Social Contagion and Innovation: Cohesion versus Structural Equivalence[1]

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Two classes of network models are used to reanalyze a sociological classic often cited as evidence of social contagion in the diffusion of technological innovation: *Medical Innovation*. Debate between the cohesion and structural equivalence models poses the following question for study: Did the physicians resolve the uncertainty of adopting the new drug through conversations with colleagues (cohesion) or through their perception of the action proper for an occupant of their position in the social structure of colleagues (structural equivalence)? The alternative models are defined, compared, and tested. Four conclusions are drawn: (*a*) Contagion was not the dominant factor driving tetracycline's diffusion. Where there is evidence of contagion, there is evidence of personal preferences at work.

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(*b*) Where contagion occurred, its effect was through structural equivalence not cohesion. (*c*) Regardless of contagion, adoption was strongly determined by a physician's personal preferences, but these preferences did not dampen or enhance contagion. (*d*) There is no evidence of a physician's network position influencing his adoption when contagion is properly specified in terms of structural equivalence. The ostensible prestige effect is spurious, resulting from biases created when cohesion is used to model contagion. In short, the product of reanalyzing the *Medical Innovation* data with recent developments in network theory is clearer, stronger evidence of social contagion and a redefinition of the social structural conditions responsible for contagion.

The spread of new ideas and practices is often argued to be contingent on the way in which social structure brings people together. Adopting an innovation entails a risk, an uncertain balance of costs and benefits, and people manage that uncertainty by drawing on others to define a socially acceptable interpretation of the risk. Social contagion arises from people proximate in social structure using one another to manage the uncertainty of innovation. At the heart of social contagion is the interpersonal synapse over which innovation is transmitted. Here, agreement breaks down. What is it about the social structural circumstances of two people that makes them proximate such that one's adoption of an innovation can be expected to trigger the other's adoption? Debate in network theory has crystallized around two answers to this question: cohesion and structural equivalence. This paper is a comparison of the two answers. I draw out the theoretical arguments for cohesion and structural equivalence, highlighting the empirical circumstances in which they could contradict one another, and use them to reanalyze a sociological classic often cited as evidence of social contagion in the diffusion of technological innovation: Coleman, Katz, and Menzel's (1966) *Medical Innovation*.

CONTAGION IN THEORY

In the simplest case, the interpersonal synapse over which social contagion occurs involves one individual, ego, who has not yet adopted the innovation under study, and a second individual, alter, who has adopted. Something about the social structural circumstances of ego and alter makes them proximate such that ego's evaluation of the innovation is sensitive to alter's adoption. Contact, communication, and competition have been argued, each in turn, as making ego and alter proximate.

Physical proximity alone has some capacity to cause social contagion. The closer the physical contact is between ego and alter, the more likely that alter's adoption will trigger ego's. Merely witnessing alter's adoption

can transmit significant information to ego. He not only becomes aware of the innovation, he also has the benefit of a vicarious trial use, witnessing the consequences adoption has for alter. This sense of social contagion is most articulately developed in geography (see, e.g., Cliff et al. 1981) and epidemiology (see, e.g., Bailey 1976), but it is occasionally found in a social network analysis (see, e.g., White, Burton, and Dow 1981) and of course has precedents in early sociological and anthropological accounts.

Cohesion and structural equivalence generalize physical proximity, addressing a fundamental change in the availability of information on innovations. With the omnipresence of mass media and people paid to disseminate information on an innovation, obtaining information is less a problem for the modern innovator than finding trustworthy information; even worse, the problem lies in finding ways to ignore as much as possible of the otherwise overwhelming horde of facts.[2] Cohesion and structural equivalence shift attention from the question of whether people are adopting in ego's physical surroundings to the question of who is adopting. Taking access to information for granted, they focus on the problem of managing uncertainty in making the proper response to information.

Cohesion

The cohesion model focuses on socialization between ego and alter. The more frequent and empathic the communication is between ego and alter, the more likely that alter's adoption will trigger ego's. Discussing the innovation with others, ego comes to a normative understanding of adoption's costs and benefits, a social understanding colored by the interests of the people with whom the innovation has been discussed.

With the spread of mass media and development of sociometric social psychology in the decades surrounding World War II, it was shown that social proximity (such as friendships) developed from physical proximity and that shared attitudes developed from social proximity. Homans's

[2] This theme is entertainingly elaborated by Klapp (1978) with respect to conditions under which we are open or closed to receiving information. Coleman et al. make the same point, seeing this as a routine dilemma facing the physician evaluating medical innovations: "The problem exists not because information is inaccessible. On the contrary. . . . The physician's serious problem is in knowing how to sift through the deluge of material that reaches him, and how to assess the value of it. . . . Most practitioners, of course, are far too busy to follow the advances in all specialty journals, to take time out for a postgraduate refresher course, or to attend more than a very few out-of-town conventions. . . . At the same time, the physician is under pressure from his own conscience and his professional commitment to afford his patients the benefit of recent discoveries. . . . Decisions as to what to use, when to use it, and on whom, must constantly be made—even when the doctor has little basis on which to decide" (1966, pp. 13–14).

