Spatial and Temporal Heterogeneity in Diffusion[1]

David Strang and Nancy Brandon Tuma
Cornell University

Standard models of diffusion assume spatial and temporal homogeneity. This article develops a class of diffusion models that incorporate spatial and temporal heterogeneity by turning to the individual level within an event-history framework. These models permit the analyst to represent social structural relations thought to channel diffusion, and to model decay in the influence of events over time. Heterogeneous diffusion models are applied to a reanalysis of data reported in Coleman, Katz, and Menzel's classic diffusion study. Network centrality and local structures of influence based on cohesive relations and structural equivalence are all shown to channel the diffusion of tetracycline.

A variety of phenomena of interest to social scientists involve the diffusion of some trait or characteristic through a population. Examples include spread of a disease, adoption of an innovation, and acquisition of a skill (for a general review, see Rogers [1983]). Social scientists often think of such processes as driven not only by the atomistic behavior of adopters, but as involving processes like *contagion*—contact between members of the population who have and have not yet adopted. Such contact involves some form of meaningful communication and influence, grounded in social relations ranging from face-to-face interaction to highly constructed forms of perceived similarity (Burt 1987; Strang and Meyer 1993).

To learn about social interaction and social structure, however, we need to move beyond standard diffusion models, which assume spatial and temporal homogeneity. Spatial homogeneity means that all members of the population have the same chance of affecting and being affected by each other. Temporal homogeneity means that the potential influence of prior adoption events does not vary with the length of time since their occurrence. These assumptions make the mathematics of diffusion relatively tractable. But they also render diffusion analyses uninformed by, and uninformative about, the social structure of the population under study.

A number of diffusion models relaxing the assumptions of spatial and temporal homogeneity have been proposed (e.g., see the review in Bartholomew [1982]).[2] Hernes (1972) and Diekmann (1989) consider variations on classic diffusion processes that provide plausible representations of population marriage rates. But population-level diffusion models are difficult to extend in more concrete ways. Faced with specific social structures, analysts generally replace stochastic with deterministic formulations and simulate diffusion in simplified contexts rather than model complex empirical processes.

In this article we develop individual-level models of diffusion that allow heterogeneity both within the population and over time. (We use the term "individual" to stand for the adopter, which may be a person, organization, or other social actor.) Such models can be estimated from event-history data—data on the times of adoptions by individual members of the population.[3] We propose models in which adoption by an individual is a function of prior adoption events by other members of the population. In contrast to standard diffusion models, the population-level process of spread is not explicitly modeled. Instead, we formulate explicit models of the individual-level dynamics, which reduce to the standard population-level model when spatial and temporal homogeneity holds. By means of Monte Carlo simulations, one could use these models to forecast population-level dynamics, assuming the distribution of exogenous explanatory variables is known.

The shift to the microlevel opens up an exciting array of possible extensions to standard diffusion formulations. Building on Marsden and Podolny's (1990) and Strang's (1991*a*) application of standard event-history

[1] Previous versions of this article were presented at the 1990 annual meetings of the Population Association of America, the 1990 World Congress of Sociology, and the 1992 annual meetings of the American Sociological Association. We thank David Pasta not only for helpful comments and superior programming assistance but for helping to clarify some modeling issues, and Ronald Breiger, Arthur Stinchcombe, and Tony Tam for their helpful comments on this work. We especially thank Ronald Burt, Peter Marsden, and Joel Podolny for generously making data available from prior reanalyses of the Coleman et al. study. Research support was provided by the National Institutes of Child Health and Human Development grant HD 21738 and the National Science Foundation grants SES 8911666, SES 9213152, and SES 9213258. Partial support for an earlier draft was also provided to Tuma by the Hoover Institution on War, Revolution, and Peace. Correspondence should be directed to David Strang, Department of Sociology, Cornell University, Ithaca, New York 14853.

0002-9602/94/9903-0003$01.50

[2] See also Granovetter (1978), who reframes diffusion around the notion that individuals respond to the number of prior adopters rather than to contact with prior adopters.

[3] More generally, one could have data on times of adoptions and the onset of contagion, which need not be the same events.

methods to the study of diffusion, we suggest classes of models that capture both spatial and temporal heterogeneity. We present results of Monte Carlo simulation studies of the properties of maximum-likelihood estimators of some of these new models. Finally, we illustrate our approach with an application of these new models to Coleman, Katz, and Menzel's (1966) classic study, *Medical Innovation*.[4]

MODELS OF DIFFUSION

Population-Level Models

We begin with models of diffusion at the population level. For a population of size I, we may describe the time path of adoption in terms of $N(t)$, the number who have not yet adopted at time t, or equivalently, by $S(t) = I - N(t)$, the number who have adopted by time t and are now spreading it to those still at risk of adoption. Later we also make use of two related concepts, $\mathcal{N}(t)$ and $\mathcal{S}(t)$, the set of cases at time t that have not adopted and those that are spreading, respectively. In standard diffusion models, these two sets provide an exhaustive and mutually exclusive partition of the population. One can also imagine extensions of standard diffusion models in which these two sets may be defined in alternative ways, as we discuss further below.

The standard population-level formulation of a diffusion process is (see, e.g., Bailey 1976; Bartholomew 1982):

$$\lim_{\Delta t \downarrow 0} \frac{\text{prob}[N(t + \Delta t) = n - 1 \,|\, N(t) = n]}{\Delta t} = \lim_{\Delta t \downarrow 0} \frac{\text{prob}[S(t + \Delta t) = s + 1 \,|\, S(t) = s]}{\Delta t} = [a + bs(t)]n(t). \tag{1}$$

To explicate the logic underlying equation (1), we decompose its right-hand side into diffusion influences originating outside and inside the population. The impact of factors outside the population (often called an "external source") is proportional to the number of individuals at risk of adoption, $n(t)$; the effect of this influence is a. The impact of contacts or linkages within the population is proportional to the number of spreaders $s(t)$ multiplied by the number of individuals at risk $n(t)$; the effect of this impact is b. Thus, the contribution to the diffusion process from outside the population is $a\ n(t)$, and the contribution from inside the population is $bn(t)s(t)$.[5]

[4] Burt (1987) reanalyzes the Coleman et al. study; Marsden and Podolny (1990) reanalyze Burt. We reanalyze both reanalyses.

[5] Of course, in modeling the spread of certain diseases, such as smallpox, it is appropriate to assume that there is only contagion and no external source (or intrinsic tendency to adopt). Models for processes of this type would suppress a.

This model embodies some unrealistic assumptions. It implies that all members of the population are equally susceptible to the external factors. It implies that contacts between all pairs consisting of a spreader and a potential adopter are equally likely and equally contagious. And it implies that the rate and contagiousness of contacts do not vary with time.

Population-level models also involve a less obvious but important limitation. There is no straightforward way to introduce differences across individuals in their intrinsic tendency to adopt in models like equation (1), precisely because they are framed at the level of the population. This divorces diffusion analysis from the larger regression tradition that focuses on internal determinants of behavior. The study of diffusion becomes an alternative or supplement to standard individual-level analysis.

Though one can build population-level heterogeneity and temporal inhomogeneity (e.g., time trends) into population-level models like (1), such extensions are difficult. In fact, an analytical solution for even the general homogeneous mixing formulation in (1) is not available (see Bartholomew 1982, pp. 255–59). We believe that progress can be made by shifting to the level of the individual population member. This strategy simplifies the mathematics, turning a nonlinear differential equation into a more manageable model about the dynamics of individual behavior.

