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Simple Processes: One-Way Transitions, Growth, and Diffusion

Many of the dynamic processes that have been of greatest continuing interest to social scientists are relatively simple single-state systems that produce monotonic trends. Psychologists and social psychologists have sought to understand the process of cognitive development; economists and historians have theorized about the processes of economic growth and development; political scientists have focused attention on "political development" and the growth of the state; anthropologists and sociologists have devoted considerable attention to the processes generating increasing complexity of social organization. While the particulars of these research traditions are quite different, they have many commonalities when viewed from a systems perspective as theoretical problems. In each of these cases our primary attention turns to the behavior of a single "dependent" variable, and we seek to understand the forces that lead to its rate of change over time. In most of the models of interest, this change tends to be in one direction: toward increasing development, complexity, or whatever. Of course processes of decline, decay, entropy, and extinction could be modeled as the inverse of growth and development.

Because such systems are so important in all of the social sciences, it is quite important that we grasp their dynamics from the current perspective before we move on to more "complex" problems. In this chapter we will develop a series of related models of such "one-way" processes using the substantive example of the diffusion of some trait in a closed population. This particular problem has received considerable attention in and of itself because of its wide application (e.g., the spread of rumors, diseases, religious conversions, etc.). We have chosen it, however, because the several basic models of diffusion processes provide prototypes for a very wide range of problems. The several models that we will explore are all quite "simple" in terms of their state-spaces, but differ in the complexity of the control structures that drive and limit change in the "dependent" state. They provide a good

illustration of the principles of "complexity of control" that we discussed in the previous chapter, and help us to understand the dynamics of more complex systems—which are made up of these simpler parts.

The Problem: Diffusion in Populations

The spread of ideas, beliefs, and behaviors in human populations is one of the fundamental processes of social change. Social scientists have been intrigued by the puzzles of why some ideas and practices become more widespread than others and why some spread "like wildfire," while others diffuse very slowly. Diffusion has been studied in the adoption of agricultural practices medical technology, the spread of rumors, and a wide variety of other specific contexts.[1] In all of these cases, the central concern is explicitly dynamic: Why is it that the rate of change in the level of adoption or belief in a population is higher or lower? What is the time-shape of the process? How far does the process proceed before it stops?

Because diffusion processes are so important, they have received a good deal of attention from theorists, as well as from analysts concerned with specific substantive problems. There are extensive literatures that utilize statistical and mathematical approaches to formalize and analyze theories about the dynamics of diffusion. The dynamic models that we will develop here are parallel to these statistical and mathematical approaches.[2]

Models of diffusion processes are but one example of a wider class of models of growth and development. Diffusion models take as their dependent variable the probability that a transition occurs from a source (e.g., unaware of a rumor) to an absorbing state (e.g., aware of the rumor). The "realization" of the underlying stochastic process that generates such transitions is the proportion of the population that makes a change in a period of time. There is no reason, however, for us to restrict our attention to systems that deal with qualitative change. Quantitative variables as well (like levels of cognitive, economic, political, or organizational development) can be treated with models like those that we will discuss below.

Developing the Baseline Model

In a fixed population, let us suppose that actors can fall in one of two categories: Either they don't know of a proposed innovation or they do.

Alternatively, one could think about dividing the population into groups that have adopted or not adopted an innovation, or that do and don't display any trait of interest. At any point in time, some proportion of the population are "knowers," and this proportion changes over time as some of those who don't know become knowers. Those who "know," however, never become "nonknowers"; the process is unidirectional.

The "material states" or levels in this case are two aggregates: those who "don't know" and those who do. These are conserved quantities (a given actor must either know or not know, but not both), connected by a single flow rate from not knowing to knowing. The flow in this case goes in only one direction, as those who don't know become aware (we leave aside, for the moment at least, the elaboration of the model to include people who "forget"). Since the flows are in one direction only, the state "knower" is an "absorbing state," and there is a single transition rate. The chain of material states for this model, then, would be diagramed as in the first panel of Figure 7.1. A single "level equation" defining the number of knowers can represent this aspect of the process. We also want to keep track of the number who don't know as a separate or "auxiliary" quantity for reasons that we will explain shortly. Thus:

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L     K.K = K.J+(DT)(RI.JK)
A     POP.K = K.K+DK.K
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The level equation here says that the number of knowers at the later point in time (K.K) is equal to the number at the prior point in time (K.J) plus the integration or accumulation (DT) of the rate of increase or transition (RI) from not knowing to knowing over the time interval between J and K. Now we need to make hypotheses about the causes of change in the number who know or the rate of transition. This is where the model begins to become interesting.

Stimulus-Response Control

Although we can clearly do better, let's start theorizing by thinking about the system in question as one characterized by dumb or simple stimulus-response control. In such a system, the rate of change is a function of only exogenous factors: constants, noise, and the action of independent variables. One very common baseline diffusion model is to suppose that there is a single exogenous source of stimuli that operates at a constant rate over time. In this case, the model would have the very simple form:

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R     RI.KL = EXOG.K
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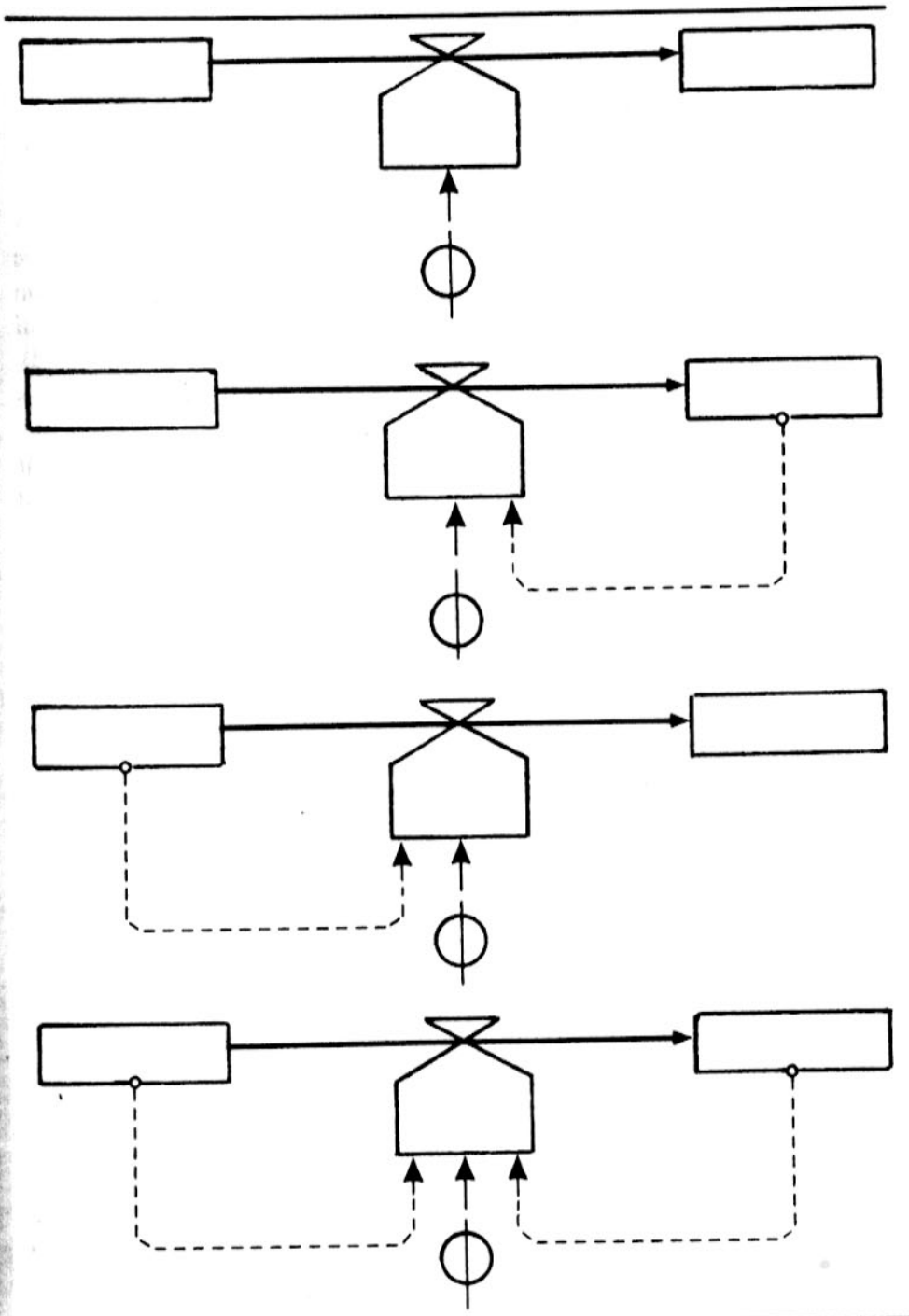

Figure 7.1: Diffusion models.