(1950) *The Human Group* provides a theoretical exemplar for this period (continued in Homans's *Social Behavior* [1961, pp. 112–29]). Festinger, Schachter, and Back's (1950) study of housing, friendship, and involvement in a voluntary association provides a research exemplar. Building explicitly on Sherif's (1935) experimental studies of interpersonal influences created by physical proximity between socially similar people (Columbia University undergraduate psychology students), Festinger et al. (1950) emphasize the causal force of normative understandings created in informal social groups (see Homans 1961, pp. 120–25). When confronted with an empirically ambiguous question, a question that cannot be resolved by concrete facts, people turn to the people with whom such questions are discussed and, in their reciprocally socializing debate, create a consensual, normative understanding of the question, resolving the question's uncertainty in their own minds, if not in fact. As a result of this understanding, ego's adoption quickly follows alter's because they have come to share the same evaluation of adoption's costs and benefits. This line of thought underlies the seminal studies of informal social pressures on voting in the 1940 and 1948 presidential elections (Lazarsfeld, Berelson, and Gaudet 1944; Berelson, Lazarsfeld, and McPhee 1954); it lies behind the studies of opinion leaders in the two-step flow of mass-media diffusion (Merton 1949, see also 1957; Katz and Lazarsfeld 1955); and it is the driving force in Coleman et al.'s (1966) *Medical Innovation*.[3] Echoing Festinger et al., Coleman et al. (1966, pp. 118–19) argue: "Confronted with the need to make a decision in an ambiguous situation—a situation that does not speak for itself—people turn to each other for cues as to the structure of the situation. When a new drug appears, doctors who are in close interaction with their colleagues will similarly interpret for one another the new stimulus that has presented itself, and will arrive at some shared way of looking at it." They go on to present evidence of a tendency for physicians to begin prescribing the new drug at about the same time if they had a relationship of sharing advice on cases or discussing medical matters. This theme continues today in studies reporting attitude, belief, or behavior similarity between people connected by strong communication relations (see, e.g., Duncan, Haller, and Portes [1968] on occupational and educational aspirations; Fischer [1978] on the diffusion of innovations between distant urban centers before reaching surrounding rural areas; Shrout and Kandel [1981] on the use of illegal drugs; and Friedkin [1984] on perceived consensus on educational policy).

[3] Of course, these are merely highlights in the broad program of empirical interpersonal influence research disseminated from Columbia University's Bureau of Applied Social Research during the 1940s and 1950s. Barton (1982) provides an informal insider's account of how the research program developed at the bureau.

Structural Equivalence

The structural equivalence model highlights competition between ego and alter. This includes, in the extreme, the competition of people fighting one another for survival but applies more generally to the competition of people merely using one another to evaluate their relative adequacy—for example, two siblings close in age and trying to get good grades in the same subjects who are encouraged by their parents, two graduate students publishing the same kind of work and trained by the same professors, or two physicians trying to keep up with the rush of medical developments in order to live up to their image of a good physician and maintain their position in the social structure of medical advice and discussion. The more similar ego's and alter's relations with other persons are—that is, the more that alter could substitute for ego in ego's role relations, and so the more intense that ego's feelings of competition with alter are—the more likely it is that ego will quickly adopt any innovation perceived to make alter more attractive as the object or source of relations. Discussing an innovation with others, ego comes to a normative understanding of adoption's costs and benefits to a person fulfilling his roles, a social understanding shared by others in those roles and colored by ego's interest in the advantage accruing to anyone performing his roles.

Structurally equivalent people occupy the same position in the social structure and so are proximate to the extent that they have the same pattern of relations with occupants of other positions. More specifically, two people are structurally equivalent to the extent that they have identical relations with all other individuals in the study population. As illustrated by the starkly oversimplified situations in figure 1, structural equivalence overlaps, restricts, and extends the concept of cohesion.

Figure 1*A* illustrates the kind of situation in which structural equivalence and cohesion make identical predictions. Ego and alter have strong relations with each other, so that contagion between them is predicted by cohesion. At the same time, they have identical patterns of relations—having strong relations with the same people and no relations with the same people—so that contagion between them is also predicted by structural equivalence. More generally, structural equivalence and cohesion both predict contagion (if for different reasons) between people strongly tied to each other and similarly tied to other persons.

Figure 1*B* illustrates the kind of situation in which cohesion predicts contagion and structural equivalence does not. Ego and alter have strong relations with each other and so are again expected to act similarly under cohesion. However, they have different patterns of relations—ego strongly tied to one person and alter strongly tied to another—so that they are not structurally equivalent. More generally, structural equivalence

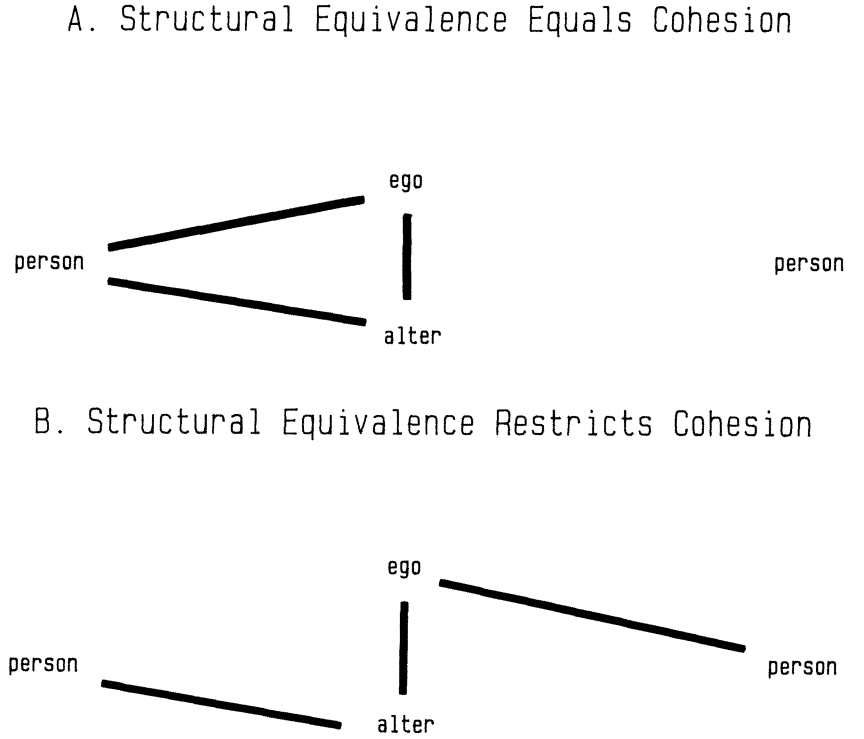

FIG. 1.—Kinds of social structural situations in which structural equivalence and/or cohesion predict contagion between ego and alter.

predicts that the socializing influence of cohesive ties within a clique can be eliminated by conflicting ties outside the clique.

