NDIX

1 Numerical Assumptions for Lecture 3 Figures

	Figure Number[1]											
r	3.2	3.3	3.4	3.5	3.10	3.11	3.12	3.13	3.14	3.15	3.16	3.17
	0.003		-0.003	-0.003								
	0.0085											
	0.0051											
	0.0068											
	0.002		-0.002	-0.002								
	0.0034											
	0.0051											
	0.0085											
	0.001		-0.001	-0.001								
	1.0				1.7	1.7	1.2	1.2	1.2	1.7	1.7	1.7
	0.0	1.0		1.0		0.01		0.01	0.10	0.02	0.025	
	0.0											10.0
	10.0											
	5.0											
	3.0											

c indicates the same value as in column 1 (figure 3.2).

LECTURE 4
Revolutions, Epidemics, and Ecosystems: Some Dynamical Analogies

From:

"Nonlinear Dynamics Mathematical Biology and Social Science"
By Joshua Epstein
Santa Fe Institute
Addison Wesley Publishing Company, 1997

The preceding lectures have concerned interstate arms racing (mutualism) and war (competition). Let us now turn attention to intrastate processes. This lecture concerns revolutions. The next concerns the spread of drugs. Clinging to our Volterra-like "grand unified theory," the processes of interest in these lectures is the threshold transmission of some "signal" through a population, epidemic-like processes, in short. Epidemics proper are fascinating—and obviously very important—things. You would certainly enjoy William McNeill's wonderful book, *Plagues and Peoples*, which concerns the role of infectious diseases in human history.[66] Since these lectures proceed from the analogy to epidemics, perhaps an introductory word or two on dynamical analogies *per se* is in order.

ANALOGIES

Any two processes whose mathematical descriptions have the same functional form, and whose state variables and parameters can be put in one-to-one correspondence,

[66] McNeill (1976).

are said to be *dynamical analogies*. It is a startling fact that a huge variety of seemingly unrelated processes are analogous in this sense. For example, the same equation that describes a damped harmonic oscillator, such as a pendulum with friction, also describes an oscillating electric circuit:

> "all that is required is to relabel the state variables and parameters involved. Thus, the state variable representing the displacement of the mechanical system becomes the electrical charge of the electrical system; the velocity becomes the current; the mass of the particle becomes the inductance, mechanical force becomes EMF, etc. With similar reinterpretations, the same dynamical equation can be regarded as describing rotational systems, acoustic systems, hydraulic systems, and so on" (Rosen, 1970, p. 54).[67]

Another example is the analogy between electrostatic attraction under Coulomb's Law and gravitational attraction under Newton's Law. The magnitude of each force is proportional to the product of the two charges/masses, inversely proportional to the square of the distance separating them, and directed along the line joining them. As another instance, Kelvin's circulation theorem in fluid mechanics is identical in its mathematical form to Faraday's Law in electrodynamics. Both relate, via Stokes' Theorem, the flux of a vector field to the circulation (or current) in a boundary such as a conducting loop.[68] Countless further examples could be provided. The physical diversity of diffusive processes satisfying the "heat" equation, or oscillatory processes satisfying the "wave" equation, is virtually boundless.

But dynamical analogies are more than beautiful testaments to the unifying power of mathematics: they are *useful*. In particular, "Analogies are useful for analysis in unexplored fields. By means of analogies an unfamiliar system may be compared with one that is better known. The relations and actions are more easily visualized, the mathematics more readily applied and the analytical solutions more readily obtained in the familiar system."[69]

Analogy in this sense has played a powerful role in the development of science, engineering, and also social science, a notable example of the latter being Samuelson's application to economics of classical maximum principles of physics. In one colorful discussion, for instance, he argues that "if you look at the monopolistic firm as an example of a maximum system, you can connect up its structural relations with those that prevail for an entropy-maximizing thermodynamic system. Pressure and volume, and for that matter absolute temperature and entropy, have to each other the same conjugate or dualist relation that the wage rate has to labor or the land rent has to acres of land." Samuelson provides an elegant diagram that, in

[67] EMF is electromotive force.

[68] See, for example, Marsden and Tromba (1976, p. 338).

[69] Olson (1958, p. iv).

his words, does "double duty, depicting the economic relationships as well as the thermodynamic ones."[70]

Murray Gell-Mann has written on the application of nonlinear dynamics to various systems, including social systems. In his words,

> "Many of these applications are highly speculative. Furthermore, much of the theoretical work is still at the level of 'mathematical metaphor.' But, I think this situation should cause us to respond with enthusiasm to the challenge of trying to turn these metaphorical connections into real scientific explanations" (Gell-Mann, 1988, p. 4).