A EXOG.K = Constant

Suppose, for example, we were interested in customer awareness of a new product advertised on television. If we ran the advertisement at a

constant 100 times per day over the period of interest, we might suppose that the number of persons aware of the product was a simple function of the number of times that the advertisement had been run. There are a number of problems with this theory of how diffusion processes work, that we shall return to presently.

This formulation of the dynamics of one-way transitions focuses our attention on the nature of the exogenous stimulus and its effect on the rate of change. This effect might be hypothesized to be constant function of stimulation (e.g., each time the advertisement is run, x number of people become aware of the product until everyone has been converted), or might be regarded as having (probably decreasing) marginal returns such that the second advertisement convinces fewer people than the first, and so on. As elaborations on this very basic approach, we might choose to model the rate of external stimuli as a function of causal variables, or include delayed response of the system to stimuli as parts of the model.

Self-Referencing Control

As useful as simple stimulus-response models of diffusion (or growth, or survival) are, they have a number of obvious shortcomings.

One major problem is the assumption in these models that the "observations are independent." As we've formulated the problem so far, the only source of stimulus is exogenous. That is, there is only "point source" diffusion in that the knowers don't become tellers. There are many cases of diffusion where such a presumption might be reasonable (or at least a reasonable approximation), as in the case of advertising in mass media. While advertisers, of course, hope that "knowers" will become "tellers" of others, they may not to any great degree. Analogously, if we applied the model to the biological survival of humans, the death of one individual probably has relatively little effect on the survival chances of others.[3] But, there are many cases where those who do "know" become sources for further diffusion. In the case of the spread of disease, for example, the rate of change in the number of persons ill is a positive function of the number who are already ill, because those who are sick infect others. The spread of rumors in groups or the process of economic "takeoff to sustained growth" can be seen as similar processes.

From the systems analysis perspective, this kind of a theory is quite different from the stimulus-response model. The current theory supposes that the process of diffusion is a self-referencing one: The rate of change depends upon the current state of the system (that is, the number who become aware in a period of time depends on the number who already

know). The process has become self-referencing, and now can be diagramed as in the second panel of Figure 7.1. Exogenous sources of stimulus may or may not be included, as we see fit, and the "rate equations" take the following form:

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R     RI.KL = EXOG.K+f(K.K)
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The key question here is "f." That is, in what way does the rate of change in knowing (RI.KL) depend on the number who already know (K.K)? One obvious thought is that each "knower" becomes a "teller" at a constant rate (or, in a more stochastic variation, each knower has an expected value and variance in telling). In this case, the function "f" becomes a constant (or distribution) number of tellings per "knower" per unit time.

Another very common variation on this model proposes that those who become knowers early in the diffusion process are more enthusiastic tellers than those who come later. In this case, the value of the function "f" is itself a function of the level of the process, declining as some rate as K.K increases. For example, each knower at the early stages of the diffusion may tell five new people per unit of time; those who become converted later may be less enthusiastic, telling only one or two new people per unit of time. In the DYNAMO language, we might represent such hypotheses with a table expression to draw a picture of the hypothesized relation, or use some mathematical function. For example:

```
R     RI.KL = TELLING.K
A     TELLING.K = TABLE(VIGOR,K.K,0,100,20)
T     VIGOR = 5/4/3/2/1/0
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The first statement sets the rate of change in those knowing (RI) to a quantity called "telling." This is simply a convenience to help keep the logical steps clear. The second statement is a calculation involving the table function. It says that the value of the term TELLING at any time point K is defined by a table called VIGOR (an arbitrary name) that defines values corresponding to the variable K.K between 0 and 100 in increments of 20. The last statement identifies the values corresponding to K = 0, K = 20, K = 40, K = 60, K = 80, and K = 100 as 5, 4, 3, 2, 1, and 0, respectively. That is, as K.K increases from 0 to 100, the rate of telling declines from five tellings per unit time to no tellings per unit time. The function defined here is arbitrary, and one might wish to use an ogive, gamma, Weibull or other parametric form.

A third frequent variation on the same theme is to hypothesize that each new knower is initially enthusiastic and tells others at a high rate, but gradually becomes less enthusiastic. That is, tellers become "exhausted." The rate of telling in this case is a negative function not of the level of the process, but rather of the rate of increase in the process. That is, when a large number of people become knowers in a period of time, the rate of telling increases even more rapidly. When conversions are few, there are few enthusiasts, and the rate of telling declines. Processes of this type can be represented by setting the rate of telling to be a "delay" of past rates. The hypothesis of initial enthusiasm followed by rapid decline could be effectively captured with a first-order delay; an alternative hypothesis might suggest that new converts are initially hesitant, but then increase telling for a time before losing enthusiasm. This latter process could be captured by making current rates of conversion a function of a third-order delay of past rates.

Each of these variations has received some attention in theoretical and empirical literatures on growth, survival, and diffusion, and may be more or less applicable to a particular case. The important commonality across these variations is that the rate of increase (or survival rate, or transition rate) is dependent in some way upon the current level of the process itself. In short, these theories of diffusion, growth, survival, and other such one-way transitions are self-referencing and controlled by feedback.

Goal-Referencing Control

The various theories that we have examined so far deal with the ways in which growth and diffusion depend on external stimuli and the ways that they are self-generating. But we have missed something important. All of the processes that we have discussed so far can be used to explain why growth or diffusion occur, but have little to say about why it stops or is limited. To capture this aspect of the processes we must add negative feedback.

Whatever the source of the stimuli, be they from the environment or from actors who already know, not all "tellings" result in conversions from "not knowing" to "knowing." Since "knowing" is a final or absorbing state, when stimuli are directed at those who already know, they have no effect, and hence have no consequence for rates of change. In closed populations or where there are resource constraints, growth and diffusion processes are inherently limited by the supply of available unconverted resources (be they raw materials of some sort, people, or whatever).

As an elaboration of this basic idea, we might suppose that all of the people in the population are not equally "at risk" of making a transition, even if they are exposed to stimuli. Some may be completely resistant to change. For example, people who do not have television sets will never make purchases of products that are advertised only on television. This kind of "population heterogeneity" is similar to the "mover-stayer" problem in social mobility analysis, and has often been dealt with in models of one-way transitions by supposing that there are a group in the population who cannot be reached, so that the diffusion dynamics operate with an "upper bound," or ceiling that is less than the whole population. In this case, this upper bound of "mobilizable" population serves as a "goal" state affecting the rate of transition.[4]

Alternative sets of assumptions about the distribution of "mobilizability" or resistance to exogenous or self-generating stimuli are quite frequently used in "survival" analysis. Among the most common of these sets of assumptions is that of a "liability of newness" in survival chances or, stated inversely for purposes of diffusion rather than survival analysis, decreasing marginal returns to stimuli.[5] Roughly, this assumption suggests that the odds that a given stimuli will reach a given member of the population are distributed as some form of negative exponential. A relatively large proportion of the population is quite easy to convert, but smaller additional proportions are increasingly difficult to reach. In statistical models of such processes, various distributions are often used to represent the distributions of such odds of "resistance" to transition.

Thinking about this aspect of the problem from the systems perspective, we would say that there is a connection between the rate of change and the system state representing the population of those who have not undergone transitions, as is shown in the third panel of Figure 7.1. The number of unmobilized or unconverted in the population acts as a constraint or goal state that is referenced in determining rates of transition. To capture this aspect of the problem, our rate equations must now have this general form:

R RI.KL = EXOG.K+g(DK.K)

Where DK.K is the difference between the total population and the number who already know.

The question for the theorist in this model becomes In what way does the rate of increase in the population of knowers depend on the number who don't already know? That is, what is "g"? DK.K represents the pool of those available for conversion from not knowing to knowing, and its

impact on rates of change has been conceptualized in several alternative ways.

The most obvious way in which the rate of transition depends on the number of actors who have not yet made transitions is as an absolute upper bound. That is, the rate of transition cannot, logically, exceed the number of actors "at risk." Thus, even if our model predicted that there were 100 stimuli to change ("tellings") generated by either exogenous sources or by actors who already had made the transition, if there were only 10 actors available who had not already been converted, 90 of the tellings would, necessarily, fail to result in conversions. This limitation could be represented with the simple statement:

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R     RI.KL = MIN(TELLING.K,DK.K)
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That is, the rate of transition (RI) is equal to either the rate of telling (as we discussed it in several alternative formulations above), or the number of remaining actors at risk (those who "don't know" or DK), whichever is the smaller.

This first formulation of the negative feedback or limiting effects of the size of the population at risk assumes, implicitly, that a given telling or stimulus to make a transition will reach an actor at risk if one is available. This may be a reasonable model in cases where it is easy for "tellers" to easily distinguish between those who have already made transitions and those who have not, so that stimuli are directed only at the unconverted. In many cases, though, this seems rather unrealistic.

We might find an alternative assumption more reasonable for some transition processes: that stimuli are distributed at random, and, consequently the probability that a given stimulus will reach an actor that has not yet made a transition is simply equal to the proportion of the population that have not yet changed. The number of conversions or transitions, then, is equal to the number of tellings or stimuli multiplied by the odds that a given telling reaches an actor still "at risk." That is,

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A     PR.K = DK.K/POP
R     RI.KL = (TELLING.K)*(PR.K)
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Here the rate of transition (RI) is limited by the proportion of the population who are at risk (PR), regardless of what process is generating the stimuli. This statement implicitly incorporates the insight of the previous one; where there are no unconverted available, the probability of success (PR) becomes zero, and consequently the rate of transition is zero.

All of the models thus far assume, again implicitly, that all of the actors who have not already made transitions are equally likely to, should they be exposed to a stimulus. In some cases this is a reasonable assumption, but more often it is not. Actors are likely to differ from one another (i.e., they are "heterogeneous") in a large number of ways that affect the probability that they will undergo a transition if they are exposed to stimuli. Physicians presumably differ in their propensity to experiment with new technologies, some being more willing to try new treatments, some being more conservative. Some peasants are more likely to take chances on a new variety of seed recommended by a government agricultural agent than others.

A variety of assumptions might be made about the ways in which individual differences among those who have not made transitions affect the rate of transition. The most common such models of "population heterogeneity" in transition probabilities assume that resistance is distributed according to one of several statistical models. Alternative models suggest that (1) most individuals have low resistance, and progressively fewer and fewer have higher resistances, or (2) resistance is "normally" distributed, with relatively few individuals having low resistance, most having some, and again relatively few having high levels of resistance.

These alternative assumptions about the distribution of resistance to change in the population can be embodied in our model by assuming that those easiest to convert are earliest to make transitions. Consequently, the resistance to conversion increases nonlinearly (either monotonically or nonmonotonically, depending on how one believes resistance to be distributed) as the size of the unconverted population declines. Rather than using the simple multiplier of the previous set of equations, a mathematical or table function is used to define the probability (PR) that a given stimulus results in a conversion:

```
A    PR.K = f(DK.K/POP)
R    RI.KL = TELLING.K*PR.K
```

where f is a mathematical function like a logarithm, or an arbitrary function (like a TABLE statement) reflecting the dependence of the probability of a successful telling on the proportion of the population who are at risk.

One could, of course, go further in developing baseline models, and we will suggest some additional possibilities after a time. The basic forms of "dumb," "self-referencing," and "goal-referencing" control models, though, are of great generality and importance. It is worth spending some time with them and understanding their dynamic behavior.

Dynamics of the Baseline Model

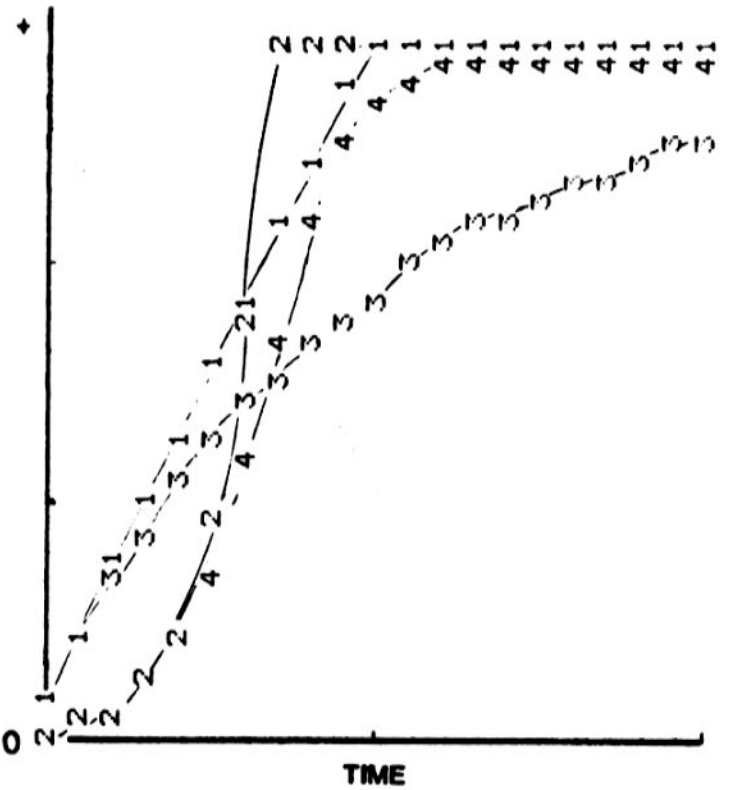
Over time patterns of monotonic growth (or decline) can be produced by theories that are quite different from one another. In their simplest forms, however, the stimulus-response, self-referencing, and goal-referencing control structure models have different characteristic dynamic behaviors. Before considering some slightly more elaborate variations, let's perform simulation experiments to get a firm grasp on the shapes of the over-time growth paths implied by the models discussed in the previous section.

We will consider four alternative formulations of the problem. On one hand, the stimuli to change may be either exogenous or endogenous. In the case of diffusion processes, this is equivalent to having the "telling" come either from external sources (e.g., mass media), or from "knowers" becoming "tellers" (as in rumor processes). On the other hand, limitations on the extent and rate of change (goal-referencing negative feedback) can be either a simple function of the available resources, or can be seen as increasing as resource limits are approached. In the case of diffusion, these alternative assumptions are equivalent to supposing that "tellings" are directed only at those who do not know (until there are no more), or that "tellings" are distributed at random, and are hence successful in direct proportion to the percentage of the population that do not already know.

Combining the two alternative ideas about the causes of growth and the two alternative ideas about the way that growth is limited produces four possible models, each with a characteristic dynamic behavior. In Figure 7.2, plots of the time traces of simulations of models of these four types are shown (the DYNAMO program to produce the four scenarios is appended).

The first trace line in the figure is produced by a model that embodies the hypotheses that change is driven by exogenous factors (at a constant rate of five "tellings" per unit of time), and that all tellings reach and convert nonknowers until the supply of such actors is exhausted. The growth path is quite predictable from these assumptions: Increases in the number of actors who know are linear until no more are available for conversion, at which point the rate of change becomes zero.

The second trace line supposes that there is no exogenous stimulus to change, that one individual initially "knows," and that each individual who "knows" tells, on the average, .75 other actors per unit of time. As in the first example, "telling" is directed only at those who don't already know and continues until there are no more such actors. The over-time behavior of this process is fundamentally different from the first, as it produces exponential rather than simple accumulating growth. This, of



1. constant telling
2. knowers become tellers
3. constant telling with proportional success
4. knowers become tellers with proportional success

Figure 7.2: Basic models of diffusion.

course, is a direct consequence of the self-referencing positive feedback loop: Knowers become tellers. In the first time period there is one "knower," in the second time period 1.75, in the third 1.75 plus 1.75(.75) etc. The process here is one of contagion in its most rampant form.

In the third scenario we combine the idea of exogenously driven growth with the notion that the rate of success of these tellings is proportional to the number of actors who don't already know. That is, in the early stages of the process virtually every "telling" results in a conversion to knowing; in later stages, most telling is redundant. The consequence of this change in the theory is quite notable. The growth trace is no longer linear to an upper bound (as in the first model), but is now a "waning exponential" in which the rate of change is inversely proportional to the distance from the "goal state" (i.e., complete elimination of nonknowers).

In the final scenario the notions of self-referencing growth and goal-referencing limitation are both present. This model is similar to the second, in which one individual initially knows, and all individuals who know tell others. However, in the current model these tellings are random and are successful in direct proportion to the size of the nonknowing population. This model produces what is often thought of as the classical or typical diffusion pattern of S-shaped growth. In the early stages, the rate of change accelerates as more and more knowers also become tellers. In the later stages growth slows as a consequence of the increasing redundancy of the tellings.

For different phenomena, the four models shown in the figure may provide more or less plausible baselines. The important point about this exercise follows from the differences among these models. The simplest of dynamic models (those of monotonic change) have inherently different realizations depending on the presence or absence of self and goal-referencing positive and negative feedback. The theorist must, therefore, give careful thought to the ways in which a particular case of growth, transition, survival, or diffusion may be self-generating and/or self-limiting. Careful prior thought about the causes of change and limits on change is doubly important because, as we shall see below, it is possible for models from quite different theories to produce realizations that appear quite similar. Here, as in most other cases of dynamics, it is often quite dangerous to reason backwards from the data to the theory.

Variations: Exhaustion and Heterogeneity

The basic models of diffusion processes can be elaborated in a number of ways to make more realistic and interesting theories. Two of the most common of these variations are: (1) to make different assumptions about the propensities of knowers to become tellers and (2) to make different assumptions about the distribution of resistance to change in the population. With little difficulty, the basic models can be elaborated to explore these variations.

Exhaustion

In the self-referencing models that we have considered so far (i.e., models in which all "knowers" become "tellers"), we have assumed that all of the individuals in the population are the same in their propensity to become tellers, and that they "tell" at a constant rate. In many circumstances both of these assumptions might be questionable. One might offer the alternative hypotheses that those who are converted

early in the process of diffusion are more enthusiastic than those who are converted later, and hence are likely to "tell" at higher rates. One might also suppose that the rate of telling by those who know varies with time: Perhaps converts are initially more likely to try to convert others, but their enthusiasm for telling declines after a time. Let's take a closer look at the consequences of this latter idea—that tellers become "exhausted."

The notion that knowers become "exhausted" can be captured by use of the DELAY functions in DYNAMO. By setting the rate of new tellings proportional to "delayed" changes in the number of knowers, alternative time-shapes of telling can be represented. In Figure 7.3 three alternative scenarios are presented that make different suppositions about the rate at which knowers become tellers.

The leftmost trace in the figure (#) corresponds to the hypothesis that knowers become tellers immediately, and continue telling at constant rates until the population of available nonknowers is used up. The second trace-line (*) is generated by a model that utilizes a first-order exponential delay. This time-shape corresponds to tellers having high levels of enthusiasm initially, but then declining exponentially in their rates of telling. The third trace-line in the figure (+), utilizes the third-order or S-shaped delay function. This shape corresponds to a hypothesis that new converts are initially hesitant to become tellers but then become tellers with high intensity before becoming exhausted.

The most obvious difference among the three realizations shown in Figure 7.3 is the speed with which the diffusion process is completed. With no exhaustion in telling, of course, the process operates much more rapidly than with either of the other two hypotheses. The model that supposes initial enthusiasm (the first-order delay) operates more rapidly than that which supposes initial hesitancy on the part of new knowers (the third-order delay) because of the multiplication of all changes through the self-referencing positive feedback of the system.

Beyond the difference in the speed of the process, it is very difficult to tell the three scenarios of Figure 7.3 from one another. All three have the same exponential growth pattern, and appear (in this scale of plotting at least) to have identical smooth traces. The important lesson in this observation is that rather different theories can often produce results that are nearly indistinguishable at the empirical level.[6] The three trace lines in Figure 7.3 could differ because the basic rates of telling differ, because some or all have delays of different average lengths, and/or because of different time-shapes of the exhaustion of telling. The distinctions among these theories may be rather marked, as might the effects of policy interventions based on belief in the efficacy of alternative models.

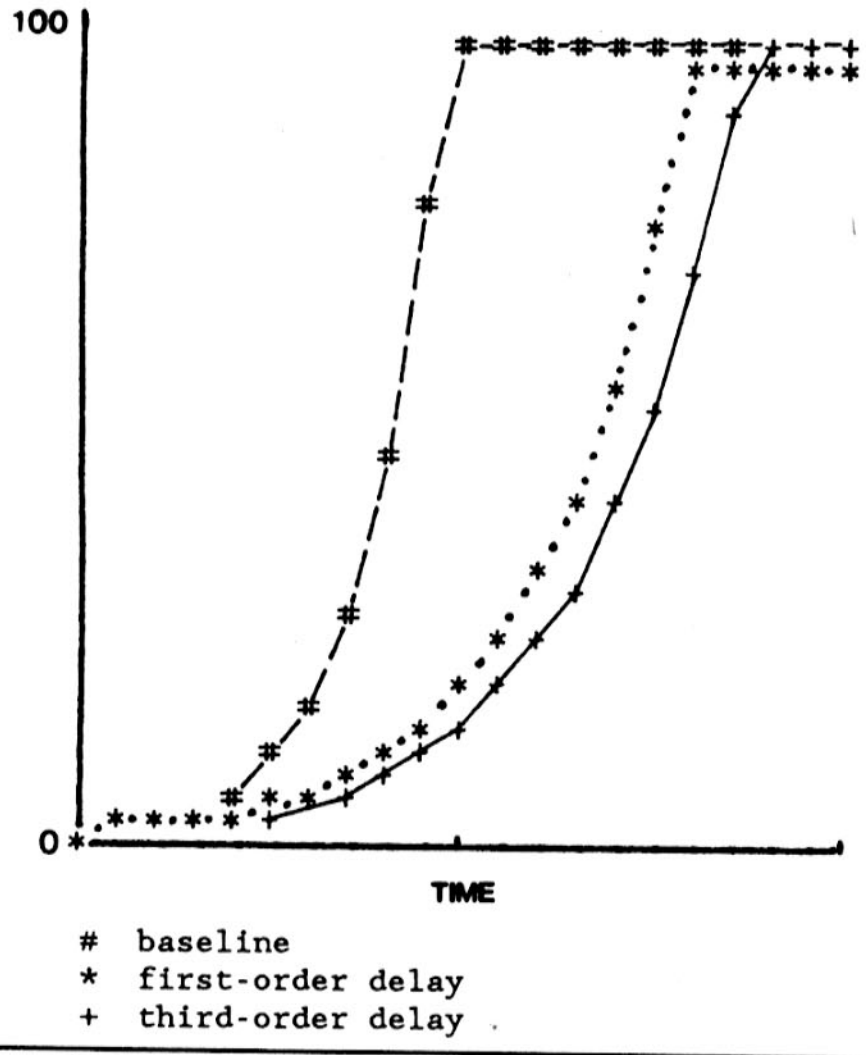


Figure 7.3: Exhaustion of telling.

Heterogeneity

In the theories that we have modeled thus far we have made assumptions about the distribution of propensities of individuals to make transitions that may be unrealistic. We have hypothesized either that all tellings result in conversions, or that the probability that a telling results in a conversion is a linear function of the distribution of the population between those who know and those who don't know. In many cases it might be more realistic to assume that the propensities to change are not homogeneous across the individuals in a population.

Rather than assuming that individuals will automatically undergo

a transition if they receive a stimulus (and have not already been converted), we might suppose that individuals differ in their resistances. The most common models embodying this idea of heterogeneity assume that the underlying distribution of resistance to change is described by either a negative exponential or cumulative normal (S-shaped) distribution. In the former case, most individuals have low resistance to change, and will undergo transitions if stimulated, but progressively fewer and fewer have higher and higher resistance. In the latter case, most individuals are seen as having moderate levels of resistance to change, while relatively few are very likely and very unlikely to change if simulated.