One individual-level analytic approach is to model the time of adoption using a linear regression framework with spatial effects (Doreian 1981). However, such models are designed to handle mutual instantaneous relations between cases where outcomes are measured at a single point in time. They are not suited to temporal processes because use of the timing of other adoptions as regressors permits later events to influence earlier ones (for an example, see Burt's [1987] construction of "adoption norms"). In addition, a linear regression framework does not deal naturally with the right censoring generally present in longitudinal data on change in discrete outcomes. This is made particularly problematic by the frequent presence of important time-varying explanatory factors, which also cannot be straightforwardly accommodated within cross-sectional linear regression models.

It is more natural to develop individual-level models of diffusion within an event-history framework (Tuma and Hannan 1984). At the *micro*-level, let $Y_i(t)$ be a binary variable which equals one if individual i has adopted by time t and zero otherwise. It is useful to model the hazard rate of adoption,

$$r_i(t) = \lim_{\Delta t \downarrow 0} \frac{\text{prob}[Y_i(t + \Delta t) = 1 \,|\, Y_i(t) = 0]}{\Delta t}, \tag{2}$$

where $r_i(t)$ gives the limiting probability that an individual case i adopts the diffusing trait between t and $t + \Delta t$ per unit of time, given that i did not adopt before t.

To translate the population-level diffusion equation into an analogous equation at the microlevel, we can unpack (1) into two components: the number of individuals at risk of adoption and the hazard rate of adoption for each individual. By definition the number of individuals at risk is $n(t)$. The individual hazard rate of adoption corresponding to equation (1) is

$$r_n(t) = a + bs(t) = a + \sum_{s \in \mathcal{S}(t)} b, \qquad n \in \mathcal{N}(t). \tag{3}$$

The hazard rate is subscripted by n to emphasize that it applies to those individuals who have not yet adopted, that is, those in the set $\mathcal{N}(t)$. (For simplicity, we suppress the explicit reminder that the rate applies only to members of $\mathcal{N}(t)$ below.) Thus, equation (1) implies that there are $n(t)$ hazard rates like equation (3).

We write the set of influencing events very generally as membership in $\mathcal{S}(t)$ to emphasize that defining the set of possible influences is a theoretical task. A homogeneous mixing model assumes that every adoption prior to t is potentially influential. But theories of social structure suggest more interesting ways to think about patterns of influence. These include ideas about communication rates, reference groups, relational or structural equivalence, and isomorphic cultural identities. In addition, study designs may suggest obvious restrictions on the linkages thought to underlie $\mathcal{S}(t)$. In *Medical Innovation* (Coleman et al. 1966), for example, doctors are assumed to respond to other doctors working in the same city, but not to ones in other cities.

The identification of $\mathcal{S}(t)$ involves assumptions about temporal effects as well as spatial relations. In many contexts, researchers may require simple temporal ordering as a prerequisite of influence: $\mathcal{S}(t)$ contains all spatially relevant individuals adopting before t. But in other contexts, researchers may wish to permit instantaneous influences. Here $\mathcal{S}(t)$ also contains spatially relevant individuals having events at t. Situations in which the latter would be appropriate include settings where adoptions are jointly planned and explicitly coordinated. Instantaneous influence might also be allowed where measurement techniques produce artifactually equivalent adoption times, though here no simple assumption about influence is without defects.

When explanatory variables are introduced in models of hazard rates, it is conventional to exponentiate the right-hand side of the model (or, equivalently, to model the logarithm of the rate) to ensure that hazard rates are nonnegative. This suggests two plausible variations on equation

(3). A *multiplicative* diffusion model treats individual tendencies and contagious influences as multiplying each other:

$$r_n(t) = \exp\left(a + \sum_{s \in \mathcal{S}(t)} b\right). \tag{4a}$$

An *additive* diffusion model sums separately multiplicative functions of individual tendencies and contagious influences to form the overall hazard rate:

$$r_n(t) = \exp(a) + \sum_{s \in \mathcal{S}(t)} \exp(b). \tag{4b}$$

We consider the advantages of equations (4a) and (4b) below after indicating more specifically how spatial and temporal heterogeneity can be introduced.

Incorporating Heterogeneity

Rewriting the population-level model of diffusion at the individual level suggests that the parameter a can be treated as a function of measured characteristics of i, $\mathbf{x}_i$. The tradition of regarding a as the effect of an external signal can be usefully extended: individuals may be differentially affected by common environmental influences. But we suspect that it is often more useful to regard the elements of a as measuring an individual's "intrinsic" rate of adoption, which is distinct from the intrapopulation diffusion process. Below we employ the second interpretation, which is consistent with standard usage in event-history models.

In recent work, Marsden and Podolny (1990) and Strang (1991*a*) have modeled heterogeneity in diffusion using standard event-history formulations and estimation methods. For spatial heterogeneity this can easily be accomplished via the multiplicative model in (4a), although the resulting equation does not reduce to the classical diffusion model in (1) when there is spatial homogeneity. Both articles focus on variations in the social proximity of n and s. They develop models of the form

$$r_n(t) = \exp\left(\boldsymbol{\alpha}'\mathbf{x}_n + \sum_{s \in \mathcal{S}(t)} \boldsymbol{\delta}'\mathbf{z}_{ns}\right), \tag{5}$$

where $\mathbf{z}_{ns}$ represents a vector of social proximity measures defined on n and s.[6] Marsden and Podolny consider weighting schemes based on network relations (direct contacts vs. structural equivalence) in a reanalysis

[6] Here and below we assume that all covariates are potentially time varying.

of Coleman et al.'s (1966) classic *Medical Innovation*. Strang (1990, 1991*b*) considers the diffusion of decolonization within different partitions (empires vs. regions) of the population of colonial dependencies.[7]

Models like (5) may be further elaborated in conventional fashion to include parametric or nonparametric dependence on various measures of time. For example, Marsden and Podolny (1990) assume that the impact of individual propensities and intrapopulation influences is multiplied by an unknown function of calendar time, $q(t)$, and estimate this model using Cox's (1972, 1975) method of partial likelihood. Similarly, Strang's work on decolonization assumes parameterized dependence on historical time.

Adding temporal heterogeneity in diffusion to standard event-history models is not so straightforward. Strang (1991*a*) suggests that one may develop models involving dependence on time since the most recent event (t_e) in the population:

$$r_n(t) = \exp(\boldsymbol{\alpha}'\mathbf{x}_n) + \exp(\boldsymbol{\beta}'\mathbf{v}_n + \gamma t_e). \tag{6}$$

But this approach (known as a Makeham model in the literature) is very restrictive. It ignores the impact of events prior to the most recent one, and does not marry well with the study of spatial heterogeneity.[8] A general conclusion is that it is especially difficult to permit both spatial and temporal heterogeneity in diffusion within the framework of conventional hazard rate models.

It seems useful, then, to develop more general models of the way prior events influence the rate of adoption. Such models may include four kinds of terms: the susceptibility of some potential adopter n to diffusion, the infectiousness of some spreader s, the proximity of pairs consisting of one spreader s and one potential adopter n, and temporal variation as a function of time since adoption by spreaders.