It is in this highly speculative, metaphorical spirit that I proceed in the next two lectures. This essay examines the analogy between epidemics (for which a well-developed mathematical theory exists) and processes of explosive social change, such as revolutions (for which no comparable body of mathematical theory exists). Are revolutions "like" epidemics? More precisely, is it useful to think of these processes as analogous? Connections to predator-prey systems are also explored and the spatio-temporal generalizations of these revolution/epidemic/predator-prey models—reaction-diffusion equations—are examined. The realm of reaction-diffusion equations is a natural one to explore. Such equations are central to the mathematical theory of pattern formation, and it is the evolution, propagation, and stability of social patterns that is, ultimately, our concern. We begin with the simple analogy between revolutions and infectious diseases.[71]

REVOLUTIONS AS EPIDEMICS

The particular aims of revolutionary action, of course, vary widely from case to case. In one instance, the revolutionaries' goal may be the overthrow of monarchy; in another, it may be the installation of theocracy; in yet a third, it may be the establishment of democracy. Given this enormous variation in objectives, the thought that there might be an underlying structure common to all revolutions is an intriguing one. By a "common structure," I of course mean a mathematical model whose dynamics—at least at some crude level—are mimicked by revolutionary processes in general, regardless of their political "substance," as it were—regardless, that is,

[70] Samuelson (1972, pp. 8–9).

[71] While the connections between revolutions, epidemics, and ecological systems presented here have not, to my knowledge, been presented elsewhere, the thought that the spread of ideas might be analogous to the spread of disease has been explored. A small literature sprang up in the 1960's, following the publication in 1957 of the seminal work, Bailey (1957). For a good overview with references, see Dietz (1967). See also Rappaport (1974, pp. 47–59).

of what the revolution "is about." In considering this notion, I take, as one tantalizing point of departure, the mathematical theory of epidemics; these processes exhibit common dynamical structures despite obvious differences among communicable diseases. Measles, mumps, and smallpox are clearly different diseases; yet their *propagational dynamics* may be indistinguishable from a mathematical standpoint.[72] Although the points of correspondence between epidemics and revolutions will be quickly evident, it will prove useful to delay specific analogizing until a simple epidemic model is presented.

A BASIC EPIDEMIC MODEL

The epidemiologist's problem, as Paul Waltman puts it, "is to describe the spread of an infection within a population. As a canonical example one thinks of a small group of individuals who have a communicable infection being inserted into a large population of individuals capable of 'catching' the disease. Then an attempt is made to describe the spread of the infection in the larger group."[73] In the simple model first developed by Kermack and McKendrick,[74] the population is assumed to be constant and divided into three disjoint classes:

$S(t)$: the susceptible class comprised of individuals who, though not infective, are capable of becoming infective;
$I(t)$: the infective class, comprised of individuals capable of transmitting the disease to others; and
$R(t)$: the removed class, consisting of those who have had the disease and are dead, or who have recovered and are permanently immune, or are isolated until recovery and permanent immunity occur.

The following rules are assumed to govern the spread of the disease:

(i) The population is constant over the time interval of interest. Births, deaths from causes other than the disease in question, immigration, and emigration are all ignored.
(ii) The rate of change of the susceptible class is proportional to the product of the number of susceptibles $S(t)$ and the number of infectives $I(t)$.
(iii) Individuals are removed from the infectious class at a rate proportional to $I(t)$.

Rule (i) is a straightforward simplifying assumption whose relaxation is discussed below. Rule (ii) represents the assumption that the transfer of individuals from the

[72] See Hethcote (1976, p. 336). See also Hethcote (1989, pp. 119–44).
[73] Waltman (1974, p. 1).
[74] Kermack and McKendrick (1927). See Murray (1989, pp. 611–18).

susceptible class into the infectious pool proceeds at a rate proportional to the number of contacts between infectives and susceptibles. That the contact rate should be proportional to the product of the class sizes I and S implies uniform mixing of the two groups and instantaneous contraction of the disease upon exposure (latency and incubation periods are both zero). In effect, the law of mass action is assumed to apply. As Waltman notes, "This is reasonable if the population consists of students in a school whose changing classes, attending athletic events, etc. mix the population." Importantly for our purposes, he continues, "It would not be true in an environment where socio-economic factors have a major influence on contacts."[75] Finally, rule (iii) implies that all infectives have the same probability of removal (recovery, death, or isolation). The model does not account for the length of time an individual has been infective.

Accepting these definitions and rules, and treating the overall population as a continuum, the flow of individuals from the susceptible to the infective to the removed class is described by the following system of nonlinear differential equations:[76]

$$\begin{aligned} \frac{dS}{dt} &= -rSI\,, \\ \frac{dI}{dt} &= rSI - \gamma I\,, \\ \frac{dR}{dt} &= \gamma I\,, \end{aligned} \tag{4.1}$$

with initial conditions $S(0) = S_0 > 0, I(0) = I_0 > 0$ and $R(0) = 0$.

The constants r and γ are called the infection rate and the removal rate, and $\rho = \gamma/r$ is termed the relative removal rate.

THE THRESHOLD CONDITION

Now, under what conditions will an epidemic occur in this model? To say that an epidemic occurs is to say that the infectious class grows or, equivalently, that $dI/dt > 0$, which from (4.1) implies that $rSI - \gamma I > 0$ or, simply, that

$$S > \frac{\gamma}{r} = \rho\,. \tag{4.2}$$

[75] Waltman (1974, p. 2).
[76] Because the flow is from susceptible (S) to infective (I) to removed (R), this is termed an SIR model. If the infectious phase is followed, not by removal (e.g., immunity), but by reentry into the susceptible pool, an SIS model would be called for. "In general, SIR models are appropriate for viral agent diseases such as measles, mumps, and smallpox, while SIS models are appropriate for some bacterial agent diseases such as meningitis, plague, and venereal diseases, and for protozoan agent diseases such as malaria and sleeping sickness." Hethcote (1976, p. 336). See also Hethcote (1989). The cornerstone of the mathematical epidemiology literature remains Bailey (1957). See also Bailey (1975). A comprehensive contemporary text is Anderson and May (1991).