Finally, figure 1*C* illustrates the kind of situation in which structural equivalence predicts contagion and cohesion does not. Ego and alter have no relations with each other and so do not socialize each other directly. However, ego and alter are structurally equivalent because of their identical pattern of relations with others—both ego and alter are tied to the same people—so that contagion between them is predicted by structural equivalence. Contagion is expected, for the same reason as for the two people in figure 1*A*, because they are identically outsiders to the ego-alter clique. More generally, structural equivalence predicts that two people identically positioned in the flow of influential communication will use each other as a frame of reference for subjective judgments and so make similar judgments even if they have no direct communication with each other. Frequent or empathic communication is not essential to their keen awareness of each other. People involved in relations with the same people are likely to have a direct and indirect awareness of each other: direct by meeting when interacting with their mutual acquaintances and indirect by hearing about each other through mutual acquaintances. They may or may not have strong relations with each other. It is their similar relations with others that determine their structural equivalence, not their relations with each other.[4]

These are familiar ideas, fundamental to the traditional view of social structure as a system of statuses interlocked by role relations (see, e.g., Linton 1936; Merton 1957; Nadel 1957); indeed, structural equivalence models were developed during the 1970s explicitly as a vehicle for describing the structure of role relations defining statuses across multiple networks (see, e.g., Burt 1982, pp. 42–49, 63–69, and 333–47 for re-

[4] It is easy to misperceive the shift in theory that structural equivalence represents. A vulgar understanding of structural equivalence views social contagion by structural equivalence to be no more than an indirect effect of cohesion. To the extent that two people have identical relations with others, they are involved in the same socializing communication and so come to share the same evaluation of empirically ambiguous objects (see, e.g., Burt 1978; Friedkin 1984). Figure 1*C* illustrates this in the extreme case, in which ego and alter have no direct communication with each other but extensive indirect communication through shared contacts. So viewed, however, there is no difference between cohesion and structural equivalence as the driving force in social contagion; in either case, ego is expected to reflect the attitudes and behaviors of the people with whom he has strong relations. Consistent empirical differences between the predictions of cohesion and structural equivalence cannot be explained with such an understanding of structural equivalence. E.g., if we return to figure 1*C* and anticipate the lack of empirical support in the *Medical Innovation* data for contagion by cohesion, it seems wrong to attribute evidence of ego-alter contagion to indirect communication through shared contacts when there is no evidence of contagion where communication is direct.

view). More important, the relational meaning of the status/role-set duality gained enormous rigor in network models of structural equivalence, and the rapid deployment of these models in empirical research marked a major departure from the cohesion models dominant at the time. In structural equivalence models, the analytical frame of reference shifts from dyad to social system, and the process responsible for social influence shifts from communication within a primary group to competition and relative deprivation within a status (Burt 1982, chaps. 5–6).

With respect to innovation adoption, who adopts is still important. However, adoption by people in other statuses—people above, below, and apart from ego—do not matter in ego's evaluation of innovation adoption, regardless of the frequency and empathy of ego's communication with them. Their adoption might begin to make ego nervous about his own adoption inasmuch as they indicate to ego that he will soon have to resolve his own evaluation of the innovation, but the trigger to ego's adoption is adoption by the people with whom he jointly occupies a position in the social structure, the people who could replace him in his role relations if he were removed from the social structure. It is here where feelings of envy, relative deprivation, and advantage are felt, and it is here where the interpersonal synapse is fired. Thus, ego can enjoy the luxury of paying little attention to information about the innovation until diffusion reaches his status. Once the occupants of his status begin adopting, ego is expected to follow suit rapidly in order to avoid the embarrassment of being the last to espouse a belief or practice that has become a recognized feature of occupying his status.[5]

Formal Theory

Of the alternative ways to derive the predictions of social contagion by cohesion versus structural equivalence, there are advantages to beginning in psychophysics. Suppose, for a moment, that the desirable qualities affected by adopting an innovation could be measured quantitatively in one dimension. Let t_j be the discernible level of those resources held by some person j, ego in the above discussion. Empirical evidence from psychophysics indicates that ego's subjective perception of these resources, u_j, can be described in many circumstances by the following power function of the discernible resources he in fact has: $u_j = \mu t_j^{\nu}$, where μ and ν are parameters describing anyone in the study population making this evaluation (see Stevens 1957 and 1962 for illustrative review). Ego's

[5] The driving force of relative deprivation in innovation adoption is discussed in detail elsewhere, with numerical illustration for the formal models to be presented (Burt 1982, pp. 198–211).

evaluation of the advantages to be had by adopting the innovation is a function of the rate at which subjective perception would increase with an actual increase:

$$du_j/dt_j = \nu\mu t_j^{(\nu-1)} = \nu u_j/t_j.$$

Note the marginal nature of this evaluation. Perceived advantage is contingent on the current level of resource. With ν greater than one, as seems likely (see, e.g., Hamblin 1971), the perceived advantage of adopting would be small for people already holding high levels of the resource increased by adoption.[6]

The experimental evidence supporting this formulation is obtained from people in isolation. Suppose that the marginal evaluation in the above derivative is extended in a social situation to include those people who provide a frame of reference for ego's perceptions. Beginning with a simple, additive linear form, ego's evaluation of adoption's advantages could be expressed as follows:

$$dU_j/dt_j = b_p(\nu u_j/t_j) + b_s(\Sigma_i\, w_{ji}u_j/t_i),$$

where w_{ji} is a fraction ($w_{jj} = 0$, $0 \leq w_{ji} \leq 1$) expressing the extent to which person i defines the social frame of reference for ego's evaluation. Given network data on the study population, one set of w_{ji} could be defined to measure cohesion and another set to measure structural equivalence. The first term in parentheses in this expression is ego's personal evaluation, given above as du_j/dt_j. The second term in parentheses is his social evaluation, generated by ego asking himself how advantageous adoption would be (defined by $\nu u_j/t_i$) if he were each other person i socially significant to his evaluation (defined by w_{ji}). The coefficients b_p and b_s express the relative importance of personal and social factors in ego's overall evaluation.