We define $\mathbf{x}_n$ as a vector of variables describing n's intrinsic rate of adoption (i.e., ignoring intrapopulation linkages); $\mathbf{v}_n$ as a vector of variables describing n's susceptibility to intrapopulation linkages; $\mathbf{w}_s$ as a vector of variables describing the infectiousness of s; $\mathbf{z}_{ns}$ as a vector of variables describing the social proximity of n and s;[9] and t_s as the time

[7] Other empirical analyses of contagion within an event-history framework include studies of Progressive Era municipal and civil service reform by Knoke (1982) and by Tolbert and Zucker (1983).

[8] The t_e may be replaced by t_{ne}: time since the last event by a population member to which n is connected. This approach is feasible only if social proximity can be measured as a binary relation. And since t_{ne} is undefined until the first adoption event by a connected member of the population, this approach may work well only when the population can be partitioned into a few large groups.

[9] Social proximity may be regarded as an inverse of social distance. Sometimes it may be easier to theorize about or to measure social distance rather than social proximity.

that s starts spreading, which is usually (but need not be) assumed to equal the time s adopts. We assume that the first elements in $\mathbf{x}$ and $\mathbf{v}$ are unity: these permit distinct intercepts for intrinsic propensities to adopt and for diffusive influences from members of $\mathcal{S}(t)$. Note that the latter term can be regarded as representing a combination of average susceptibility, infectiousness, and social proximity: we locate this effect in $\mathbf{v}$ by convention. Only one intercept is identified for the three vectors $\mathbf{v}$, $\mathbf{w}$, and $\mathbf{z}$.

If we assume no time dependence and no temporal heterogeneity in diffusion, a formulation of equation (3) within an additive framework is

$$\begin{aligned} r_n(t) &= \exp(\boldsymbol{\alpha}'\mathbf{x}_n) + \sum_{s\in\mathcal{S}(t)} \exp(\boldsymbol{\beta}'\mathbf{v}_n + \boldsymbol{\gamma}'\mathbf{w}_s + \boldsymbol{\delta}'\mathbf{z}_{ns}) \\ &= \exp(\boldsymbol{\alpha}'\mathbf{x}_n) + \exp(\boldsymbol{\beta}'\mathbf{v}_n) \sum_{s\in\mathcal{S}(t)} \exp(\boldsymbol{\gamma}'\mathbf{w}_s + \boldsymbol{\delta}'\mathbf{z}_{ns}), \end{aligned} \tag{7a}$$

where $\boldsymbol{\alpha}$, $\boldsymbol{\beta}$, $\boldsymbol{\gamma}$, and $\boldsymbol{\delta}$ are vectors of parameters giving the effects of variables in the vectors $\mathbf{x}_n$, $\mathbf{v}_n$, $\mathbf{w}_s$, and $\mathbf{z}_{ns}$, respectively.

Similarly, in the absence of temporal heterogeneity in diffusion, a formulation of equation (3) within a multiplicative framework is

$$\begin{aligned} r_n(t) &= \exp\left(\boldsymbol{\alpha}'\mathbf{x}_n + \sum_{s\in\mathcal{S}(t)} \boldsymbol{\beta}'\mathbf{v}_n + \boldsymbol{\gamma}'\mathbf{w}_s + \boldsymbol{\delta}'\mathbf{z}_{ns}\right) \\ &= \exp\left(\boldsymbol{\alpha}'\mathbf{x}_n + \sum_{s\in\mathcal{S}(t)} \boldsymbol{\beta}'\mathbf{v}_n + \sum_{s\in\mathcal{S}(t)} \boldsymbol{\gamma}'\mathbf{w}_s + \sum_{s\in\mathcal{S}(t)} \boldsymbol{\delta}'\mathbf{z}_{ns}\right). \end{aligned} \tag{7b}$$

When spatial but not temporal heterogeneity is assumed, the trade-off between the additive and multiplicative formulations seems to be primarily a matter of their substantive appropriateness. The additive formulation is more faithful to the standard population-level model in equation (1) in that parameters like α and β can be translated into a and b (e.g., $\alpha = \ln a$), whereas parameters from the multiplicative formulation cannot. But this does not imply empirical superiority.

However, an additive formulation better accommodates the introduction of temporal heterogeneity. For example, consider a simple model in which the impact of each prior event falls off exponentially with the length of time since its occurrence. (There is in fact some empirical evidence that exponential decline in salience is often a useful model; see Zielske and Henry [1980].) It is straightforward to allow exponential decline in the additive model:

$$r_n(t) = \exp(\boldsymbol{\alpha}'\mathbf{x}_n) + \exp(\boldsymbol{\beta}'\mathbf{v}_n) \sum_{s\in\mathcal{S}(t)} \exp[\boldsymbol{\gamma}'\mathbf{w}_s + \boldsymbol{\delta}'\mathbf{z}_{ns} + \zeta(t - t_s)], \tag{8}$$

where t_s is the adoption time of the sth member of $\mathcal{S}(t)$. Other forms of variation with the time since previous events can also easily be incorporated into the additive formulation. For example, one can replace $(t - t_s)$ with some other function $g(t, t_s)$ in equation (8). A further extension would let ζ be a function of covariates describing n, s, and their linkages.

A multiplicative framework has difficulty in accommodating the assumption that the impact of each event falls off since its occurrence. If we multiply (7b) by $\exp[\zeta \Sigma_{s\in\mathcal{S}(t)}(t - t_s)]$ in a manner analogous to equation (8), problems arise because $\Sigma_{s\in\mathcal{S}(t)}(t - t_s)$ increases with both the number of prior events and with their remoteness in time—yet we expect the hazard rate to rise with the number of prior events but to decline with their remoteness in time. For example, as any event grows distant in time, $\Sigma_{s\in\mathcal{S}(t)}(t - t_s)$ becomes very large. It seems difficult to adjust for this flaw working from the multiplicative formulation in (7b).

ESTIMATION

The models presented above can be estimated by the method of maximum likelihood. Usually one maximizes the logarithm of the likelihood, rather than the likelihood, which for right-censored data on adoption dates (Tuma and Hannan, 1984, p. 126) is

$$\log \mathcal{L} = \sum_{n=1}^{N} d_n \log r_n(t) + \log G_n(t|t_0), \tag{9}$$

where d_n is an indicator variable equaling one if the case adopts and $G_n(t|t_0)$ gives the probability that n has not adopted by time t for a process starting at time t_0.

In general, maximum-likelihood estimators have good properties in large samples. They are asymptotically normal, unbiased, and consistent. Tuma and Hannan (1984, chap. 5) demonstrate that these large sample properties translate well in event-history analyses with independent random samples in the sizes usually available to sociologists (i.e., at least a few hundred cases). However, the heterogeneous diffusion framework described above involves interdependence in outcomes of a type not previously investigated, to our knowledge. Moreover, diffusion studies involving contagion are typically based on fairly small populations rather than on large samples.[10] They thus pose fresh questions of estimator bias and efficiency. For this reason, we performed a Monte Carlo study to

[10] In general, within a diffusion context we sample populations from a universe of populations.

examine the properties of maximum-likelihood estimation of event history models allowing diffusive influences.

We simulated event times according to some simple versions of equation (7a).[11] For each set of parameter values, we generated 100 data sets, each consisting of 100 cases. Details of the simulation procedure are available from the authors. We avoided parameter combinations that caused nearly all events to occur within a very short interval and therefore caused estimates to have high variance. Simulated data sets were analyzed using a version of RATE (Tuma 1980) that was modified to permit estimation of models like equations (7a) and (8).[12]

Table 1 describes the performance of maximum-likelihood estimators for two simulation studies. The first study examines a model including two intercepts, one for an intrinsic tendency to adopt and the second for contagion due to all prior adoptions. This model corresponds to the standard population-level diffusion model in (3). In addition to the true values used to construct the simulated data, the table reports the mean, the standard deviation, and the mean estimated standard error of each parameter estimate.