This is a basic result.[77] For an epidemic to occur, the number of susceptibles must exceed the threshold level ρ—the relative removal rate defined above.

POLITICAL INTERPRETATION

The basic analogy to revolutionary dynamics is direct. The infection, or disease, is of course the revolutionary idea. The infectives $I(t)$ are individuals who are actively engaged in articulating the revolutionary vision and winning over ("infecting") the susceptible class $S(t)$, comprised of those who are receptive to the revolutionary idea, but who are not infective (not actively engaged in transmitting the disease to others). Removal is most naturally interpreted as the political imprisonment of infectives—$R(t)$ is the "Gulag" population, the set of unfortunate revolutionaries who have been captured and isolated from the susceptible population.[78]

Many familiar tactics of totalitarian rule can be seen as measures to minimize r (the effective contact rate between infectives and susceptibles) or maximize γ (the rate of political removal). Press censorship and the systematic inculcation of counterrevolutionary beliefs reduce r, while increases in the rate of domestic spying (to identify infectives) and of imprisonment without trial increase γ.

Symmetrically, familiar revolutionary tactics—such as the publication of underground literature, or "samizdat"—seek to increase r. Similarly, Mao's directive that revolutionaries must "swim like fish in the sea," making themselves indistinguishable (to authorities) from the surrounding susceptible population, is intended to reduce γ.

GORBACHEV, DeTOQUEVILLE, AND SENSITIVITY TO INITIAL CONDITIONS

Interpreting relation (4.2) somewhat differently, if the number of susceptibles, S_0, is in fact quite close to ρ, then even modest reductions (voluntary or not) in central authority can push society over the epidemic threshold, producing an explosive overthrow of the existing social order. To take the example of Gorbachev, the policy of Glasnost obviously produced a sharp increase in r, while the relaxation of political repression (e.g., the weakening of the KGB, the release of prominent political prisoners, the dismantling of Stalin's Gulag system) constituted a reduction in γ. Combined, these measures evidently depressed ρ to a level below S_0, and the "revolutions of 1989" unfolded. Perhaps DeToqueville intuited relation (4.2), describing

[77] Obviously, the system (4.1) has a great many further mathematical properties of interest. For a discussion, see Braun (1983, pp. 456–73).

[78] In this discussion, we ignore executions.

this sensitivity to initial conditions, when he remarked that "liberalization is the most difficult of political arts."

TRAVELING WAVES

In the discussion thus far, the *spatial* dimension has only been implicit. In fact, epidemics spread across geographical areas over time. And one generally thinks of revolutions spreading as well. Specifically, we often invoke the terminology of *waves*. Recently, we saw "a wave of democratic revolutions" sweep across Eastern Europe. Perhaps this sort of language seems natural for a reason: if one generalizes model (4.1) to explicitly include the *spatial diffusion* of infectives, traveling waves do indeed emerge. And this process, of course, has a political interpretation.

The one-dimensional spatio-temporal generalization of (4.1) is:

$$\begin{aligned} \frac{\partial S}{\partial t} &= -rIS\,, \\ \frac{\partial I}{\partial t} &= (rIS - \gamma I) + D\frac{\partial^2 I}{\partial x^2}\,. \end{aligned} \tag{4.3}$$

An infective spatial diffusion term, $D\partial^2 I/\partial x^2$, has been introduced into the second equation, which bears some resemblance to the classical heat equation, $I_t = DI_{xx}$, where D is the thermal—or, in this case, the political—"diffusivity" of the medium. The presence of the parenthesized term makes the equation a so-called reaction-diffusion relation.

Now, as set forth in lecture 6, one posits traveling wave solutions to (4.3) of the form

$$S(x,t) = S(z), \quad I(x,t) = I(z), \quad z = x - ct\,, \tag{4.4}$$

where c is the wave speed. The boundary conditions $S(\infty) = 1, S(-\infty) = 0, I(\infty) = I(-\infty) = 0$ must also be met. Bypassing mathematical specifics that are well presented elsewhere,[79] the basic conclusions are, first, that no epidemic wave propagates if $S_0 < \gamma/r$. This, of course, is the basic threshold condition from model (4.1). What is new, however, is that if that threshold level of susceptibility is exceeded, an epidemic/revolutionary *wavefront* propagates. And its speed of propagation, c, is given by

$$c = 2[D(rS_0 - \gamma)]^{1/2}\,. \tag{4.5}$$

Basic counterrevolutionary tactics aim not only to minimize r (the rate at which contact produces a transmission) and maximize γ (the removal rate), but to minimize D as well. Physical curfews, restrictions on free assembly, internal

[79] See Murray (1989, pp. 661–63), Britton (1986, pp. 61–71), and the discussion in lecture 6 of this volume.