This equation has to be stated in cruder terms before it can be used to guide empirical research on innovation diffusion. The unidimensional resource t_j affected by innovation is in fact an unknown mixture of empirical circumstances in ego's life. Any effort to measure such a quality with current methods and concepts seems at best capricious. For the purposes of this study I operationalize the general equation with the following:

$$x_j = b_p(p_j) + b_s(\Sigma_i\, w_{ji}x_i) + e_j = b_p(p_j) + b_s(x_j^*) + e_j, \qquad (1)$$

[6] Cancian (1967; 1979, p. 12) elaborates this idea in his argument for recognizing the inhibiting effect of wealth on innovation: the greater ego's wealth, the less he has to gain by running the risk of adopting a bad innovation. Cross-cutting the negative effect of wealth on innovation against the fact that wealth makes it easier to innovate, Cancian identifies a well-documented, middle-class conservatism in agricultural innovation (see Homans [1961], pp. 349–55, for a similar cost-benefit analysis predicting leaders to be innovative and the middle class to be conservative).

where x_j is ego's response to an innovation, p_j is some mixture of personal background variables significant in determining ego's response (vu_j/t_j in the above equation defining dU_j/dt_j), e_j is a residual term, and x_j^* is ego's adoption norm, the response expected of ego based on the responses of people defining the social frame of reference for his evaluation. For example, I will present results in which x_j is the date on which j adopted (measured in months after the innovation was available) and x_j^* is the date on which j's alters as a group adopted. In many circumstances, the betas in equation (1) can be estimated as parameters in a network autocorrelation model (see App.). To the extent that social contagion affects ego's response to the innovation, observed adoption will be strongly associated with normative adoption, making b_s significantly greater than zero.

I have two reasons for taking this theoretical route to equation (1). First, it clarifies the way in which the model to be applied in this study is grounded in more general theory. Integrating dU_j/dt_j yields a general model to study the association between subjective evaluation and social structure (Burt 1982, pp. 178–85). Taking partial derivatives of that model with respect to t_i opens an avenue to study the social structural conditions responsible for feelings of envy and relative deprivation (see, e.g., Burt 1982, pp. 191–98). Conclusions reached here on the relative merit of cohesion and structural equivalence thus have clear implications for research well beyond the question of innovation adoption.

Second, my route to equation (1) highlights the fact that concrete social structural conditions themselves can be subjectively distorted by ego as he evaluates an innovation. The social frame of reference in which one kind of innovation is evaluated need not be the same as the frame for evaluating a different innovation. Given some concrete measure of ego's proximity in social structure to alter i, ego's subjective perception of that proximity can be described by the familiar power function μ (proximity j to $i)^\nu$, and the network weights in equation (1) can be written as follows:

$$w_{ji} = \frac{(\text{proximity } j \text{ to } i)^\nu}{\Sigma_k \, (\text{proximity } j \text{ to } k)^\nu}, \; k \neq j, \tag{2}$$

where the summation is across everyone in the study population, excluding ego. The extent to which ego is conservative in relying on others is given by the magnitude of the exponent ν. Values of ν much larger than one indicate that ego's evaluation of the innovation under study is affected only by his closest confidants (cohesion) or his nearest rivals (structural equivalence). Small, fractional values of ν indicate that the evaluation is affected by almost anyone with whom ego communicates (cohesion) or shares mutual acquaintances (structural equivalence). In other words, ν defines the scope of the social frame of reference for ego's evaluation, with high values of ν indicating that only the closest alters are

pertinent. The high values of ν obtained for the *Medical Innovation* physicians (see App.) indicate that contagion operated only over very short distances between the physicians.

Cohesion and structural equivalence models of social contagion in equation (1) can be tested by manipulating the way in which proximity is measured for equation (2). If proximity is measured by the frequency and empathy of j's communication to i, then w_{ji} operationalizes cohesion, and x_j^* is the normative response expected from ego reflecting the adoption behavior of the people with whom he discusses things such as the innovation. If proximity is measured by the similarity in each person's relations with j and i, then w_{ji} operationalizes structural equivalence, and x_j^* is the normative response expected from ego reflecting the adoption behavior of the people who jointly occupy his status in the social structure of the study population. The associations between x_j and the alternative definitions of x_j^* indicate the extent to which social contagion had an effect on innovation diffusion and the extent to which it was driven by cohesion versus structural equivalence. The network data used to define the w_{ji} for this study are described below in the review of the *Medical Innovation* study, with technical details given in the Appendix.

Before I describe the *Medical Innovation* data, it is worth noting that the social structure of a study population determines the power of empirical research testing cohesion against structural equivalence. As illustrated in figure 1, the two network concepts lead to identical predictions in certain social structures (ego and alter in fig. 1*A*). A study population composed of cohesive groups of structurally equivalent people—for example, a population of unconnected cliques—cannot be used to distinguish the two network models because both models predict contagion within such groups. It is only where relationships cut across statuses (figs. 1*B*, 1*C*) that the contagion predictions of cohesion can differ from those of structural equivalence—not will differ but *can* differ. The magnitude of their difference is an empirical question. Thus, past empirical support for cohesion's effect on attitudes and behavior carries no implication of rejecting structural equivalence. Depending on the social structure of the populations selected for study, past support could just as well have been structural equivalence effects misinterpreted as cohesion effects. Such is the case in *Medical Innovation*.