The results indicate that individual-level maximum-likelihood estimation can capture the properties of simple diffusion processes. The estimates have averages close to their true values and exhibit little variance. At least in this simple case, samples of quite modest size permit the detection of intrapopulation contagion. (When the sample size is decreased from 100 to 50, SEs roughly double for both parameter sets reported here.)

In a second study, we add four covariates. These represent the four kinds of effects suggested in equation (8): variations in the intrinsic tendency to adopt, in susceptibility to influence, in infectiousness, and in social proximity. Values for these covariates were pseudorandomly and independently drawn from a standard Gaussian distribution.

Before discussing the results of this study, we describe how social proximity variables can be handled in our modified version of RATE. Three possible data structures are permitted: a full case-by-case matrix of social proximities, a weighted list for each case of the other cases that can influence it, and a class of distance metrics relating characteristics

[11] We also explored the estimation of simple forms of temporal heterogeneity as postulated in eq. (8). Results for these models are very similar to those reported here for models focusing on spatial heterogeneity and are omitted to conserve space.

[12] Models like eq. (7b) can be estimated with standard event-history software, although the construction of a variable like $\Sigma_{s\in\mathcal{S}(t)} z_{ns}$ is tiresome.

TABLE 1

MONTE CARLO STUDIES OF ESTIMATORS FOR HETEROGENEOUS DIFFUSION MODELS

	True Value	ML ESTIMATE		
		Mean	SD	Mean SE
Study 1:				
Intrinsic tendencies:				
Intercept	−6.00	−5.98	.43	.45
Contagion:				
Intercept	−8.00	−8.01	.16	.16
Study 2:				
Intrinsic tendencies:				
Intercept	−6.00	−6.03	.66	.69
Variable propensity	5.00	5.06	.49	.49
Contagion:				
Intercept	−8.00	−8.16	.67	.63
Susceptibility of n	2.00	2.00	.13	.14
Contagiousness of s	−2.00	−2.09	.44	.40
Proximity of n and s	4.00	4.10	.39	.41

NOTE.—SD = Standard deviation of the ML estimates; mean SE = mean of the estimated standard error of the ML estimate.

of each pair of cases.[13] The full case-by-case proximity matrix includes the list as a special case. We expect, however, that the list structure will often be useful, both because network data are often collected in this fashion and because large networks are often sparse. The full proximity matrix is primarily useful for populations of a modest size (say on the order of $N = 100$), due to $O(n^2)$ data storage and manipulation requirements. In our Monte Carlo studies and empirical examples below, we make use of both the case-by-case matrix of proximities and the list of direct relations. In the Monte Carlo study, social proximity is constructed by pseudorandom assignment to each case of 1–3 other population members to which the case is close.

In the second study all parameter estimates again appear to be unbiased: all mean estimates are close to their true values. The variances of

[13] The class of distance metrics is that of absolute power functions:

$$f(p, q) = D_{ns} = \left(\sum_{k=1}^{K} |x_{nk} - x_{sk}|^p \right)^{1/q},$$

where D_{ns} is the distance between n and s with respect to K variables. When $p = q = 2$, the metric is Euclidean distance. By convention, $p = 0$ and $q = 0$ implies that the measure equals zero if values on all K variables for the dyad are identical, and one otherwise. We can take the inverse of these measures to work in terms of proximities, or use the distance directly.

the estimated parameters remain low, suggesting the capacity to distinguish features of the diffusion process such as susceptibility and infectiousness. It also seems straightforward to estimate parameters describing network relations within a population.[14] We conclude that diffusion models can be effectively estimated using an event-history framework, for at least some ranges of true parameter values.[15]

MEDICAL INNOVATION AS HETEROGENEOUS DIFFUSION

We illustrate the models above through an examination of the adoption process reported by Coleman et al. (1966) in *Medical Innovation*. Their sociometric analysis of the decision to employ tetracycline (named gammanym in the report) as a prescription drug by physicians in four cities is a classic in the sociological study of diffusion. In particular, *Medical Innovation* highlighted relational elements in the adoption process by noting differences between socially integrated and socially isolated physicians. To Coleman et al., the S-shaped curve of cumulative adoptions among centrally located doctors suggested a process of social contagion in which these doctors learned from the prior adoptions of others. In contrast, the constant rate of adoption among socially isolated doctors signaled dependence on sources of information outside the local medical community.

Figure 1 shows estimates of the integrated hazard (Nelson 1972; Aalen 1978) of tetracycline adoption in each of the four cities. Although *Medical Innovation* is commonly regarded as the classic diffusion study in sociology, it is noteworthy that figure 1 only faintly suggests the monotonically increasing hazard rate over time that is characteristic of globally contagious processes. Peoria does show evidence of a simple contagion process, but the other three cities do not. However, the graph is not inconsistent with diffusion operating more locally or for some kinds of physicians and not others.

Coleman et al.'s data have been the subject of considerable recent reanalysis. Burt (1987) examined the local network structure of adoption,

[14] We also examined the case where network density is substantially higher by relating each case to up to 10 (rather than three) neighbors. Estimate quality is not affected by this shift.

[15] In this article we simply provide evidence for the viability of estimation given a properly specified diffusion process. In ongoing work, Greve, Strang, and Tuma (1993) examine estimation properties in much greater detail. One issue has to do with the robustness of diffusion effects across parameter space. A second issue has to do with the viability of estimating diffusion effects from a sample and from populations with some missing data. Prior work on diffusion, to our knowledge, has always assumed full information on the population of spreaders and the population at risk.