LECTURE 5
A Theoretical Perspective on The Spread of Drugs

INTRODUCTION

This lecture explores another social process of considerable interest, the spread of drugs, and is divided into three parts. In Part I, a simple dynamic model of a drug epidemic in an idealized community is built up from basic assumptions concerning the interaction of subpopulations—pushers, police, and not-yet-addicted residents of the community.[99] The model combines elements of the epidemic, ecosystem, combat, and arms race models discussed above. Equilibria of the resulting dynamical system are located and classified using tools of linearized stability analysis. Trajectories are plotted for a set of initial conditions. In Part II, a spatial—reaction-diffusion—variant is presented. Then in Part III, supply, demand, and price considerations are introduced; essential, and perhaps counterintuitive, relationships

[99] Obviously, particular dynamics depend on particular drugs. No particular drug is mentioned here. We imagine an idealized drug that is totally and irreversibly addictive after some small, but hard to predict, number of uses.

between legalization, price, and crime are revealed. And in this light, the role of education is discussed.

PART I. A DRUG EPIDEMIC MODEL

We begin with definitions and a brief discussion of variables and parameters. At any time, the population is assumed to be divided into four *disjoint* groups.

$S(t)$: The nonaddicted and susceptible population.
$I(t)$: The population of addicts, all of whom are assumed, in this simple model, to be pushers. The variable I is used because this group plays a role that is mathematically analogous to the infective group in epidemiology, a parallel we shall exploit.
$L(t)$: The law enforcement, or police, force, whose sole function is assumed to be the arrest and removal of pushers.
$R(t)$: The arrested and removed, or imprisoned, population. For this simple model, removal is assumed to be permanent.

In addition to these variables, a number of parameters are involved.

β: The rate at which a contact between a pusher and a susceptible produces a new addict/pusher (price dependence is discussed in Part III below).
μ: The natural growth rate in the susceptible pool, as youths come of age, say.
γ: The rate at which a contact between a pusher and a cop results in removal of the former.
α: The rate at which an increase in pushers increases the growth rate in police. This variable reflects social alarm.[100]
b: The economic damping to which the police growth rate is subject.

All parameters are nonnegative real numbers.

Let us see if we cannot arrive at a plausible model by reasoning from first principles, noting connections to related phenomena as we go. Pedagogically, the exercise may illuminate the type of reasoning that often goes into the construction of models in mathematical biology, a field which, ultimately, subsumes the social sciences.

[100] Tragically, problems get more attention when they impinge on the elite than when they are confined to the ghetto. In a more realistic model, therefore, α would depend on the socio-economic classes into which drug abuse, and/or the crime associated with it, had spread. Here α is a constant.

As the simplest conceivable model, then, let us imagine that there is no population growth and no police force. At every time t, the population is constant at N and is the sum of susceptibles $S(t)$ and pushers $I(t)$. That is,

$$N = S(t) + I(t)\,. \tag{5.1}$$

How do S and I evolve? Well, for a susceptible to become an addict/pusher, he or she must first come into contact with a pusher. Recognizing that real societies are heterogeneous and patchy, let us nonetheless follow the practice of theoretical epidemiology and ecology and, as a first cut, assume homogeneous mixing of pushers and susceptibles. The number of contacts is then taken to be SI. Of course, only some fraction β of contacts produces new addicts. One may think of β as the "just say no" parameter. If $\beta = 0$, every susceptible says no and there is no growth in the addicted, or "infected," pool. If $\beta = 1$, then every contact produces a new addict/pusher. On these very primitive assumptions, then, the flow out of the susceptible pool and into the addicted pool is fully described by the equations

$$\frac{dS}{dt} = -\beta SI\,, \tag{5.2}$$

$$\frac{dI}{dt} = \beta SI\,. \tag{5.3}$$

This system is none other than the most basic epidemic model, termed an "SI" model since the flow is strictly from susceptible to infective. Now, by virtue of (5.1), we may write $S = N - I$, and (5.3) becomes

$$\frac{dI}{dt} = \beta I(N - I) \tag{5.4}$$

whose solution is the well-known equation of logistic growth. The addicted population increases until it equals the entire population; the "epidemic" whips through the whole of society.

In fact, there are some brakes on this process. Hewing to our assumption that the drug is illegal, there is some rate at which pusher/addicts are removed from general circulation. As a first refinement on our model, let us imagine a fixed police force of size L_0. As in the pusher-susceptible sphere, "law of mass action" dynamics are assumed. There is homogeneous mixing of pushers and police, so that contacts proceed as $L_0 I$. And, per contact, the removal rate is γ. The idea, then, is that, as before, susceptibles flow into the addicted/pushing pool at rate βSI. But, pushers flow out of circulation and into the "removed" class at rate $\gamma L_0 I$. Since γL_0 is just a constant, call it σ. Then we have the model:

$$\frac{dS}{dt} = -\beta SI\,, \tag{5.5}$$

$$\frac{dI}{dt} = \beta SI - \sigma I\,, \tag{5.6}$$

$$\frac{dR}{dt} = \sigma I\,. \tag{5.7}$$

Students of mathematical epidemiology will recognize this as the classic Kermack-McKendrick SIR epidemic model. It is a threshold model in that susceptibles must exceed some minimum level in order for the infected, or addicted, class to grow. This is straightforward. To say the addicted class grows is to say that