We can explore the consequences of these alternative hypotheses by modifying our basic model slightly. As a baseline, we will assume that the population is homogeneous in the probability that a telling will result in a conversion, and that telling is distributed at random from all knowers. This is the same as the fourth of the baseline models discussed previously. As a first alternative hypothesis about the distribution of resistance, we will suppose that most people are relatively easy to convert (i.e., that resistance is distributed as a negative exponential). This alternative hypothesis is embodied in our model by using a table function to map such a relationship between the number of persons in the population who don't know, and the probability that a telling will result in a conversion. That is, as the proportion of the population who have not been converted declines, the probability of further conversions also declines. As a second alternative hypothesis, we will assume that the probability that a telling fails to result in a conversion is a cumulative normal (S-shaped) function of the proportion of the population who have not already been converted. That is, the probability of conversion as a consequence of telling is initially low, increases up to the mean, then declines again as more and more of the population are converted. This hypothesis is also modeled by using a table function to map the relationship between the proportion of the population who have not already been converted and the probability that a telling will result in a conversion (the DYNAMO code for these models is appended). The results of simulations of the three alternative assumptions about the distribution of resistance are shown as Figure 7.4.

The most important thing to notice about the results of these experiments with alternative hypotheses about the distribution of resistance to change is that the shapes of the curves differ. The baseline model (*) of population homogeneity describes a smooth and symmetric S-shaped curve over the time period, a consequence of similar shapes of the effects of the positive and negative feedback forces. The other two

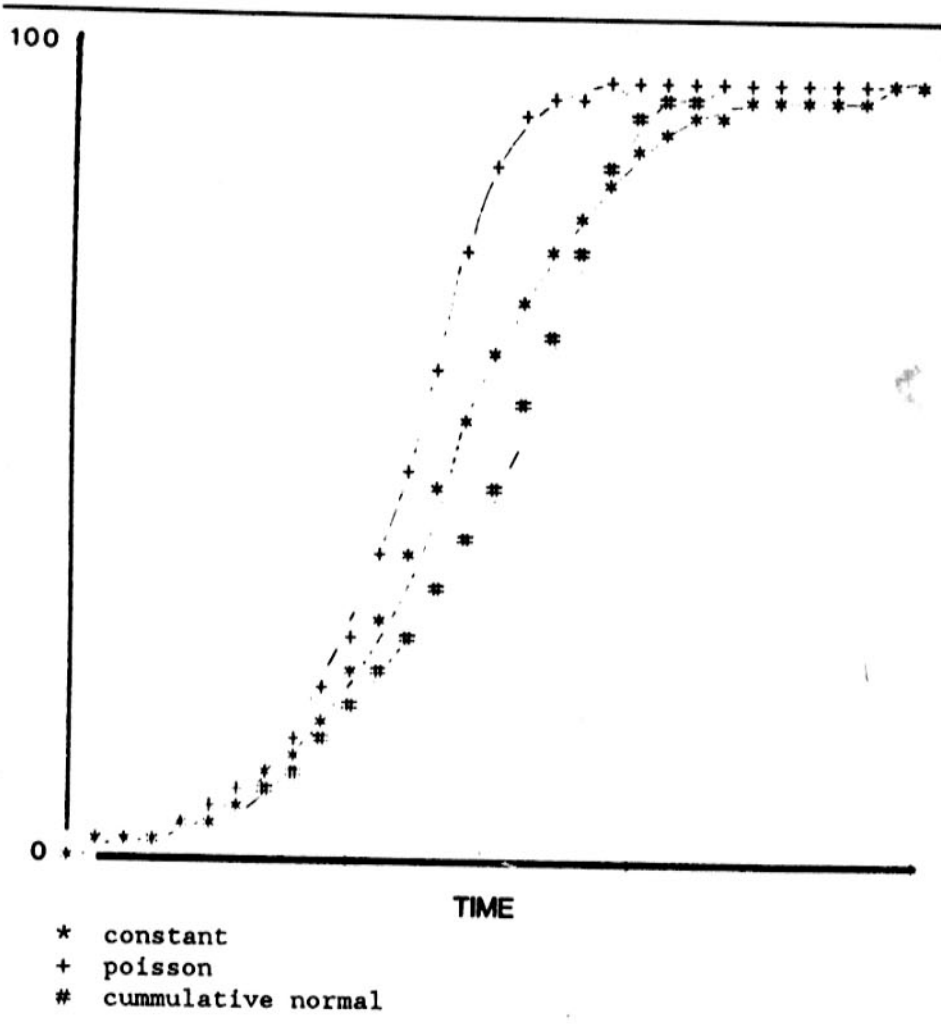


Figure 7.4: Heterogeneity.

models do not produce symmetric patterns.

If we assume that a large part of the population has little resistance to change, we would expect the early stages of the diffusion process to be more rapid than under the assumption of homogeneity. And, indeed, this is the case, as shown by the trace line (+) of the exponential model. Since some of the population has quite high resistance to change under the assumption of the exponential model, however, the final stages of the diffusion are slower than under the assumptions of the baseline model. One might describe the consequences of assuming a negative exponential distribution of resistance, then, as skewing the whole diffusion process to the "left." Although we do not perform the calculations here, the inverse assumption of a positive exponential

distribution of resistances (that is, most have high resistance) is to produce a "skew" to the "right."

The assumption that resistance is normally distributed, rather than constant across members of the population produces a more complicated pattern (the #s in Figure 7.4). Since there are relatively few actors who are easy to convert under this hypothesis, the diffusion process is slower initially than in the baseline example (though it is difficult to see this in the figure due to the small number of converts in the early stages of either process). As the large "middle mass" of actors are reached and converted, however, the process becomes more rapid than in the baseline (compare the slopes of the *s and the #s in the middle of the diagram). Finally, as we reach the advanced stage of the process in which only those with high levels of resistance to change remain, the process again moves more slowly than in the baseline example.

The alternative hypotheses about the distribution of resistance in the population produce results that are importantly different: The shape of the realizations differs, not just the speeds of the processes. Again, however, the theories might be said to be more different than the empirical realizations. While the three sets of assumptions that have been made about the distribution of resistance in the population are very different, the resulting diffusion curves are sufficiently similar that exploratory data analysis might not distinguish them.

An Elaboration: Contagion

A wide range of phenomena can be usefully conceptualized as one-way transition processes or monotonic growths or declines. An even wider array of interesting social dynamics can be captured with quite minor modifications of the models that we have considered thus far. To get a flavor of the possibilities, let's consider one more elaboration on the basic diffusion model that enables it to describe the process of contagion. While the model here is created specifically with reference to infectious disease, the spread of rumors, fads, and crazes might also be analyzed with modifications of this model.

In the processes that we have been considering so far, the "event history" of individuals consists of two states: For a time individuals are "nonknowers," then they make a transition to the absorbing state of "knowing." Nonfatal infectious diseases have a slightly more elaborate historical process: First one is well, then ill, and then recovered (assuming that the disease did not produce death). In the case of a fad or fashion cycle, individuals are at first unconverted, become active

believers for a time, then "drop out." Some may, however, enter an alternative absorbing state of "permanent convert." If we allow only a single absorbing state (i.e., the disease is not fatal, or everyone eventually gives up on the fad), a simple chain of three states describes the "material flows" of such a process, as in Figure 7.5.

Since movements among the states go in only one direction, two rates (an infection rate and a recovery rate) are sufficient to capture the dynamics of the situation. Infectious diseases are spread by direct contact between persons who are currently ill and those who have not yet been infected. Consequently, the rate of infection references both of these states, creating the same dynamic of self-generating and self-limiting growth that we explored previously. In the current model, however, there is a difference. Not all persons who have ever been ill act as sources for further spread of the disease. After a period of time being ill, individuals recover and no longer act as sources for further infection. The recovery rate (number of persons recovering per unit time) is determined by the number who are currently ill (and hence "at risk" of recovering) and a constant reflecting the average time it takes for recovery to occur.

The diagram of this process points out the central role played by the number of persons currently ill in this model. The number of people who are currently ill has effects on both the rate of recovery and on the rate infection and hence is the key to the model. How many people are ill at any given time of course depends on the balance of the intensities of the infection and recovery processes. These, in turn, are critically dependent upon the assumptions embodied in the "contact rate" between ill and susceptible persons, and on the recovery time constant. The effect of the contact rate is obvious: The more that sick persons come into contact with susceptible ones, the larger the power of the exponential growth tendency of the model. The effect of the recovery time constant is also fairly obvious: The longer that persons are ill, the more rapid will be the contagion (holding constant the contact rate). Because these two loops both reference the number of persons who are ill, the rate at which the diffusion occurs is dependent on the interaction of these two terms. If the contact rate were zero, for example, it would not matter what the recovery time was; similarly, though less immediately apparent, if recovery times are sufficiently short there will be no spread of the disease (because no people are ill long enough to come into contact with susceptible while they remain infectious).

The time patterns produced by such a model are shown in the simulation in Figure 7.6 (the DYNAMO program is, again, appended).

In this model the basic features of our two-state diffusion processes

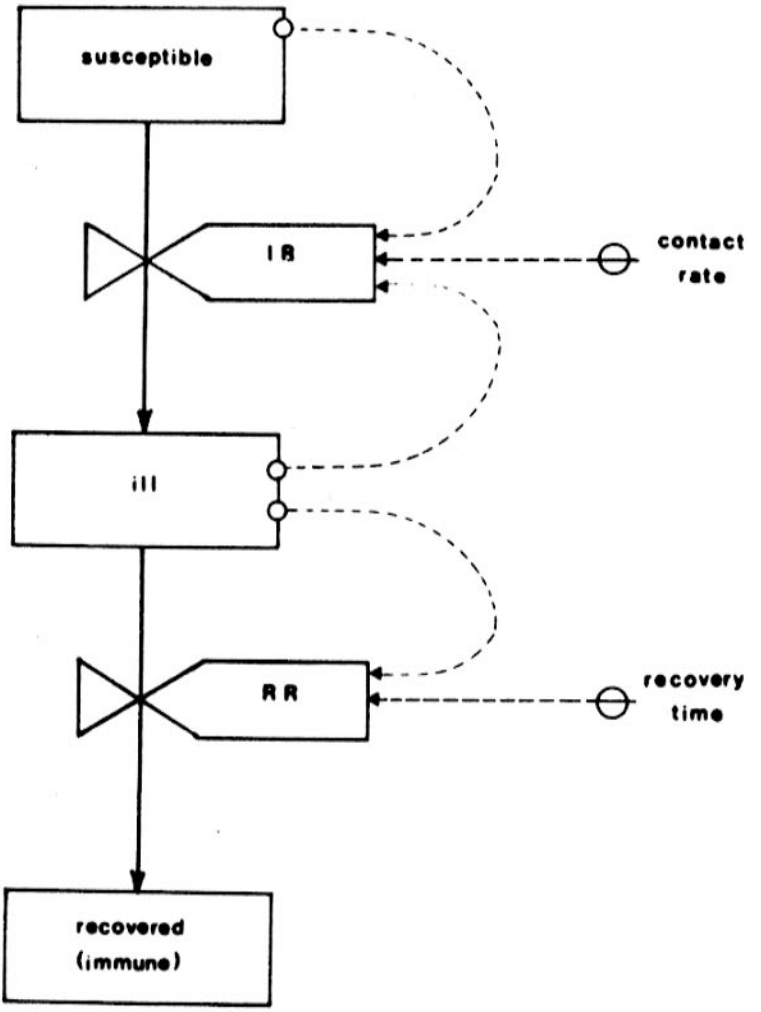


Figure 7.5: Contagion.

are preserved. The number of persons at risk (i.e., susceptible) follows an S-shaped downward path, and the number of recovered follows the now familiar upward S-shaped trajectory. The two curves, however, are not mirror images of each other: The growth of the immune population occurs only after the delay of the average length of the illness period. In this model then, the new state of "ill" is acting as a "delay," similar to the delays introduced in the models previously discussed where we assumed that tellers became "exhausted".[7]

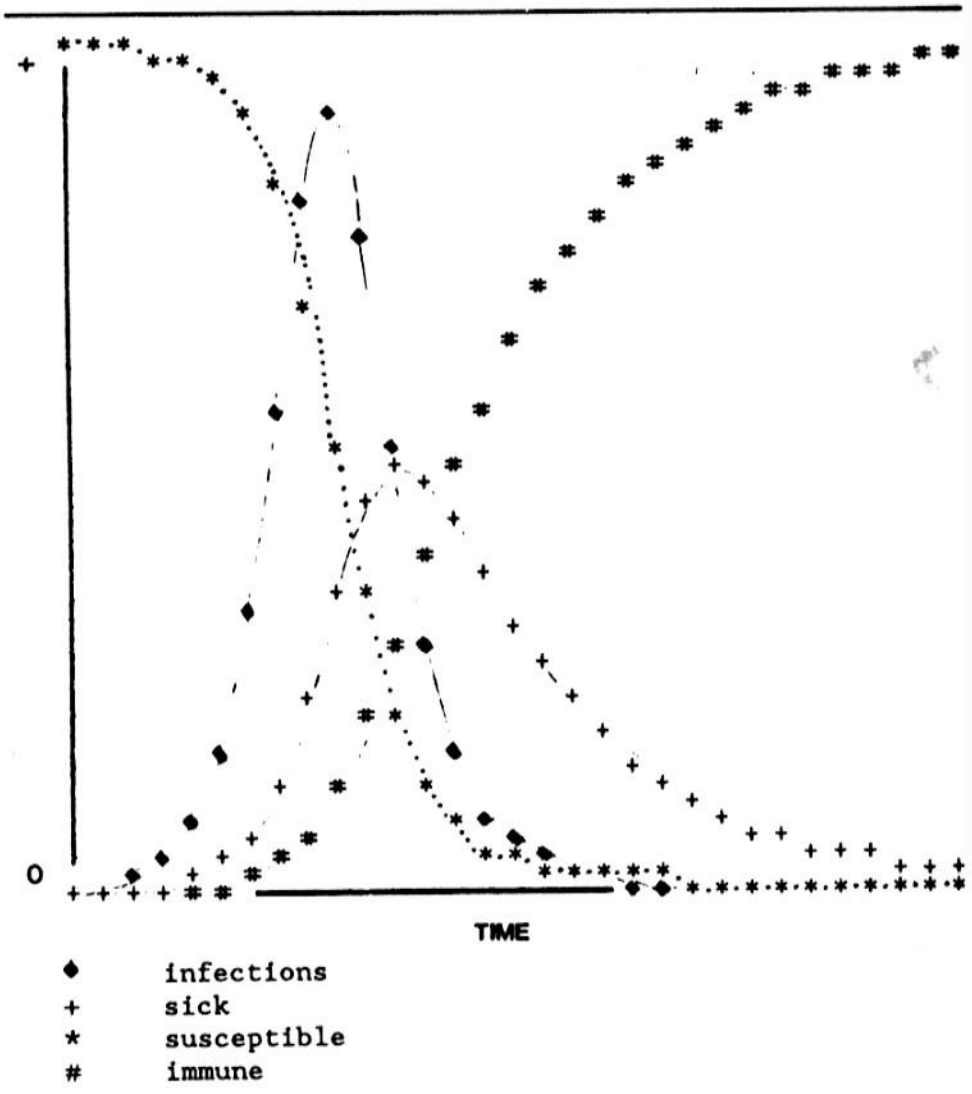


Figure 7.6: Influenza model.

Directions for Development

Modeled heterogeneity

All of the models of diffusion and contagion that we have discussed in this chapter have made quite simple assumptions about the propensities of individuals to undergo or to resist transitions. In many of the models we have assumed that all of the individuals in the population are homogeneous in this regard and that which individuals make transitions is due to purely random processes. In other models we have been

somewhat more sophisticated, assuming that some individuals are more difficult to convert than others, leading to population heterogeneity in resistance to change.

In many circumstances, models such as those that we have developed here are perfectly adequate to represent the phenomena of interest. This is particularly the case where the interest of the theorist focuses on the behavior of the aggregate as a whole, and not on the status of the individuals making up that aggregate. In some cases the models that we have developed here are good representations because it is reasonable to treat all of the individuals in the population as essentially homogeneous with regard to their propensities to respond to a given stimulus. But sometimes it is not.

Suppose that we were interested in the problem of the diffusion of a new seed variety among the peasants of a village. The simplest theory of the dynamics of this situation might assume that the peasants were homogeneous with regard to their propensities to adopt the new seed. Under this theory, the pattern of diffusion would depend simply on the time shape of the stimulus and the homogeneous propensity to change. Becoming somewhat more sophisticated, we might propose that the peasants are not identical, but are normally distributed around some mean propensity. The general pattern of diffusions occurring under this assumption would be the same as in the simpler model, but each particular diffusion history might appear rather different from the ideal type.

But peasants are not homogeneous in their propensity to adopt innovations. Nor can the differences among them be captured very well by assuming a normal distribution of propensities. Poor peasants in particular, who might benefit most from the adoption of the new seed, are often least likely to adopt it. They can least afford the consequences if the new technology does not live up to expectations. Models of the diffusion process that do not take this important form of "population heterogeneity" into account are not likely to be very adequate representations of the dynamics of diffusion of agricultural innovations.

There are several approaches to dealing with the complexity raised by heterogeneity in the population. In some cases the effect of the heterogeneity can be directly modeled as a simple function. This is the approach that we used when we supposed that the distribution of resistance to change was a negative exponential or cumulative normal function. In the case of the diffusion of agricultural innovation we might proceed by supposing that resistances to change were distributed in the population of interest as a simple function of the distribution of wealth in the population. In this way, information about the effects of

"independent variables" (such as the distribution of wealth) can be added to the basic diffusion model.

