a good standard reference, see [1].
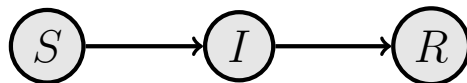

Figure 1: *General framework for the Kermack-McKendrick SIR model.*

In my work, I have a strong commitment to generating well-articulated, well-formulated, and well-motivated models. I also have a particular interest in linking qualitative system behavior to analytic and numerical results. Generally, all of my projects are driven by the following questions:

- Under what conditions will the population experience an epidemic of a given disease?
- Under what conditions will the disease become endemic in the population?
- What are the effects of various control measures to eliminate or eradicate the disease?

In the literature, the primary tool used to tackle these questions is known as the *basic reproduction number* ($\mathscr{R}_0$, also known as the basic reproduction ratio or rate) of a system. Epidemiologically speaking, this is the number of secondary cases produced by a single index case introduced into a wholly susceptible population. Mathematically, this corresponds to a threshold for the stability of the disease-free equilibrium. For ODE models, a method to determine this value using the next generation matrix was put forth by Diekmann, Heesterbeek, Metz in 1990 – which was then generalized for heterogeneous populations by van den Driessche and Watmough [3, 4, 14].

Dissertation Research

My dissertation was largely focused on two projects. The first was concerned with the effect of delayed dispersal in a small metapopulation which has been exposed to an infectious disease exhibiting susceptible-infectious-susceptible dynamics. The second adopts the same disease structure, but considers it within a two dimensional domain with an embedded metapopulation.

The Effect of Delayed Dispersal on Disease Dynamics in a Small Metapopulation

In general, my research follows along a similar path; however, it is largely centered around situations where there is a naturally occurring delay. In order to model such delays, we can turn to a specific type of functional differential equations known as *delay differential equations* (DDEs, also known as differential-difference equations). While similar in appearance to ordinary differential equations (ODEs), DDEs have an infinite-dimensional state space and computing their characteristic equation results in a nonlinear eigenvalue problem with an

infinite number of roots. For general references on DDEs, see [2, 5, 6]. For a reference on the use of DDEs in mathematical biology, see [11].

By in large part, the body of work at the intersection of DDEs and epidemiology is focused on analyzing the effect of delays in the disease dynamics or the life cycle of the host population; the effect of delays in the physical population structure is less scrutinized. In this project, I am analyzing a model of delayed dispersal within a metapopulation.

Metapopulations are formed by multiple groups of a single species that are separated (by geographic or other means) but are somehow capable of interacting. Physical movement between the subpopulations is known as dispersal. Leaving a region is called emigration; whereas movement into a region is called immigration.

In standard metapopulation models, dispersal is assumed to be instantaneous; however that is not physically feasible. Instead individuals leave a region and then arrive in another region after some travel delay. Our goal is to investigate how such a delay may affect the dynamics of a disease that exhibits SIS behavior. That is, a disease where individuals are susceptible to a disease or infectious with that disease. To simplify the investigation, we may assume the disease is non-fatal and that the effects of natural demographics (birth and death) is negligible. Figure 2 summarizes the general framework of the model.
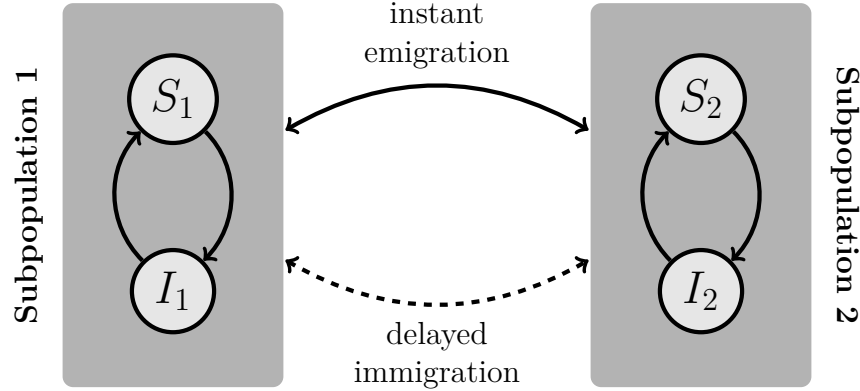
Figure 2: *General framework for a metapopulation model with two subpopulations and delayed dispersal.*

The value of the basic reproduction number is significantly more complicated to determine for systems of DDEs; however, Thieme in 2009 was able to extend this theory to models with an infinite-dimensional state space [13]. Circumventing this, my dissertation showed how we can tie the stability of the disease-free equilibrium in the delayed model to that of the non-delayed model.

The Effect of Dispersal in a Continuous Region on Disease Dynamics

The delays in the DDE model above are considered to be a result of time it takes to travel

between the two subregions in the metapopulation. As such, we might wish to directly model this movement. In particular, I considered an SIS infectious disease where the metapopulation is "embedded" in a one-dimensional continuous domain using an idea from [10] and then extended this to a two-dimensional continuous domain similar to that of [8]. For both cases, I first explore the underlying population model; and then move on to simulate what happens when the disease dynamics are added to this.

Directions for the Future

I strongly believe in biologically-driven mathematics. Given a particular biological phenomenon, researchers should challenge themselves to find the right mathematical tool for the job at hand. In my current and past work, I have stumbled upon a number of problems that I would like to explore further – some of which have already been at least partially studied by others, and all of which could be explored with undergraduate students.

Possible directions include:

- **Delayed Self-Quarentine in Infectious Disease:** Researching how delayed reaction can affect the disease dynamics in models that account for self-quarantine.
- **Cellular Automata of Disease Spread:** The compartmental framework using differential equations is very useful in designing infectious disease models; however, it inherently assumes the population is continuous and makes incorporating geographic information difficult. Cellular automata off a possible fix to these issues. How do they compare to the standard modeling methods?
- **Plant Epidemiology:** The assumption of a well-mixed population made in most compartmental models does not hold true with plants because they are immobile. Moreover, plant disease is often spread by airborne pathogens; and it is highly effected by weather and climate. What are the ramifications of these issues when modeling plant disease?

Student Involvement

While these projects stem from my own experiences, I am also devoted to helping my students explore their own interests. In my education, I was lucky to find faculty members who were willing to work on ideas that I found enjoyable. Their flexibility allowed me to pursue topics that I wouldn't have normally been able to go after at the small regional universities I attended. With that in mind, I am committed to serve a similar role for my future students.

Finally, it is my desire to establish a small mathematical biology research group. Since I recognize my own training has been largely from a mathematics perspective, I hope to do this with a faculty member from the biology department. Together, we could recruit interested mathematics and biology students and involve them in holistic and collaborative research projects. Such a group would provide a unique environment to foster students along their chosen academic and career paths and better prepare them for their own futures.

References

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