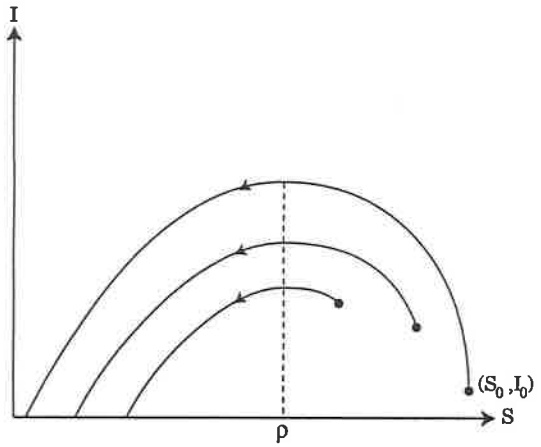
$$\frac{dI}{dt} > 0\,,$$

which is to say that $\beta SI - \sigma I > 0$, or that

$$S > \frac{\sigma}{\beta}\,. \tag{5.8}$$

The ratio σ/β is often termed the relative removal rate of the infection. It is the epidemic threshold. While the infection ultimately dies out—since everything eventually flows into the removed compartment—it decreases monotonically only if $S < \sigma/\beta$. Otherwise, it enjoys a period of growth—the epidemic phase—before dying out, as shown in figure 5.1, in which $\rho = \sigma/\beta$.

FIGURE 5.1 An SIR Epidemic Model



Now, from (5.5), (5.6), and (5.7), it is evident that population is still constant, since

$$\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0\,.$$

Of course, population is not generally constant. There are so-called vital dynamics, birth and death.

As the next obvious refinement on our elementary model, then, let us assume net "births" or entrants of μS into the susceptible cohort, where μ is the per capita growth rate. Needless to say, logistic rather than Malthusian growth is another possibility. But, keeping matters as simple as possible, we then obtain the model:

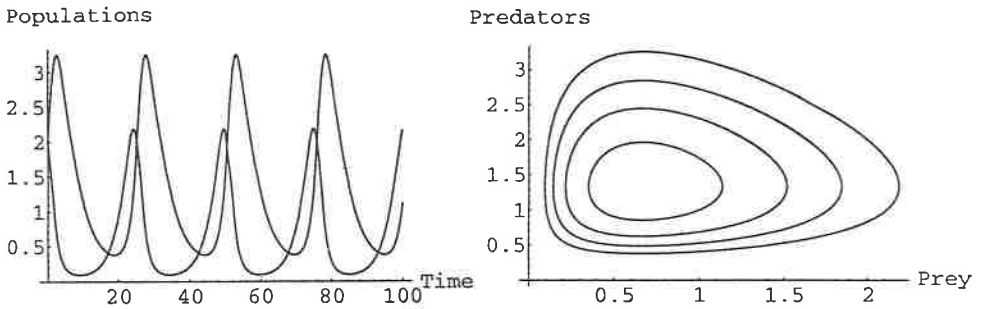
$$\frac{dS}{dt} = -\beta SI + \mu S\,, \tag{5.9}$$

$$\frac{dI}{dt} = \beta SI - \sigma I\,, \tag{5.10}$$

$$\frac{dR}{dt} = \sigma I\,. \tag{5.11}$$

Notice that (5.9) and (5.10) are the classic Lotka-Volterra predator-prey model, with pushers as predators and not-yet-addicted susceptibles as prey. Predators would die out (at rate $-\sigma I$) were there no prey to feed on (at rate βSI); and prey would flourish (at rate μS) were they not consumed (at rate βSI) by predators. Aside from the origin, this system has as its equilibrium the point $(\bar{S}, \bar{I}) = (\sigma/\beta, \mu/\beta)$, which is a center.[101] As shown in figure 5.2, the populations oscillate; the orbits are closed curves in the SI phase plane.

FIGURE 5.2 Lotka-Volterra Predator-Prey Model



Above, we saw that in the SIR epidemic model, the infected or addicted class ultimately goes to zero, as all addicts are removed. Here, we have Lotka-Volterra dynamics in which predators and prey cycle around an equilibrium. Could it be that the complete model will combine these—damped and oscillatory—tendencies in some way? We will return to this question.

[101] As developed in lecture 6, at the equilibrium, the eigenvalues of the Jacobian of (5.9)–(5.10) are imaginary. The equilibrium is nonhyperbolic and linearized stability analysis does *not* apply. But because the eigenvalues are imaginary, the equilibrium is a center or a focus; and because the system admits a Hamiltonian formulation (see lecture 4), the equilibrum is a center or a saddle. Hence, it is a center.

THE ARMS RACE COMPONENT

Above, we defined σ as the product γL_0 where L_0 was some *fixed* level of law enforcement or police. But, the police force is not necessarily constant. So, let us relax this assumption. What, to a first order, would determine the size of the police force? Well, if no one cares about the level of addiction in society, $I(t)$, there will not be any growth. Thinking of the parameter α as a coefficient of societal alarm, we might posit that, without any economic damping, the police force should grow as αI. But, as in arms race modeling, it is reasonable to assume some economic fatigue or damping, under which *rates* of growth decline the larger is the military establishment. If the damping coefficient is b, then the police growth rate is given by $\dot{L} = \alpha I - bL$, just as in the Richardson arms race model of lecture 3, and the complete model is as follows:

$$\frac{dS}{dt} = -\beta SI + \mu S\,, \tag{5.12}$$

$$\frac{dI}{dt} = \beta SI - \gamma IL\,, \tag{5.13}$$

$$\frac{dR}{dt} = -\gamma IL\,, \tag{5.14}$$

$$\frac{dL}{dt} = \alpha I - bL\,. \tag{5.15}$$

Notice that the term $-\gamma IL$ in (5.13) is a pusher attrition rate reminiscent of a Lanchester combat model presented in lecture 2, so this dynamical system combines elements of the epidemic, ecosystem, arms race, and combat models developed in preceding lectures. Before engaging in a linearized stability analysis of this dynamical system, let us briefly trace through the effect if, from some time, everyone says no; that is, if $\beta = 0$. Clearly, since $\beta = 0$, the growth rate in the addicted/pusher pool in (5.13) is strictly negative; this entire group is eventually removed. That being the case, (5.15) reduces to

$$\frac{dL}{dt} = -bL\,,$$

and the police force, too, "withers away," a reasonable qualitative result since the apprehension of pushers is their sole function in this model. In the end, we have a policeless society of nonaddicts and a removed population of former pushers. This little thought experiment completed, let us bring to bear some more powerful tools.

LINEARIZED STABILITY ANALYSIS

Assuming all parameters to be positive in (5.12)–(5.15), what are the nontrivial equilibria of the system, the nonzero population levels where all derivatives are zero? We really care only about S, I, and L, and a bit of algebra quickly leads to the unique positive equilibrium:

$$(\bar{S}, \bar{I}, \bar{L}) = \left(\frac{\gamma}{\beta}\bar{L}, \frac{\mu}{\beta}, \frac{\alpha}{b}\bar{I}\right)\,. \tag{5.16}$$

Evaluated at this equilibrium (call it $\bar{x}$), the Jacobian matrix of (5.12)–(5.14), which ecologists term the community matrix, is given by

$$J(\bar{x}) = \begin{pmatrix} 0 & -\gamma\bar{L} & 0 \\ \mu & 0 & -\frac{\gamma\mu}{\beta} \\ 0 & \alpha & -b \end{pmatrix}\,. \tag{5.17}$$

The eigenvalues are solutions to the third-order characteristic equation

$$\mathrm{Det}(J(\bar{x}) - \lambda id) = 0\,, \tag{5.18}$$

where id is the identity matrix. Expanding, this characteristic equation is

$$\lambda^3 + b\lambda^2 + \left(\frac{\alpha\gamma\mu}{\beta} + \mu\gamma\bar{L}\right)\lambda + b\mu\gamma\bar{L} = 0\,. \tag{5.19}$$

Equilibrium is stable if and only if the roots λ_i of this equation have $\mathrm{Re}(\lambda_i) < 0$. For a third-degree equation,

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0\,,$$

the Routh-Hurwitz necessary and sufficient conditions[102] for $\mathrm{Re}(\lambda) < 0$ are

$$a_1 > 0, a_3 > 0, \text{ and } a_1a_2 - a_3 > 0\,. \tag{5.20}$$

The first two of these are obviously satisfied by (5.19), and so is the third, since

$$a_1a_2 - a_3 = \frac{b\alpha\gamma\mu}{\beta} > 0\,.$$

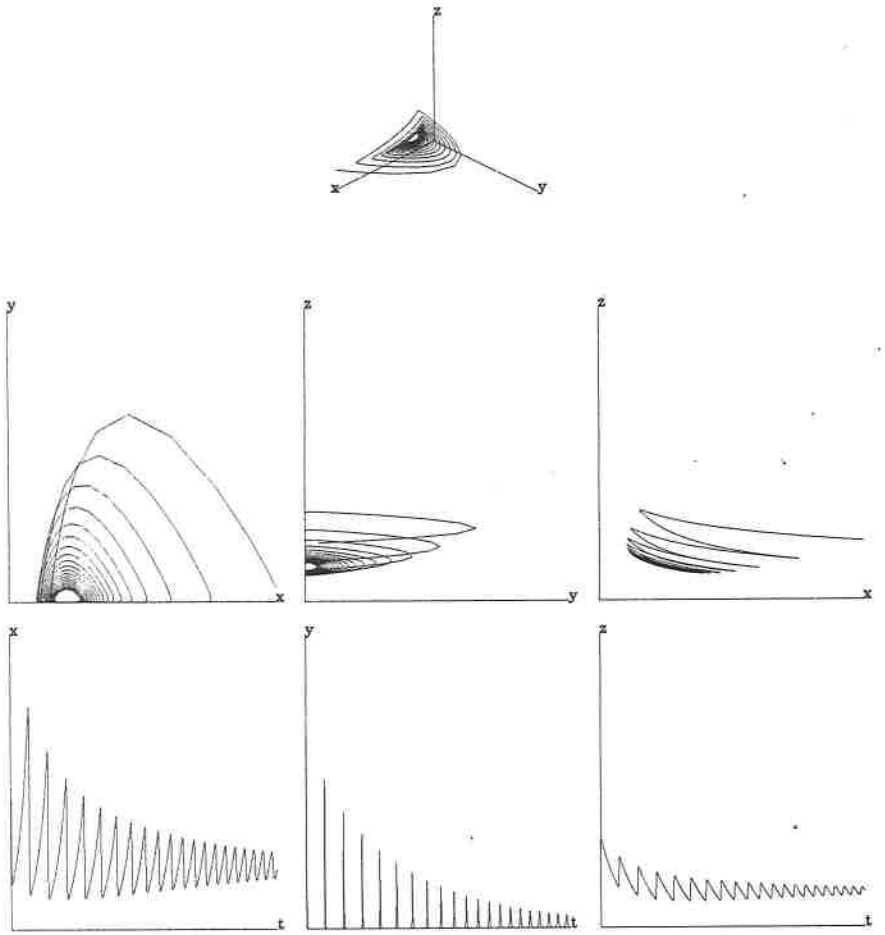
Therefore, the positive equilibrium in (5.16) is *stable*. There is an *endemic* level of addiction.