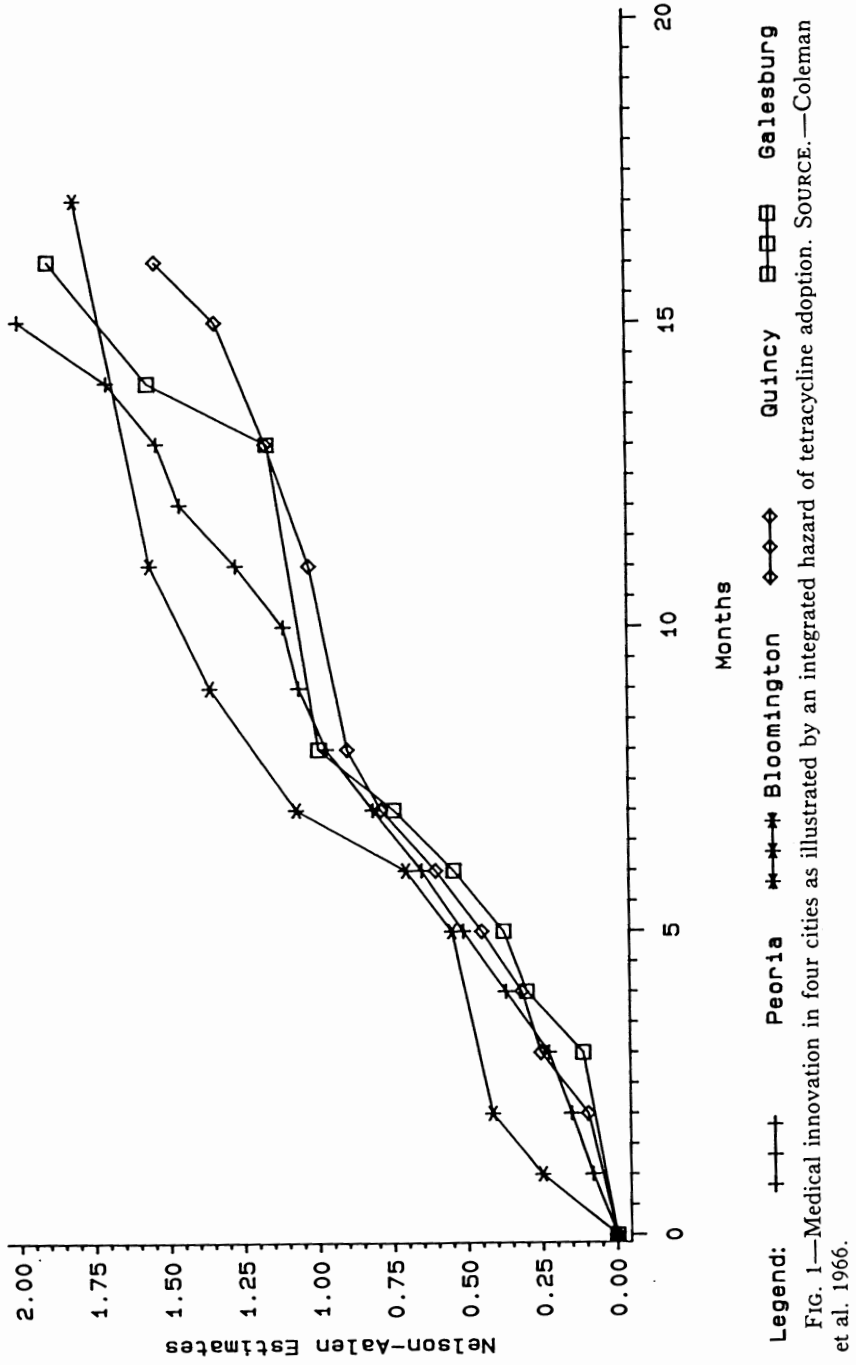

FIG. 1—Medical innovation in four cities as illustrated by an integrated hazard of tetracycline adoption. SOURCE.—Coleman et al. 1966.

contrasting "cohesion" and "structural equivalence" as bases of diffusion. Cohesion refers to the direct influence of a physician's advisors and discussion partners. Structural equivalence refers to the influence of physicians who have similar patterns of ties to others within the medical community, whether or not they are directly linked to one another.[16] In a series of correlational and regression analyses, Burt found that the timing of a physician's adoption of tetracycline is better predicted by the adoption date of structurally equivalent physicians than by the adoption date of advisors and discussion partners. He concluded that "there is strong evidence of contagion through structural equivalence and virtually no evidence of contagion through cohesion" (Burt 1987, p. 1327).[17]

Marsden and Podolny (1990) examined the case for Burt's (1987) argument using event-history methods, very much in the spirit of this article. In particular, they utilized a multiplicative diffusion model like equation (7b), estimated by partial likelihood. The proportion of structurally equivalent alters (and separately, of cohesive alters) who had adopted by the prior month was introduced as a time-varying covariate multiplying the effects of individual characteristics. Marsden and Podolny found that neither the proportion of structurally equivalent alters nor the proportion of cohesive alters significantly increased the hazard rate of adoption.

We address the "structural equivalence versus cohesion" debate yet again, using both additive and multiplicative formulations of heterogeneity in diffusion processes. We then examine some models patterned after Coleman et al.'s major finding, which was not about local network structure at all, but about network centrality.[18]

Data and Variables

We briefly describe the *Medical Innovation* study. Of particular relevance are limitations that point to broader data collection and research design issues for diffusion analysis.

[16] Borgatti and Everett (1992) argue that since structural equivalence measures the openness of dyads to the same mixture of signals from combinations of other actors, its effects may be best understood as capturing a complex combination of direct interpersonal influences. In contrast, Burt suggests that structurally equivalent actors imitate each other because they stand in an implicitly competitive relation (1987, pp. 1291–94).

[17] As noted above, we think that linear regression analyses are inappropriate to the study of diffusion and, indeed, of any adoption process occurring over time.

[18] Burt (1987) uses the term *prominence* instead of centrality; Marsden and Podolny (1990) use the term *integration*. The actual measure is the number of times the physician was named as an advisor or discussion partner by another physician in the community. Both analyses find a zero-order effect of centrality that disappears once intrinsic propensities to adopt local network effects are incorporated in the model.

The event of interest is a physician's first prescription of tetracycline. Coleman et al. gathered a "behavioral trace" of tetracycline adoption by auditing the prescription records of 125 physicians.[19] These physicians were virtually all "generalists" (general practitioners, internists, and pediatricians) practicing medicine in the four cities under study. Prescriptions written by each physician were audited for a three-day period each month; the first month this audit uncovered a tetracycline prescription was taken as the adopting month. Of the 125 physicians, 109 prescribed tetracycline during the 17-month observation period.

Coleman et al. (1966) collected survey data on various physician characteristics that might affect adoption. In analyses below, we include several factors found to predict adoption in prior research as control variables: whether the physician had a scientific (vs. a patient-centered) orientation to medicine, the physician's "professional age" (equaling one for medical school graduation before 1930 and zero otherwise), and a score for medical journal subscriptions.[20] We ignore other individual characteristics to limit the loss of cases due to survey nonresponse. Exploratory analyses not reported here indicated that the inclusion of the other variables analyzed by Burt (1987) and by Marsden and Podolny (1990) did not affect the pattern of contagion reported below.

Finally, Coleman et al. collected sociometric data on relations within the medical community in each city. Physicians were asked to cite up to three physician "friends," "advisors," and "discussion partners." (Note the potential for missing data on intrapopulation influences, since those with four or more partner relations were allowed to mention only three.) Following Burt and Marsden and Podolny, we limit our analysis to the advising and discussion relations.

This procedure generated citations to physicians specializing in various branches of medicine, and thus outside the "prescription sample" of medical generalists. Coleman et al. (1966) collected data on 91 of these specialists to map the social structure of each medical community, but did not collect data on the specialists' prescription patterns. Thus, Coleman et al.'s data do not completely cover the entire population conceivably relevant to a diffusion study, and their "prescription sample" is not a random sample of that larger population.

We follow Burt (1987, pp. 1330–31) and Marsden and Podolny (1990) in the treatment of cohesive relations (prior adoption by advisors and

[19] Coleman et al. (1966) sought to collect data on 130 physicians, but no prescriptions of any kind could be recovered for five, who are omitted from the analyses below.

[20] Several scales were constructed by Burt (1987) to maximize the effect of individual propensities, thus permitting a strong test of contagion effects. The three scales that we use are the ones that Burt found to show the strongest relation to adoption.

discussion partners) and in our general approach to structural equivalence. But while Burt used his informed judgment as a network analyst to assign physicians to structurally equivalent blocs, we use standardized proximity scores between each pair of doctors in the same city (Burt's w_{ij}, p. 1329). We differ more substantially by allowing only generalists to enter into $\mathcal{S}(t)$, although global network measures are formed using all available data on the network structure of each medical community (see below for discussion).