THE *MEDICAL INNOVATION* STUDY

Coleman et al.'s (1966) *Medical Innovation* is a description of the manner in which a new antibiotic found acceptance during the mid-1950s among selected physicians in the Midwest. The study population was confined to a small geographic area so that physicians could be studied in the context

of their professional relations with colleagues. Because of a variety of practical constraints, four Illinois cities were selected as research sites: Peoria, Bloomington, Quincy, and Galesburg.[7] The study focused on the physicians especially likely to find tetracycline useful: general practitioners, internists, and pediatricians. There were 148 such physicians in the four cities, and interviews were completed with 126 (85%) of them. An additional four physicians, listed as having other specialties, turned out to have essentially a general practice, so they were added to the sample, bringing the total to 130 physicians. Each was asked in a personal interview whether he had ever used the new antibiotic and then was asked follow-up questions on when he became aware of it and what sources provided him with information on it.[8]

The drug selected for study, tetracycline (discussed in the study under the name "gammanym"), was well suited to revealing evidence of social contagion for the following reasons: (*a*) It could only be obtained by prescription, so that any physician adopting the new drug had to leave a written record of his adoption. (*b*) It was argued to be useful for a wide variety of conditions, so that it could have found "almost daily use by a physician in general practice" (Coleman et al. 1966, p. 17). (*c*) It was a powerful drug, especially useful in acute conditions, so that its virtues could be quickly determined and spread by word of mouth. (*d*) There were few alternatives to the new antibiotic, so that a physician who did not prescribe tetracycline was unlikely to be prescribing some other drug as a substitute. The new antibiotic was released in 1953 and gained widespread acceptance by the completion of fieldwork late in 1954. Physicians could have begun prescribing tetracycline at any time after its re-

[7] These were not major urban centers (Coleman et al. 1966, p. 192): "All four of these cities are somewhat industrialized and are surrounded by rich farming areas. The largest of the cities had a population of over 100,000 and had 182 physicians in active practice at the time of interviewing. It contained two hospitals offering residencies and a third hospital that offered no residencies. The other three cities had populations varying from 30,000 to 40,000; each contained between 45 and 75 physicians in active practice, and two or three hospitals, none of which offered residencies."

[8] A dummy variable of recalled adoption was constructed from these data ("1" if a physician recalled adopting tetracycline, "0" otherwise), but the variable reveals no evidence of contagion, so results obtained with it are not presented, under pressure to conserve space. In addition to eliciting data on recalled adoption, the personal interviews covered diverse topics, including the respondent's social and professional background, his attitudes toward the community and various medical practices, his own health habits (e.g., smoking), his medical practice, the information channels through which he kept up with medical developments, and sociometric data on his social and professional relationships. Portions of the survey instrument are reproduced in *Medical Innovation* (Coleman et al. 1966, pp. 195–205). Much of the data in the original study, and all the variables mentioned here, can be obtained on microcomputer diskette (see n. 1). Also contained on the diskette are the preference and network variables prepared for this study.

lease. Most were prescribing it but varied considerably in when they had first accepted it. The study was designed to answer why they began when they did.

The main conclusion advanced in *Medical Innovation* is that informal professional discussions between physicians created social contagion in tetracycline's diffusion, especially for physicians extensively involved in such discussions, and especially when the new antibiotic was released. I have three reasons for returning to this study: the adoption data, the network data, and the study's emergence as an exemplar for detecting social contagion.

A Behavioral Measure of Adoption

Beyond the usual retrospective adoption data in personal interviews, behavioral adoption data were obtained in the study. Records at the pharmacies filling the bulk of the tetracycline prescriptions for the study population were audited. Prescriptions written during three-day periods, separated by intervals of about one month, were audited for over a year following tetracycline's release.[9] The result was an adoption-date variable ranging from 1 to 17, roughly indicating the month after tetracycline's release in which a physician first began prescribing the new antibiotic. Within the time covered by the study, 16 physicians were nonadopters in that the prescription sampling turned up prescriptions that they had written but no tetracycline prescriptions. As did the original study, I use these physicians to define a final point in tetracycline's diffusion, category 18 on the adoption-date variable. They either never adopted tetracycline, or adopted it after the time period covered by the study, or wrote prescriptions for it on days not covered in the prescription sampling. Since they were exposed to the same risk of being missed in the sampling as the physicians whose tetracycline prescriptions were detected, either of the first two possibilities seems more likely than the third. In contrast, there were five physicians for whom no prescriptions were found, neither tetracycline prescriptions nor prescriptions for anything else. As in the original study, I have made no attempt to predict adoptions by these five physicians—leaving 125 physicians whose adoption date is defined by the prescription sampling and to be explained.

[9] The original study provides a detailed account of the prescription sampling (Coleman et al. 1966, pp. 193–94). Prescriptions were audited for sampling periods of three successive days at approximately monthly intervals. The three-day sample periods were stratified to occur on different days of the week, skipping Sundays and holidays, over the course of tetracycline's diffusion. The average interval between sampling periods was 28.5 days, defining 17 intervals for the adoption variable over the 16 months for which prescriptions were audited.

These adoption data should be much more reliable than the retrospective survey data typical of diffusion research, since they are based on behavioral traces rather than a physician's memory of more than a year's prescriptions. At the same time, the data are not a census. Prescriptions were only audited for three working days per month. It is quite possible for a physician to have begun prescribing tetracycline and have had his prescriptions filled during the days for which prescription records were not audited. However, such sampling errors could only occur in one direction. It is possible for a physician to have begun prescribing tetracycline earlier than the prescription data would indicate, but he had definitely begun prescribing by the time one of his prescriptions for it was located. In other words (and this will be important in aggregating contagion evidence), an unexpectedly late adoption could be a sampling error, but an unexpectedly early adoption could not.