The heterogeneity in the population may be qualitative, as well as quantitative. Let us suppose that not only wealth, but also ethnicity or religious beliefs affect the propensity to adopt new innovations. To capture these effects it might be necessary to divide the population into separate subpopulations and view the diffusion as a series of parallel processes occurring simultaneously within the several groups.

There are obviously limits on how far one might wish to go in modeling population heterogeneity. At some point, and for some purposes, a completely different approach to systematic theorizing and analysis of diffusion might be far more appropriate than the types of models that we have discussed in this chapter. In many cases for studying diffusion dynamics particularly, discrete-state and social-network models may provide better tools. Discrete-state modeling languages allow one to associate variable characteristics with each actor in the population and hence create models that describe situations in which the population in completely heterogeneous with regard to characteristics that affect probabilities of transitions. Each peasant, to continue our illustration, might be characterized by his or her religion, ethnicity, age, income, and other characteristics. Such models are particularly useful if the interest of the investigator is at the level of individual rather than aggregate behavior, and, of course, where high levels of relevant heterogeneity force such a choice for realism.

Social network models of phenomena like diffusion take the insights of general discrete-state models and push them much further. In social-network models, the probabilities of transitions for individuals depend not only upon the characteristics of the individuals, but also on the network of relations among individuals. For example, the probability that an particular actor will be subjected to a stimulus in a particular period of time can be seen as dependent on how many other individuals the actor has contact with (where the stimulus is spread by self-referencing growth). In the aggregate, of course, such "connectedness" is captured by assumptions about the average contact rates. Where individuals are heterogeneous, however, such average rates of connectedness are of little help in predicting dynamics. In network models, individual resistance to change may also be seen as a consequence of social position, as well as individual characteristics. In network models, individuals may reference others as well as themselves in making decisions about whether to adopt or not to adopt an innovation. Again, the network approach may be more appealing than the continuous-state approach where interest focuses on individual transition probabilities,

or when heterogeneity in distribution of network positions is so high as to require it.

Continuous States and Aggregates.

All of the examples of one-way transition processes in this chapter have been drawn from the study of diffusion and other very similar processes. Consequently, the discussion has been in terms of system states that are aggregates of actors or proportions of populations of actors. Because we have been using these particular examples, much of the discussion has used terminology that implies discrete states and qualitative transitions; for example, individuals make transitions from being nonknowers to being knowers.

It is important to emphasize that the theories and models that have been developed here deal with continuous states. Many of the models that have been used to describe diffusion and contagion processes might serve equally well as baselines for thinking about change in such things as individual's attitudes, levels of economic production, and other "continuous" variables. For example, the dynamics of the intensity of a person's confidence in a group leader could be approached as a simple growth process. The rate of change in confidence might be seen to depend on exogenous events (e.g., the leader's perceived performance), be partially self generating (perhaps by selective perception and ego defensive attribution), and partially self-limiting (perhaps as a consequence of the upper limits on the total emotional intensity possible for a given individual). Similarly, the process of economic production could be approached as a simple "transition" model in which raw materials make transitions to finished goods which make further transitions, with delay, to become waste.

Theories that see emotional intensity and economic production as relatively simple chains of transitions governed by exogenous, self-referencing, and goal-referencing feedback may or may not be useful ways of thinking about the dynamics of these phenomena. And the ways that we think about the dynamics of diffusion and contagion as involving "contact rates," "tellings," "immunity," "resistance to change," and so on do not apply to theorizing about the dynamics of belief or material production. Our point is simply that the models developed in previous sections with specific reference to diffusion have similar structures as systems to models of other continuous state dynamics. Economic production is not the same thing as the spread of a rumor, but it is possible to theorize about the dynamics of the two processes using systems that have very similar structures, and hence similar forms of possible dynamic behavior.

Conclusions

Many dynamic processes of interest to theorists can be usefully thought of as relatively simple one-way transitions or processes. Such macrosocial phenomena as diffusion, contagion, growth, and decline can all be captured by models with relatively few "states." Microlevel parallels involving one-way transitions among "qualitative" states or monotonic change in "quantitative" states are also easy to imagine. We have by no means exhausted the range of possible applications with the examples in this chapter. We have, however, provided an introduction to some of the most commonly occurring types of systems models of this type.

Our discussion has been in terms of the increasing complexity of models with few states as they move from having "simple" control structures to having more "complex" ones. "Simple" systems that respond only to external stimuli can produce a very wide variety of behavior, depending on the nature of the stimulus, and on the complexity of the response. It is useful to think of almost all simple systems representations of the dynamics of social action as being "open" in the sense that they respond to exogenous stimuli. Theorizing about what external factors affect rates of transition and growth is always a useful first step in approaching problems such as those that we have dealt with here.

In many ways, though, the more interesting aspects of social dynamics are produced by more complicated aspects of control structures that are self-referencing and goal-referencing. Models of diffusion are particularly useful as illustrations because the ways in which self-referencing and goal-referencing control is occurring are quite apparent. As we suggested in the previous chapter, models with these more complex forms of control structures have more complicated characteristic dynamics and are capable of far more varied behavior. Such more complex control structures are quite common in social phenomena, and the theorist should search for them when thinking about problems of this type.

Two additional lessons should be taken from the examples in this chapter: (1) the same phenomenon can be usefully conceptualized and analyzed in a variety of ways depending on the purpose of the investigation, and (2) quite different theories can produce outcomes that as so similar that they are very difficult to distinguish.

As an illustration of the first point, consider the discussions of how to deal with population heterogeneity in diffusion models. For some purposes such heterogeneity can be simply ignored or treated as noise, as in trying to understand the effects of exogenous factors on differences

among similar (but each heterogeneous) populations. For other purposes, it may be preferable to deal with heterogeneity by means of assumptions about population distributions and by introducing independent variables into the model to deal with the most important forms. In still other cases a completely different form of modeling using discrete language might be called for to best represent theories in which the differences among actors, rather than their average similarities, are critical.

As an illustration of the second point, that different theories can produce similar results, recall our basic models and extensions. S-shaped "diffusion" curves can be produced by a variety of quite different processes. They may be the result of relatively complicated positive and negative feedback operating simultaneously on a homogeneous population, as we have developed in our models here. S-shaped curves, though, could also be produced by nonhomogeneous distributions of propensities of knowers to become tellers, or for actors to resist conversion. In the absence of an otherwise plausible theory, it is often impossible to distinguish such alternatives from looking at the time-traces that they produce. That quite different causal processes can produce very similar-looking outcomes should be a cause of concern to the theorist concerned with dynamics. It is not enough to be able to reproduce plausible behavior using models from one's theory; and, it is often very dangerous to place great weight in building a theory on reasoning backward from the realizations of dynamic processes to hypotheses about the processes themselves.

Notes

1. For some examples of the use of diffusion models in vaious social science applications, see Brown and Philliber (1977), Boulding (1956), Burmeister and Dobell (1970), Bush and Mosteller (1955), Chow (1967), Coleman et al. (1966), Davies (1969), Dixon (1980), Dodson and Muller (1978), Dunn (1971), Eyestone (1977), Gray (1973), Griliches (1957) (a classic), Hummon (1971), Katz and Hamilton (1963), Kelly and Kranzberg (eds., 1978), Oster (1982), Pitcher et al. (1978), Rapoport (1978), Rogers (1983), and Teece (1980).

2. Mathematical and statistical approaches to unidirectional transition models are very highly developed. Among the many excellent introductions to this literature are Bailey (1957), Bartholomew (1973), Coleman (1964a, 1968, 1981), Doreian and Hummon (1976), Hamblin et al. (1973), Kemeny and Snell (1962), Leik and Meeker (1975), Nielsen and Rosenfeld (1981), Mahajan and Peterson (1985), Meade (1984), and Monin et al. (1976).

3. Survival distribution analysis and event history analysis are variations on the same kinds of models that we are considering in this chapter, though such models can also deal with many more complex dynamics as well. See particularly Tuma and Hannan (1984) and Allison (1984).

4. On the partitioning of the population into those "at risk" and those "not at risk" ("movers and stayers") in social mobility analysis, see particularly Hout (1983), Leik and Meeker (1975), Spilerman (1972a, 1972b), Singer and Spilerman (1974), and White (1965).

5. Alternative assumptions about the distribution of mobilizability give rise to the various

parametric survival models. Arbitrary heterogenity with respect to time but proportional effects for individuals can also be assumed in statistical models by use of partial likelihood methods. See particularly Allison (1984) and Tuma and Hannan (1984).

6. If our observations were subject to sampling or measurement error (as they usually are), the problem of "inducing" the proper form for the underlying process from its realization becomes even more troublesome.

7. Indeed, an alternative formulation of this model could treat the number who have recovered as a simple "boxcar" delay of the number infected.

APPENDIX 7.1. Basic Diffusion Curve Models