The design of the *Medical Innovation* study suggests two major concerns for a diffusion analysis. First, discrete and episodic measurement of the timing of adoption (three-day prescription audits converted into monthly adoption dates) presents problems. Adoption month of physicians prescribing tetracycline irregularly will be upwardly biased as the three-day audits may miss their first month of prescribing tetracycline. More important, measuring adoption only to the nearest month produces many artificial ties in adoption dates. When an advisor and advisee pair are recorded as adopting in the same month, should the advisor be assumed to have affected the advisee, or not? Or should we develop techniques that average over all possible temporal sequences?

Coleman et al. (1966) and Burt (1987) assumed that adoption in the same month is the strongest possible evidence of mutual influence. In contrast, Marsden and Podolny (1990) assumed that those who adopted in the same month exerted no influence on each other. To maximize the comparability of our results with prior research by Coleman et al. and Burt, we report analyses that permit mutual contemporaneous influence (here, within the same month).[21] We note how results differ when, like Marsden and Podolny, we assume that only adoptions in prior months are potentially influential. We emphasize that future work on diffusion might further consider how to handle artifactual ties in adoption times.

The second problem with the Coleman et al. data is that the prescription sample may be seriously incomplete. Prescription audits were performed for nearly all medical generalists in the four cities, but not for any medical specialists. This becomes a concern if the specialists' adoption of tetracycline affects the generalists' propensity to adopt. Moreover, it raises the question of whether the reverse happened—adoption by generalists may have affected adoption by specialists, who may, in turn, have affected other generalists and other specialists.

Both Burt and Marsden and Podolny assumed that the use of tetracycline by specialists affected generalists and consequently imputed adoption dates for specialists (though they analyzed only the generalists' adop-

[21] An "influencing" event occurring in the same month as an "influenced" event is treated as having occurred midway through the month.

tions). On the other hand, presumably Coleman et al. did not sample the prescriptions of specialists because they believed the adoptions of specialists and generalists were unconnected. We follow their reasoning and assume that medical generalists attended only to other medical generalists, and not to specialists with whom they were directly linked or structurally equivalent. Prescription data on specialists as well as generalists are needed to test hypotheses about who really influenced whom.

Again, the central point is that research investigating diffusion must carefully consider how to bound the population under study. The diffusion methods discussed here and in previous research assume complete data on the population at risk of adopting. This requirement is due to the fact that adoptions enter not only as the dependent variable but as explanatory covariates as well. In further work Greve, Strang, and Tuma (1993) assess the degree to which various forms of missing data adversely affect the estimation of individual-level diffusion models.

Baseline Diffusion Models

We begin by examining the form of diffusion effects: whether there is evidence for contagion within the four medical communities, and whether the impact of others' adoptions appears to vary over time. Table 2 presents some baseline models considering these influences in addition to those of individual propensities related to the physician's orientation to medicine, professional age, and attention to professional journals. We employ the additive diffusion formulation in equation (8) since a main goal here is to consider the place of temporal heterogeneity in the diffusion of tetracycline.

Model 1 in table 2 reports a homogeneous mixing model of diffusion. The addition of a diffusion effect for all prior or contemporaneous adoptions in the city produces a significant increase in the likelihood-ratio chi square. Each adoption within the city adds .0031 [= exp(−5.78)] to the hazard.[22] This suggests a modest contagion effect, in line with the slowly accelerating hazard rate indicated in figure 1. For example, after 10 physicians have adopted tetracycline in a community, the estimated rate is 1.4 times larger than the base rate (when no adoptions have occurred). In the largest city studied, Peoria, the estimated rate at the end of the study (after 55 adoptions) is nearly 3.3 times larger than the base rate.

Model 2 permits the influence of prior events to vary as an exponential

[22] In a Cox model, this term would be an element in the unobserved common temporal variation that is treated as a nuisance parameter. We think it informative for present purposes to measure the effect of homogeneous contagion explicitly, although more refined analyses might locate some of this effect in time-varying environmental factors.

TABLE 2

BASELINE DIFFUSION MODELS: ML ESTIMATES OF ADDITIVE DIFFUSION MODELS OF TETRACYCLINE ADOPTION FOR 121 PHYSICIANS (SEs in Parentheses)

	MODEL			
	1	2	3	4
Intrinsic tendencies:				
Intercept	−2.60***	−2.99***	−3.33***	−4.39***
	(.19)	(.39)	(.40)	(.79)
Scientific orientation			.37***	.55**
			(.13)	(.24)
Professional age			−.75***	−1.05*
			(.25)	(.54)
Journal subscriptions			.48***	.73**
			(.17)	(.32)
Contagion:				
Intercept	−5.78***	−4.27***		−5.98***
	(.28)	(.47)		(.73)
Time decay		−.44*		−.02
		(.23)		(.10)
Likelihood ratio χ^2 versus constant hazard model	6.5**	14.5***	27.4***	37.5***
df	1	2	3	5

* Significant at the .10 level.
** Significant at the .05 level.
*** Significant at the .01 level.

function of time since their occurrence. The coefficient for this term is negative and significant, suggesting that physicians who have recently begun to prescribe tetracycline are more "infectious" than those who have been prescribing for some time. One might imagine physicians are more likely to communicate about their use of a drug they have recently begun to use.[23]

Model 3 examines factors affecting the physician's propensity to adopt. It adds the three physician characteristics discussed above that have been found by prior work to be strong predictors of tetracycline adoption. These variables show the expected relationships: scientific orientation and journal subscriptions raise the speed of adoption, while professional age lowers the rate of adoption. Separately and together, these findings suggest that tetracycline was adopted earlier by more "modern-minded" physicians connected to technical advances in medicine.

[23] The alternative outcome is not inconceivable: physicians might not advocate a prescription drug (or colleagues might not attend) until it has stood the test of time.

Model 4 examines the role of intrinsic propensities and contagion simultaneously. The three physician characteristics remain strong predictors of adoption: in fact, the coefficients of these characteristics increase in magnitude once contagion effects are included in the model. There is evidence that prior events influence adoption, net of individual propensities. But the effect of temporal change in contagiousness disappears once individual propensities to adopt are controlled. Apparently the finding suggested in model 2 is due to aggregating the responses to contagion of physicians having different intrinsic propensities to adopt. As a first main result, we conclude that there is evidence of contagion that is temporally homogeneous.

Local Network Structure

We now turn to a core substantive issue: To what extent does influence flow between directly related (cohesive) physicians and to what extent does it flow between structurally equivalent physicians? Since table 2 indicates no temporal decay in influence once individual propensities are added to the model, we ignore such effects. This lets us compare results across the two modeling frameworks developed above. Table 3 reports results for multiplicative models of diffusion while table 4 reports results for additive models of diffusion.

In both tables we employ a baseline model 1 that includes individual propensities and homogeneous mixing. In models 2, 3, and 4 in both tables we then examine forms of spatial heterogeneity: whether diffusion is mediated by local network structures of advice giving and discussion and by similarities in network location within the medical community as a whole.

The multiplicative and additive formulations of diffusion suggest somewhat different inferences about the impact of local network structure. The multiplicative model in table 3 indicates strong influences between physicians who are structurally equivalent and little influence of advisors and discussion partners. This pattern is the one reported by Burt (1987): structural equivalence channels contagion, while cohesion does not.[24]

[24] The qualitative pattern of these effects is unchanged when we control for common temporal variation via partial likelihood estimation and expand the set of exogenous influences to include those effects discussed in prior work (i.e., when the intrinsic propensity to adopt is treated as also a function of network centrality, contact by a detail man, and prescription volume). It appears that differences between our findings and the absence of local network effects reported by Marsden and Podolny (1990) are primarily due to different assumptions about the effects of specialists on generalists, as discussed above. This underscores the importance of how the set of spreaders is defined.