Thorough Network Data on the Social Structure of Physicians

A second reason for returning to *Medical Innovation* is the thorough network data obtained for the study. A variety of data were obtained on social and professional relations, but choice data elicited by two sociometric items were the principal basis for evidence of social contagion in *Medical Innovation*. One item elicited the names of advisers ("When you need information or advice about questions of therapy where do you usually turn?"), and the other elicited the names of discussion partners ("And who are the three or four physicians with whom you most often find yourself discussing cases or therapy in the course of an ordinary week—last week for instance?"). These citations indicate the channels of informal professional advice and discussion among the physicians. They also indicate the importance of physicians outside the prescription sample. Fifty-two percent of the prescription-sample citations for advice and discussion went to individuals outside the prescription sample, and this figure does not reflect the extent to which relations from outside physicians stratified physicians within the prescription sample. To represent better the social context in which the prescription-sample physicians worked, interviews were conducted with an additional 98 physicians selected to represent the physicians, by specialty, who were most often cited as friends, advisers, and discussion partners by the prescription-sample physicians. The study design was successful. Only 18 physicians beyond the 228 contacted for the study were cited by two or more prescription-sample physicians; that is, 93% of the physicians involved in professional relationships with two or more of the prescription-sample physicians were interviewed for the study. The medical advice and discussion citations elicited from all 228 physicians have been pooled to

define a network of professional ties in each city.[10] These citation data have been used to compute network weights for cohesion and structural equivalence in each city. The network weights have been used to define the adoption expected of each prescription-sample physician—first, as a function of his advisers and discussion partners (cohesion) and, second, as a function of his position in the social structure of medical advice and discussion (structural equivalence). Computational details are given in the Appendix.

A Sociological Exemplar

The sociology of science provides a third reason for returning to *Medical Innovation*. The study has become an exemplar. The difficulty of obtaining behavioral adoption data and thorough network data, not to mention the skillful analysis of these data in the original study, have combined to make it unique in diffusion research. Research prior and subsequent to *Medical Innovation* provides a wealth of information on nonnetwork variables affecting innovation adoption and associations between aspects of network positions and recollections of adopting. Rogers (1983) continues to provide the encyclopedic synthesis of this research. Little new knowledge has emerged, however, on the manner in which social contagion operates; cohesion remains the assumed social force driving contagion, and *Medical Innovation* remains the classic evidential reference (see Rogers 1983, chap. 8, on diffusion networks in general and pp. 65–68 and 288–93 on *Medical Innovation* in particular). Thus, the original data are a strategic research site for testing new understandings of the social structural conditions responsible for contagion.

CONTAGION AT THE POPULATION LEVEL

I begin with the shape of diffusion. If social contagion was a dominant factor in tetracycline's diffusion, then the distribution of adoptions over time in the study population would have had an identifiable form. This is a long-standing theme in diffusion research (see, e.g., Pemberton 1936), a textbook exemplar in mathematical sociology (see, e.g., Leik and Meeker 1975, pp. 128–39), and a central point in *Medical Innovation,* with the rapid diffusion of tetracycline among prominent physicians advanced as

[10] This figure includes 12 physicians interviewed as informants for the study. Their interviews are not strictly comparable with those conducted with the other 216 study respondents. Analysis of advice and discussion as separate networks did not produce contagion effects different from those obtained with the simpler pooled network. Additionally, multiplexity with social relations and hospital affiliations and the social structure of advice and discussion in each city have been studied.

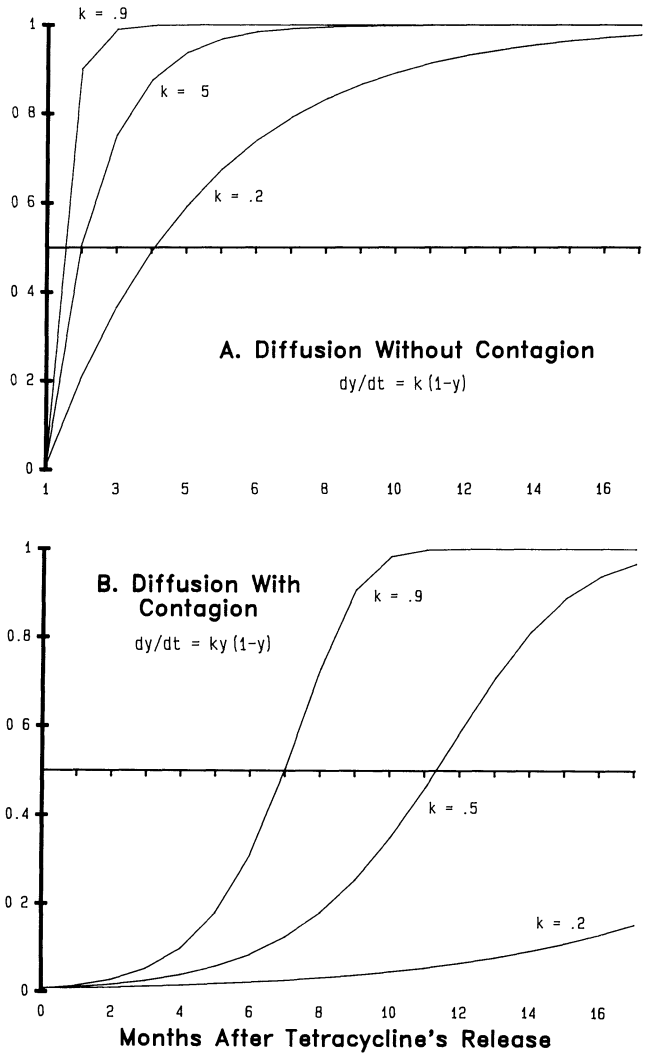

FIG. 2.—Diffusion in theory (cumulative proportion of physicians adopting over time)

evidence of social contagion (Coleman et al. 1966, pp. 95–111). The point is illustrated in figure 2, with hypothetical diffusion curves for the *Medical Innovation* population describing the cumulative proportion of physicians adopting tetracycline at each of the 17 roughly month-long sampling intervals covered by the study.

In the absence of contagion, tetracycline's diffusion would have re-

sembled the curves in figure 2*A*. Diffusion is driven by two factors: the average physician's predisposition to adopt independently of other physicians (probability k) and the proportion of the physician population available to adopt (one minus y, where y is the proportion that has adopted). The expected rate of tetracycline's diffusion is the product of the two factors: $dy/dt = k(1 - y)$. If the average physician's preference for the new drug was low, say a .2 probability, as in the bottom curve in figure 2*A*, then diffusion would have progressed very slowly: 21% would be expected to have adopted during the first month, 37% by the end of the second month, and so on across the diffusion process, ending with 98% adopting by the end of the time covered by the study.[11] Diffusion would have been much more rapid if each physician had been equally likely to adopt or reject the new drug (the middle curve in fig. 2*A*). If each physician had a strong predisposition toward adopting tetracycline, say a .9 probability of adopting, then diffusion would have progressed very rapidly, as illustrated by the top curve in figure 2*A*. Adoptions would have spread to 90% of the population during the first month and to 99% by the end of the second month, completing the bulk of tetracycline's diffusion only two months after its release.