```
* DIFFUSION, GROWTH, DECLINE PROCESSES
NOTE
NOTE FOUR MODELS ARE SHOWN WITH DIFFERING CONTROL
NOTE          STRUCTURES
NOTE
NOTE ********************************************************
NOTE MODEL ONE: LINEAR GROWTH TO A CEILING
NOTE LET "K" BE THE NUMBER OF PERSONS KNOWING,
NOTE LET "DK" BE THE NUMBER NOT KNOWING. POP = K + DK
L             K1.K = K1.J+(DT)(RI1.JK)
N             K1 =5
R             RI1.KL = CLIP(EXOG.K,0,DK1.K,0)
NOTE RATE OF INCREASE IS EQUAL TO EXOG IF DK1 IS GT ZERO
NOTE RATE OF INCREASE IS EQUAL TO ZERO IF DK1 IS ZERO
A             DK1.K = POP-K1.K
C             POP = 100
A             EXOG = CONST
C             CONST = 10
NOTE THE STIMULUS IS A CONSTANT 10 UNITS PER UNIT TIME
NOTE
NOTE ********************************************************
NOTE MODEL TWO: SELF-REFERENCING GROWTH TO A LIMIT
NOTE
NOTE One person knows initially, and all knowers become
NOTE tellers with an intensity of three-quarters tellings
NOTE to non-knowers per unit time.
NOTE
L             K2.K = K2.J+(DT)(RI2.JK)
N             K2 = 1
A             TELL1.K = (K2.K*.75)
R             RI2.KL = CLIP(TELL1.K,0,DK2.K,0)
A             DK2.K = POP-K2.K
NOTE
NOTE ********************************************************
NOTE MODEL THREE: CONSTANT STIMULUS, PROPORTIONAL RESPONSE
NOTE
NOTE In this model, telling occurs at a constant rate of 10
NOTE tellings per unit time. Success in telling (PS)
```