TABLE 3

LOCAL NETWORK STRUCTURE: ML ESTIMATES OF MULTIPLICATIVE DIFFUSION MODELS OF TETRACYCLINE ADOPTION FOR 121 PHYSICIANS (SEs in Parentheses)

	MODEL			
	1	2	3	4
Intrinsic tendencies:				
Intercept	−4.07***	−4.06***	−4.30***	−4.29***
	(.45)	(.45)	(.43)	(.46)
Scientific orientation	.42***	.41***	.46**	.45***
	(.14)	(.14)	(.13)	(.13)
Professional age	−.96**	−.94**	−1.00**	−.99***
	(.26)	(.26)	(.26)	(.26)
Journal subscriptions	.63***	.61***	.60***	.59***
	(.17)	(.17)	(.17)	(.17)
Contagion:				
Intercept	.02***	.01**	.01**	.15***
	(.006)	(.008)	(.008)	(.008)
Direct relations		.16		.07
		(.17)		(.12)
Structural equivalence			.89***	.83**
			(.32)	(.34)
Likelihood ratio χ^2 versus constant hazard rate model	40.0***	41.9***	47.2***	47.6***
df	4	5	5	6
Likelihood ratio χ^2 versus equation (1)		1.9	7.2***	7.6**
df		1	1	2

* Significant at the .10 level.
** Significant at the .05 level.
*** Significant at the .01 level.

An additive formulation of diffusion suggests that both cohesion and structural equivalence channel diffusion. Doctors whose advisors or discussion partners prescribe tetracycline are quicker to begin prescribing it themselves, as are doctors with similar patterns of relations to other members of the medical community.[25] The two effects are significant both separately and jointly. In fact, the impact of direct relations between physicians is enhanced when we control influences mediated by structural equivalence.

These results suggest that we should not be quick to discount the

[25] We should note, however, that analyses not permitting influence in the same month yield smaller and statistically insignificant effects of cohesive relations. Structural equivalence effects are not much affected by alternative treatments of apparently simultaneous adoptions.

TABLE 4

LOCAL NETWORK STRUCTURE: ML ESTIMATES OF ADDITIVE DIFFUSION MODELS OF TETRACYCLINE ADOPTION FOR 121 PHYSICIANS (SEs in Parentheses)

	MODEL			
	1	2	3	4
Intrinsic tendencies:				
Intercept	−4.37***	−4.51***	−4.56***	−4.50***
	(.68)	(.71)	(.71)	(.72)
Scientific orientation	.54***	.57***	.50**	.52***
	(.20)	(.21)	(.20)	(.21)
Professional age	−1.05**	−1.18**	−1.19**	−1.31***
	(.40)	(.46)	(.44)	(.49)
Journal subscriptions	.73***	.76***	.83***	.79***
	(.24)	(.26)	(.26)	(.27)
Contagion:				
Intercept	−6.11***	−6.71***	−6.22***	−6.79***
	(.36)	(.75)	(.35)	(.73)
Direct relations		3.11***		3.32***
		(1.11)		(.97)
Structural equivalence			5.79***	2.79**
			(.77)	(1.16)
Likelihood ratio χ^2 versus constant hazard rate model	37.5***	41.0***	44.8***	48.2***
df	4	5	5	6
Likelihood ratio χ^2 versus equation (1)		3.5*	7.3***	10.7***
df		1	1	2

* Significant at the .10 level.
** Significant at the .05 level.
*** Significant at the .01 level.

impact of direct relations on diffusion. Such effects do appear less robust than those of structural equivalence. But if we understand the additive and multiplicative formulations as suggesting different functional forms for diffusion, we should take the appearance of a cohesion effect within one of the two modeling frameworks seriously. It may be that the impact of direct relations is quite independent of the effects of individual propensities. If so, a multiplicative model errs in assuming that the two are highly interdependent. An additive model may better capture the separateness of individual effects and contagion.

Centrality

Our focus on local network influences above, like the analyses by Burt (1987) and by Marsden and Podolny (1990), runs counter to the main

argument advanced by Coleman et al. (1966), who stressed how patterns of adoption among socially central and isolated physicians differed. We next examine the effects of centrality using an additive diffusion model. This permits an illustration of the distinctive role that variables may have in different components of a diffusion process.

Network centrality is measured by the number of times a physician is cited as an advisor or discussion partner. Arguments can be suggested for three types of effects. First, a physician's centrality may directly affect his propensity to adopt a new drug. For example, a highly central physician's concern for reputation may raise (or lower) the costs of innovation. Second, physicians receiving many citations may be more susceptible to the adoptions of others, due to their wider circle of contacts. And third, physicians receiving many citations may be especially influential within their medical communities, again due to the extensiveness of their contacts. If so, their adoptions should be highly infectious.

Model 1 in table 5 suggests that centrality increases susceptibility. Physicians who are often cited as advisors and discussion partners seem more influenced by the prescription behavior of others. There is no apparent relationship between centrality and the physician's intrinsic propensity to adopt. More surprising from a relational perspective, there is also no apparent relationship between centrality and infectiousness.

When we expand the model to include features of local network structure discussed above, the enhanced susceptibility of central physicians remains strong (note the substantial difference in likelihood-ratio chi square statistics between model 4 in table 4 and model 2 in table 5). Further, while the effect is not quite significant at conventional levels, there is some indication that centrality is associated with lowered infectiousness. That is, once we control for the influences of advisors and discussion partners, and of structurally equivalent physicians, tetracycline adoptions by globally central physicians seem to exert weak influences on other members of the medical community.

It is of interest that centrally located physicians are highly susceptible but not infectious. One possible explanation is that advisor or discussion partner citations refer to informationally asymmetrical relationships, where the advisor or partner receives information from the advisee without returning it. But if informational asymmetry is driving the effects of centrality, we should see stronger direct influences running from advisee to advisor than from advisor to advisee. However, models directly testing these "reverse" influences show even weaker effects than do the models in tables 3 and 4, which examine the effects of cited physicians on citing physicians.

The fact that the infectiousness of central physicians declines once local ties are added (in model 2) suggests an alternative account. Physicians

TABLE 5

NETWORK CENTRALITY: ML ESTIMATES OF ADDITIVE DIFFUSION MODELS OF TETRACYCLINE ADOPTION FOR 121 PHYSICIANS (SEs in Parentheses)

	Model		
	1	2	3
Intrinsic tendencies:			
Intercept	−4.47***	−4.67***	−4.43***
	(.77)	(.83)	(.78)
Scientific orientation	.60**	.58**	.38*
	(.23)	(.25)	(.22)
Professional age	−1.08**	−1.40**	−1.36**
	(.47)	(.65)	(.56)
Journal subscriptions	.75**	.83**	.86**
	(.31)	(.34)	(.33)
Network centrality	−.04	−.05	−.04
	(.05)	(.05)	(.05)
Contagion:			
Intercept	−5.70**	−5.76***	−5.59***
	(.71)	(.49)	(.67)
Susceptibility of central *n*'s	.11***	.12***	.10***
	(.03)	(.03)	(.03)
Infectiousness of central *s*'s	−.20	−.40	−.08
	(.42)	(.27)	(.16)
Proximity of *n* and *s* based on:			
Direct relations		3.29***	2.54**
		(.80)	(.97)
Structural equivalence		2.05*	3.70**
		(1.10)	(1.43)
Scientific orientation			1.71*
			(.95)
Likelihood ratio χ^2 versus constant hazard rate model	42.2**	54.8***	57.8***
df	7	9	10

* Significant at the .10 level.
** Significant at the .05 level.
*** Significant at the .01 level.

with many contacts may communicate less with each advisee or discussion partner, due to limited attention. The result is lower infectiousness per contact but higher total influence, since the impact of each direct contact is considerably larger than the decrement associated with increase of one in measured centrality.[26] This effect does not appear very large, however.