Contagion changes diffusion to the familiar S-shaped curves in figure 2*B*. The rate of tetracycline's diffusion is given by the two factors above, weighted by the extent to which the innovation is already widely adopted: $dy/dt = ky(1 - y)$. If affected by contagion, use of tetracycline would have spread rapidly as a function of physicians' personal preferences for the new drug (k), the volume of physicians available to adopt ($1 - y$), and the volume of physicians who had already adopted (y). Thus, familiar distinctions emerge between stages in the diffusion process. Initially, few adoptions have taken place, so that social contagion dampens the rate at which diffusion occurs. Even with high personal preferences and a large proportion of potential adopters, a low proportion of actual adopters will keep new adoptions low. The product $ky(1 - y)$ is low while y is low. As adoptions seep through the system, more potential adopters are exposed to someone who has already adopted, so that the product $y(1 - y)$ approaches its maximum value of one-half. Diffusion is most rapid at this

[11] Several bold assumptions are required to legitimate these computations, including constant personal preferences independent of diffusion's progress (i.e., k independent of y) and constant rates of diffusion within monthly intervals (i.e., dy/dt constant in each of the 17 sampling intervals). I am not proposing that such assumptions are valid or invalid for the study population. I am merely using fig. 2 to recall the argument in *Medical Innovation* and to highlight the fact that social contagion is uniquely characterized by a slow initial rate of diffusion rather than a fast subsequent rate; this sets the stage for observing that there is no such evidence of social contagion in the observed physician population.

point and gradually slows down afterward as there are fewer and fewer people remaining to adopt (i.e., $ky[1 - y]$ decreases as $[1 - y]$ decreases).

I wish to note two differences in figure 2 between a diffusion process in which social contagion occurs and one in which it does not. First, a steep rate of diffusion need not indicate social contagion. Rapid diffusion can be generated by strong personal preferences toward adoption in a study population (top curve in fig. 2*A*) or by social contagion (middle and top curves in fig. 2*B*). Second, the most distinct evidence of social contagion is the initial period of slow diffusion among pioneer adopters. Diffusion driven by personal preference (fig. 2*A*) begins as a rapid rate, which becomes slower and slower. Diffusion driven by social contagion (fig. 2*B*) begins at a slow rate, which increases until half the population has adopted and thereafter becomes slower and slower. In fact, social contagion coupled with low physician preferences for adoption—the bottom curve in figure 2*B*—could have slowed tetracycline's diffusion down to the point at which only 16% of the study population would have adopted by the end of the study period.[12]

I note these points in figure 2 because the curves in figure 3 describing tetracycline's observed diffusion show no evidence of the slow initial diffusion characteristic of social contagion. The bold lines in figures 3*A* and 3*B* trace the cumulative proportion of the study population adopting at each of the sampling intervals. Note that there is no delay in tetracycline's diffusion; it spread quickly and progressed at a diminishing rate. As illustrated in figure 2, this diffusion could be due to social contagion or strong personal predispositions among the physicians toward adoption. The slow initial diffusion characteristic of social contagion is missing altogether. Moreover, the same conclusion holds for physicians who are either prominent or marginal in the networks of medical advice and discussion. In figure 3*A*, physicians cited by four or more others as an adviser or discussion partner adopted at a faster rate than physicians not

[12] The diffusion curves in fig. 2 begin with the first physician's adoption ($y_0 = 1/125$). The form of the diffusion curves produced by personal preference is stable over a reasonable range of alternative starting proportions, but the curves produced by social contagion are quite sensitive to the number of physicians initially adopting tetracycline. E.g., if diffusion were said to have begun with 10 physicians adopting, then y_0 would be 10/125, and the contagion curve in fig. 2*B* with $k = .2$ would predict adoption by 70% of the study population by the end of time period covered by the study rather than the 16% in fig. 2*B*. In fact, one way to speed contagious diffusion through its initially slow rate is to seed the population with a small number of initial adoptions. Nevertheless, contagion would have had its characteristic effect on diffusion curves in the *Medical Innovation* study population. Even if the curves in fig. 2*B* are begun with the 8.8% of the study population adopting by the end of the first month, they still show the characteristic slow initial diffusion phase, but it is brief for physicians strongly predisposed toward adopting the new antibiotic.