```
NOTE is proportional to the population not knowing.
L          K3.K = K3.J+(DT)(RI3.JK)
N          K3 = 5
A          TELL3.K = EXOG.K
A          PS3.K = DK3.K/POP
R          RI3.KL = (PS3.K)(TELL3.K)
A          DK3.K = POP-K3.K
NOTE
NOTE ********************************************************
NOTE MODEL FOUR: PROPORTIONAL STIMULUS AND PROPORTIONAL
NOTE RESPONSE (I.E., MODEL 2 AND 3 COMBINED)
NOTE
L          K4.K = K4.J+(DT)(RI4.KL)
N          K4 = 1
A          DK4.K = POP-K4.K
A          TELL4.K = K4.K*.75
A          PS4.K = DK4.K/POP
R          RI4.KL = (PS4.K)(TELL4.K)
NOTE
NOTE ********************************************************
NOTE OUTPUT SPECIFICATIONS
NOTE
SPEC       DT = .25/LENGTH = 15/PLTPER = 1
PLOT       K1 = 1, K2 = 2, K3 = 3, K4 = 4(0,100)
RUN
```

APPENDIX 7.2. Self-Referencing Growth Variations

```
* SELF-REFERENCING GROWTH VARIATIONS
NOTE
NOTE PROGRAMS USED TO GENERATE FIGURE 7.3
NOTE
NOTE ********************************************************
NOTE MODEL ONE:        Knowers become tellers at a rate of
NOTE                   one-half telling per unit time, but do so
NOTE                   with a first-order delay of average length
NOTE                   of three time units. All tellings are
NOTE                   successful if there are any DKs available
L          K1.K = K1.J+(DT)(RI1.JK)
N          K1 = 1
A          TELL1.K = (.5)(DELAY1(K1.K,3))
A          PS1.K = CLIP(1,0,DK1.K,0)
C          POP = 100
A          DK1.K = POP-K1.K
R          RI1.KL = TELL1.K*PS1.K
NOTE
NOTE ********************************************************
NOTE MODEL TWO:        As in model one, but now knowers become
```