[26] We owe this insight to Arthur Stinchcombe, who offers a better example: a male rock star may father many children because many women admire him, but among the women who admire him, only a small percentage become pregnant.

Finally, we note that social structure may be conceived in terms of patterned similarities in personal characteristics (Blau 1977, 1989; Carley 1986) as well as concrete (or abstract) network relations. To look for this kind of structural effect, we ask whether contagion was structured by similarities in the individual characteristics of physicians. In particular, we investigated whether physicians who hold the same value on scores for medical orientation, professional age, and journal subscriptions tend to influence each other more.

One such effect was found, which we report in model 3 of table 5. Physicians who share a common orientation toward medicine (either a scientific orientation or a patient-centered orientation or a mixed orientation) appear to attend more to each other than physicians whose orientations differ. Scientifically minded physicians attend to the decisions and experiences of similarly minded physicians; patient-oriented physicians to other patient-oriented physicians. This suggests an additional dimension to diffusion, one that stands between the effects of individual characteristics on the intrinsic propensity to adopt and the channeling of contagion by network relations.

CONCLUSIONS

In this article we discuss methods for the study of spatial and temporal heterogeneity in diffusion. We propose an event-history formulation of contagion that permits the analyst to model the impact of characteristics of the adopter and the spreader, the spreader's adoption event, and their linkages. The chief advantage of this approach, we believe, is that it opens up new avenues for the exploration and testing of social structural hypotheses: stated broadly, of how individuals or other social entities are affected by what others do. Notions about susceptibility to external influence, the infectiousness of actors and types of action, and the social proximity of sets of actors can be deployed more extensively when the separate traditions of diffusion analysis and individual-level event-history methods are combined.

Some of the possibilities (as well as the complexities) of this strategy are suggested in the analysis of the *Medical Innovation* data. The results above support many of the insights of Coleman et al. (1966) and Burt (1987) into the structure of diffusion. But they also suggest that the processes involved are more complex and varied than prior research indicates. Like Burt (1987) and Marsden and Podolny (1990), we find that network centrality does not affect the intrinsic propensity of physicians to adopt, once other features of the process are taken into account. But centrality is importantly connected to contagion via increased susceptibility to others' adoptions, in many ways consistent with the discussion in

Coleman et al. Furthermore, unlike Burt, we find that contagion in medical innovation is not a simple product of structural equivalence. Cohesive ties based on advice giving and discussion also contribute to diffusion, as do structures of similarity in physicians' orientation toward their work. These kinds of findings illustrate the importance of developing models of diffusion whose complexity better maps onto the multiple pathways of influence within social settings.

REFERENCES

Aalen, Odd O. 1978. "Nonparametric Inference for a Family of Counting Processes." *Annals of Statistics* 6:701–26.

Bailey, Norman T. 1976. *The Mathematical Theory of Infectious Diseases and Applications*. New York: Hafner.

Bartholomew, David J. 1982. *Stochastic Models for Social Processes*, 3d ed. New York: Wiley.

Blau, Peter M. 1977. *Inequality and Heterogeneity*. New York: Free Press.

———. 1989. "Structures of Social Positions and Structures of Social Relations." Chap. 3 in *Theory Building in Sociology*, edited by J. Turner. Newbury Park, Calif.: Sage.

Borgatti, Stephen P., and Martin G. Everett. 1992. "Notions of Position in Social Network Analysis." Pp. 1–35 in *Sociological Methodology*, edited by P. V. Marsden. Cambridge, Mass.: Blackwell.

Burt, Ronald S. 1987. "Social Contagion and Innovation: Cohesion versus Structural Equivalence." *American Journal of Sociology* 92:1287–1335.

Carley, Kathleen. 1986. "An Approach for Relating Social Structure to Cognitive Structure." *Journal of Mathematical Sociology* 12:137–89.

Coleman, James S., Elihu Katz, and Herbert Menzel. 1966. *Medical Innovation*. New York: Bobbs-Merrill.

Cox, David R. 1972. "Regression Models and Life Tables." *Journal of the Royal Statistical Society* B34:187–220.

———. 1975. "Partial Likelihood." *Biometrika* 62:269–76.

Diekmann, Andreas. 1989. "Diffusion and Survival Models for the Process of Entry into Marriage." *Journal of Mathematical Sociology* 14:31–44.

Doreian, Patrick. 1981. "Estimating Linear Models with Spatially Distributed Data." Pp. 359–88 in *Sociological Methodology*, edited by S. Leinhardt. San Francisco: Jossey-Bass.

Granovetter, Mark. 1978. "Threshold Models of Collective Behavior." *American Journal of Sociology* 83:1420–43.

Greve, Henrich, David Strang, and Nancy B. Tuma. 1993. "Estimation of Diffusion Processes from Incomplete Populations." Paper presented at the annual meetings of the American Sociological Association, Miami.

Hernes, Gudmund. 1972. "The Process of Entry into First Marriage." *American Sociological Review* 37:173–82.

Knoke, David. 1982. "The Spread of Municipal Reform: Temporal, Spatial, and Social Dynamics." *American Journal of Sociology* 87:1314–39.

Marsden, Peter V., and Joel Podolny. 1990. "Dynamic Analysis of Network Diffusion Processes." Pp. 197–214 in *Social Networks through Time*, edited by H. Flap and J. Weesie. Utrecht: ISOR.

Nelson, Wayne. 1972. "Theory and Application of Hazard Plotting for Censored Failure Data." *Technometrics* 14:945–65.

Rogers, Everett M. 1983. *Diffusion of Innovations*. New York: Free Press.

Strang, David. 1990. "From Dependency to Sovereignty: An Event History Analysis of Decolonization, 1870–1987." *American Sociological Review* 55:846–60.

———. 1991*a*. "Adding Social Structure to Diffusion Models: An Event History Framework." *Sociological Methods and Research* 19:324–53.

———. 1991*b*. "Global Patterns of Decolonization, 1500–1987." *International Studies Quarterly* 35:429–54.

Strang, David, and John W. Meyer. 1993. "Institutional Conditions for Diffusion." *Theory and Society* 22:487–512.

Tolbert, Pamela S., and Lynne G. Zucker. 1983. "Institutional Sources of Change in the Formal Structure of Organizations: The Diffusion of Civil Service Practices." *Administrative Science Quarterly* 28:22–39.

Tuma, Nancy B. 1980. *Invoking RATE*. Menlo Park, Calif.: SRI International.

Tuma, Nancy B., and Michael T. Hannan. 1984. *Social Dynamics: Models and Methods*. Orlando, Fla.: Academic.

Zielske, Hubert A., and Walter A. Henry. 1980. "Remembering and Forgetting Television Ads." *Journal of Advertising Research* 20:7–13.