Earlier, I raised the question whether our simple model might somehow manifest both the damped behavior of the Kermack-McKendrick SIR epidemic model and

[102] See, for example, Murray (1989, pp. 702–04), or May (1974, p. 196).

the cyclical behavior of the Lotka-Volterra predator-prey model, each of which is a special case of (5.12)–(5.15). A canonical behavior combining these would be a spiral approach to our positive equilibrium. And this is precisely the behavior we have, as shown in figure 5.3, which offers a small gallery of phase portraits. Here, the equilibrium happens to be $(\bar{S}, \bar{I}, \bar{L}) = (108, 2, 12)$.[103]

FIGURE 5.3 Drug Model Orbits and Solutions.



Note: Here x is our S, y is our I, and z is our L.

[103]The parameter values employed are: $\beta = 0.1$, $\mu = 0.2$, $\gamma = 0.9$, $\alpha = 0.6$, and $b = 0.1$.

A natural extension is to add space. As demonstrated earlier, this can be ac complished by appending various diffusion terms to an underlying dynamic model yielding a so-called reaction-diffusion system. One such generalization is offere next.

PART II. DRUG WAR ON MAIN STREET: A NONLINEAR REACTION-DIFFUSION MODEL

The population is comprised of three subgroups, whose numbers and spatial dis tributions evolve over time. We imagine that events unfold on a one-dimensiona interval—a "street." Let us define $S(x,t)$, $I(x,t)$, and $L(x,t)$ as the susceptible infective, and law enforcement levels at street position x at time t.[104] Denotin these functions (of x and t) simply as S, I, and L, the generalized equations are a follows:

$$\begin{aligned} \frac{\partial S}{\partial t} &= -\beta SI + \mu S + \delta_{SS}\frac{\partial^2 S}{\partial x^2}, \\ \frac{\partial I}{\partial t} &= \beta SI - \gamma IL - \delta_{SI}\frac{\partial^2 S}{\partial x^2} + \delta_{LI}\frac{\partial^2 L}{\partial x^2} + \delta_{II}\frac{\partial^2 I}{\partial x^2}, \\ \frac{\partial L}{\partial t} &= \xi SIL - bL - \delta_{IL}\frac{\partial^2 I}{\partial x^2} + \delta_{LL}\frac{\partial^2 L}{\partial x^2}. \end{aligned} \tag{5.21}$$

Ignoring all diffusion and cross-diffusion terms, the first two equations are exactl as before. The third equation has been refined slightly. The Richardsonian damp ing term $(-bL)$ is retained, but the first expression is now ξSIL rather than th previous αI. The idea, recall, is that the police force grows with the level of societa alarm at the drug problem itself. This level of alarm is assumed to be a functio of arrests of which the (tax-paying and police-buying) susceptibles are aware. Un der our normal assumption, the arrests are proportional to IL, and susceptibl awareness grows with exposure to these arrests, hence further multiplication by S yielding the overall term ξSIL.[105] These, then, are the reaction kinetics in th reaction-diffusion system (5.21).

Turning to the diffusive processes, the simplest is the susceptible case. Here the term $\delta_{SS}(\partial^2 S/\partial x^2)$ is added, as in the models of the previous chapter, indicat ing that the susceptibles—while interacting with other groups—diffuse. Analogou diffusion terms appear in the equations for infectives $(\delta_{II}(\partial^2 I/\partial x^2))$ and polic $(\delta_{LL}(\partial^2 L/\partial x^2))$. All these diffusivities (δ_{ii}) are positive. However, the infective an police equations are more complex than this. In the police equation, there is als

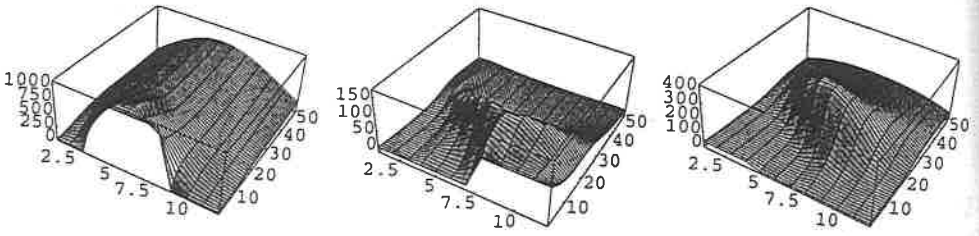
[104]Here, we will not track the removed (i.e., arrested) group explicitly.