```
NOTE                    tellers with a third-order delay instead
NOTE                    of a first-order delay.
L          K2.K = K2.J+(DT)(RI2.JK)
N          K2 = 1
A          TELL2.K = (.5)(DELAY3(K2.K,3))
A          PS2.K = CLIP(1,0,DK2.K,0)
A          DK2.K = POP-K2.K
R          RI2.KL = TELL2.K*PS2.K
NOTE
NOTE ********************************************************
NOTE MODEL THREE:       As a baseline, the same model is created,
NOTE                    but with no delays in knowers becoming
NOTE                    tellers.
L           K3.K = K3.J+(DT)(RI3.JK)
N          K3 = 1
A          TELL3.K = (.5)(K3.K)
A          PS3.K = CLIP(1,0,DK3.K,0)
A          DK3 = POP-K3.K
R          RI3.KL = TELL3.K*PS3.K
NOTE
NOTE OUTPUT SPECIFICATIONS
SPEC       DT = .1/LENGTH = 20/PLTPER = 1
PLOT       K1 = *, K2 = +, K3 = #(0,140)
RUN
```

APPENDIX 7.3. Diffusion and Population Heterogeneity

```
* DIFFUSION WITH POPULATION HETEROGENEITY
NOTE THREE MODELS OF DIFFUSION FOR FIGURE 7.4. MODELS ASSUME
NOTE FLAT (LINEAR) DISTRIBUTION OF RESPONSIVENESS TO STIMULI
NOTE POISSON-DISTRIBUTED RESPONSIVENESS, AND CUMULATIVE
NOTE NORMALLY DISTRIBUTED RESPONSIVENESS
NOTE
NOTE ********************************************************
NOTE MODEL 1:           Assumes that the population is homogeneous,
NOTE                    i.e., all members of the population have equal
NOTE                    probability of responding to a stimulus
L          K1.K = K1.J+(DT)(RI1.JK)
N          K1 = 1
A          TELL1.K = TPARM*K1.K
C          TPARM = .5
A          PS1.K = DK.K/POP
C          POP = 100
A          DK1.K = POP-K1.K
R          RI1.KL = (TELL1.K)(PS1.K)
NOTE
```

```
NOTE MODEL 2:          In this model, it is assumed that most of
NOTE                   the population has low resistance, with
NOTE                   smaller and smaller proportions having
NOTE                   greater resistance, leading to a 'Poisson'
NOTE                   distribution of resistances.
L          K2.K = K2.J+(DT)(RI2.JK)
N          K2 = 1
A          TELL2.K = (TPARM)(K2.K)
A          DK2.K = POP-K2.K
A          PS2.K = TABLE(POIS,DK2,K,0,100,10)
T          POIS = 0/.25/.45/.575/.675/.75/.825/.875/.925/.975/1.0
R          RI2.KL = (TELL2.K)(PS2.K)
NOTE
NOTE ********************************************************
NOTE MODEL 3:          In this model, we use the normal distribution
NOTE                   i.e., relatively few people have either very
NOTE                   low or very high resistance, most falling
NOTE                   around a mean.
L          K3.K = K3.J+(DT)(RI3.JK)
N          K3 = 1
A          TELL3.K = (TPARM)(K3.K)
A          DK3.K = POP-K3.K
A          PS3.K = TABLE(CNORM,DK3.K,0,100,10)
T          CNORM = 0/.25/.38/.45/.475/.50/.525/.55/.62/.75/1
R          RI3.KL = (TELL3.K)(PS3.K)
NOTE
NOTE ********************************************************
NOTE OUTPUT SPECIFICATIONS
NOTE
SPEC       DT = .1/LENGTH = 20/PLTPER = 1
PLOT       K1 = *, K2 = +, K3 = #(0,100)
RUN
```

APPENDIX 7.4. Influenza Epidemic Model*

```
* INFLUENZA MODEL
NOTE From Pugh-Roberts associates (1982) with modification by the author
NOTE
NOTE Total Population
N          TOTAL = SUSC+SICK+IMM
NOTE
NOTE Susceptible Population
L          SUSC.K = SUSC.J+(DT)(-INFEC.JK)
N          SUSC = SUSCI
C          SUSCI = 999
NOTE
NOTE Sick Population
```

```
L           SICK.K = SICK.J+(DT)(INFEC.JK-RECOV.JK)
N           SICK = SICKI
C           SICKI = 1
NOTE
NOTE Immune Population
L           IMM.K = IMM.J+(DT)(RECOV.JK)
N           IMM = IMMI
C           IMMI = 0
NOTE
NOTE Infection Rate
R           INFEC.KL = PRCON*CONTAC.K
C           PRCON = .2
A           CONTAC.K = AVCON*(SUSC.K/TOTAL)*SICK.K
C           AVCON = 5
NOTE
NOTE Recovery Rate
R           RECOV.KL = SICK.K/RECOVT
C           RECOVT = 5
NOTE
NOTE ***************************************************
NOTE OUTPUT SPECIFICATIONS
SPEC        DT = .25/PLTPER = 1/LENGTH = 30
PLOT        SUSC = X, SICK = S, IMM = Y, INFEC = I
PLOT        INFEC = I, RECOV = R
RUN
```