[105]We assume again that the exposuress occur through homogeneous mixing, or mass actio kinetics.

a cross-diffusion term ($-\delta_{IL}(\partial^2 I/\partial x^2)$), indicating that police diffuse toward infective concentrations; they engage in "crimo-taxis," if you will. In turn, infectives (i.e., pushers) cross-diffuse in the direction of susceptibles ($-\delta_{SI}(\partial^2 S/\partial x^2)$), and cross-diffuse away from police ($\delta_{LI}(\partial^2 L/\partial x^2)$). I further assume that $\delta_{LI} > \delta_{SI}$: a pusher would rather avoid arrest than convert a susceptible to a new drug user. With all constants set,[106] the assignment of initial spatial distributions for the subpopulations is all that remains to specify the model.

Imagine, then, that everything transpires on a street 12 blocks long. At time zero, the susceptibles occupy the middle four blocks, and are 1000 strong at every point. Up at blocks 8–12 are the infectives, initially numbering but 100 at each point. And way down at blocks 1–3 are the cops, initially at token levels of 25 per point. We track the spatial evolution of each group over 50 time intervals in figure 5.4.

FIGURE 5.4 Drug War Reaction-Diffusion Model



The susceptible, infective, and police evolutions are shown in the left, middle, and right graphs, respectively.[107] At t_0 the levels and positions are as noted above. How do things evolve?

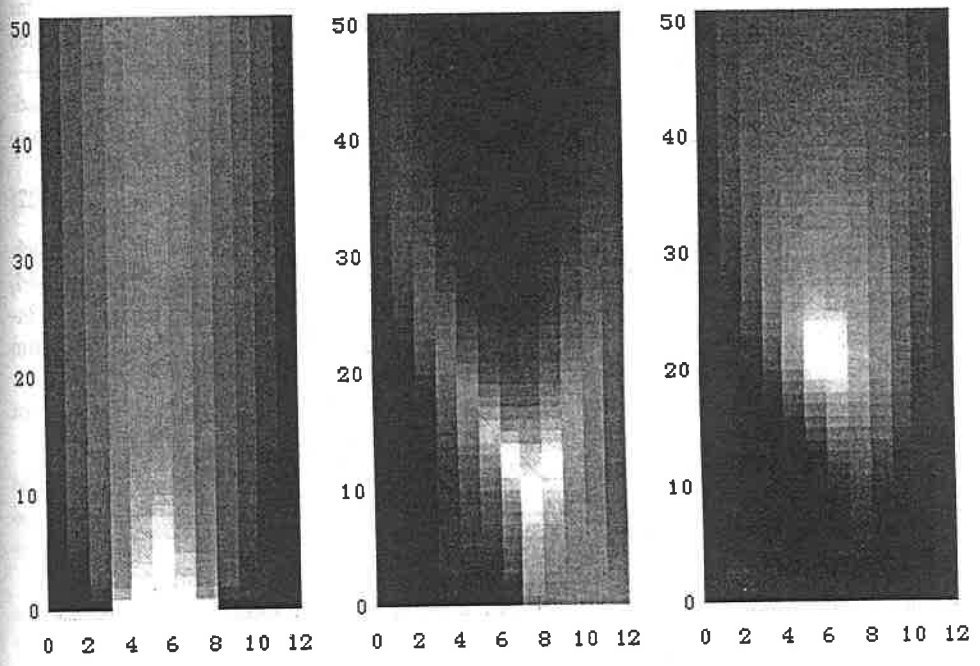
A SPATIO-TEMPORAL STORY

Seeing that there is a large concentration of susceptibles and few cops down the street from them, the infectives cross-diffuse to the center. Many susceptibles are converted into infectives, so the susceptible population falls and the infective one rises, now swelling with "converts" into the middle blocks. This bulging problem,

[106] Here, the values are: $\beta = 0.005$, $\mu = 0.5$, $\gamma = 0.03$, $\xi = 0.0001$, $b = 1.0$, $\delta_{SS} = 0.03$, $\delta_{II} = 0.01$, $\delta_{LL} = 0.02$, $\delta_{LI} = 0.006$, $\delta_{SI} = 0.001$, $\delta_{IL} = 0.006$.

[107] These were generated in *Mathematica* (Wolfram, 1991) using the Numerical Method of Lines. I thank Robert Axtell for his assistance.

FIGURE 5.5 Drug War Reaction-Diffusion Model: Overhead View



however, inspires a reaction, in the form of dramatic increases in police, who cross-diffuse from their initial barracks at the end of the street into the heart of the problem in the center. This surge in police—evident in the peak of the rightmost graph—literally scoops away the infective mound. By $t = 40$, there is hardly a problem. Hence, as before, the police "wither away" after that point, leaving the susceptibles to continue in their untroubled diffusion, as shown.

An overhead view of the same process is offered in figure 5.5. Here, the higher the numbers at a point, the lighter the shade. We can clearly see the surge of pushers, followed by the police response, the hollowing-out of the pusher mound in the center, and the withering away of the police.

A nonlinear reaction-diffusion model allows us to generate a plausible spatio-temporal story of basic interest.[108]

[108] My point here is that there exist parameter values and initial conditions under which the spatio-temporal story emerges. A seperate study would examine the robustness of this result under a wide range of parameter and initial values.