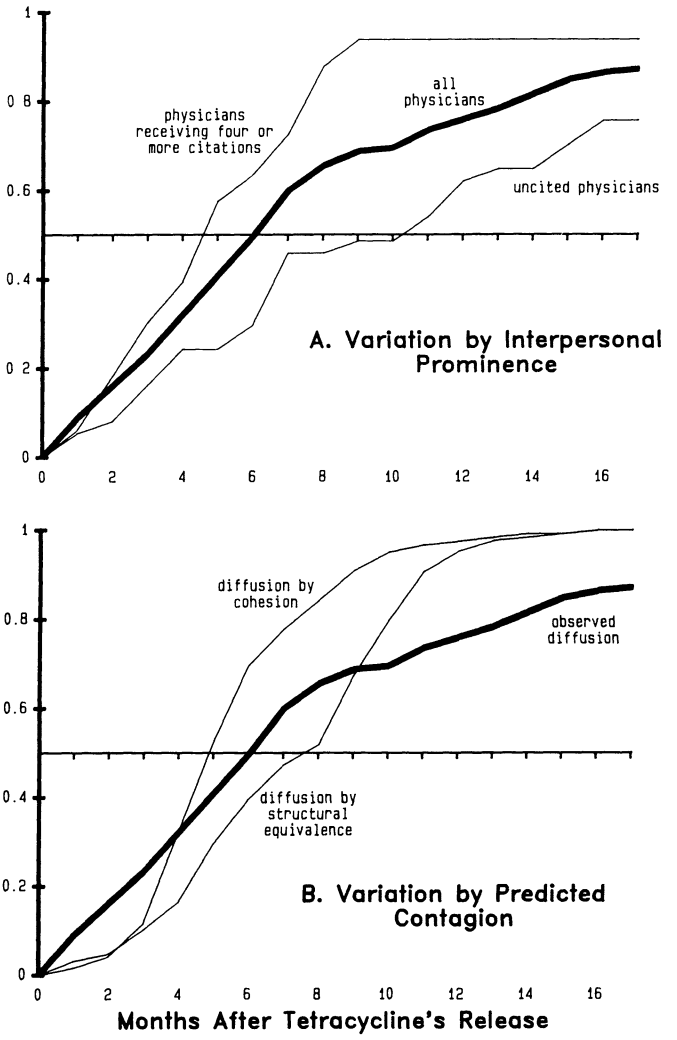

FIG. 3.—Diffusion observed (cumulative proportion of physicians adopting over time)

cited by anyone. However, diffusion in both subpopulations began quickly and progressed at a constant or diminishing rate.

It seems clear that contagion was not the principal factor driving tetracycline's diffusion. The slow initial diffusion characteristic of contagion is missing, and the steep rate of diffusion observed is evidence of both contagion and strong personal predispositions toward adoption—where

there is evidence of contagion, there is simultaneously evidence of personal preferences at work.[13]

[13] This conclusion can be stated more precisely with a diffusion model developed in marketing research to describe the spread of new products. I am grateful to Donald Lehmann for calling my attention to this work. Mahajan and Peterson (1985) provide a brief review of the work, and the papers assembled in Mahajan and Wind (1986) provide detailed discussion. The model is useful here because it distinguishes personal and contagion components in population diffusion curves. The basic model proposed by Bass (1969) defines the number of people expected to adopt in time interval t (see Mahajan and Wind 1986, p. 6):

$$dN(t)/dt = [p + (q/m)N(t)]\,[m - N(t)],$$

where $N(t)$ is the cumulative number of individuals adopting by time t, m is a diffusion "ceiling" equal to the number of individuals who will eventually adopt, p is a "coefficient of innovation" describing the tendency for individuals to adopt before anyone else has adopted (note that the equation reduces to pm when $N(t) = 0$), and q is a "coefficient of imitation" describing the tendency for individuals to adopt as others adopt. This model can be restated in terms of the factors familiar to sociologists from Coleman et al.'s analysis (see Bass 1969, pp. 217–18): $dy/dt = (p + qy)(1 - y) = p(1 - y) + qy(1 - y)$, where y is the cumulative proportion of adopters who adopted by time t (i.e., $y = N(t)/m$). In other words, the marketing model disaggregates the average adoption probability in fig. 2, k, into a personal component and a social component. The $p(1 - y)$ term is the personal component displayed in fig. 2*A* and the $qy(1 - y)$ term is the contagion component displayed in fig. 2*B*. To estimate the magnitude of these components in an observed diffusion curve, one can integrate the partial derivative dy/dt and compare adoption frequencies in successive time intervals. The number of adoptions in time interval t, $dN(t)$, is the difference $N(t) - N(t - 1)$, which equals the difference $m(y_t) - m(y_{t-1})$, which, when y is replaced with the results of integrating dy/dt, yields an equation with which the unknown parameters m, p, and q can be estimated (see Srinivasan and Mason 1986):

$$dN(t) = \frac{m[1 - e^{-(p+q)(t)}]}{[1 + (q/p)e^{-(p+q)(t)}]} - \frac{m[1 - e^{-(p+q)(t-1)}]}{[1 + (q/p)e^{-(p+q)(t-1)}]}.$$

I have estimated the parameters from adoption frequencies in the 17 sampling intervals by using the nonlinear, least-squares algorithm in SYSTAT, the ceiling initially being set to a single adopter ($m = 1$) and the adoption probabilities being set to their maximum value ($p = q = 1$), which forces the algorithm to compute derivatives across distant alternatives before reaching the final estimates. For the bold line in fig. 3 describing all prescription-sample physicians, the estimated personal component, p, is .081 and 5.4 times its standard error. The estimated social component, q, is .207 and 2.4 times its standard error. Stronger evidence of contagion can be found in the fig. 3*A* curve describing the spread of tetracycline among socially prominent physicians. The estimate of q increases to .409, which is 3.5 times its standard error. However, personal preferences remain a highly significant factor. The estimate of p for the socially prominent physicians is .061 and 2.8 times its standard error. As was concluded in the original study, there is no evidence of contagion in the fig. 3*A* curve describing tetracycline's diffusion among socially marginal physicians. The estimate of q is .102, which is less than its standard error and quite obviously less significant than the personal component, p, estimated to be .057 and 1.9 times its standard error. Thus, I reach the conclusion stated in the text: where there is evidence of contagion in tetracycline's diffusion, there is simultaneously evidence of personal preferences at work.

The missing characteristic evidence of social contagion is more obvious in figure 3*B*, where the observed population curve is plotted with corresponding plots of adoption norms under cohesion and structural equivalence. Continuous adoption norm variables have been rounded to integers to define the month in which a physician should have adopted under structural equivalence or cohesion. The cohesion curve describes what diffusion would have looked like if physicians had adopted when their advisers and discussion partners adopted. The structural equivalence curve describes diffusion under the assumption that physicians adopted when their peers in the medical hierarchy adopted. In contrast to the observed diffusion curves, both network models produce diffusion curves obviously influenced by social contagion, in the sense that both describe tetracycline diffusing at a slow rate for the first few months after its release, spreading rapidly in bandwagon fashion after a handful of physicians made the first, tentative adoptions, and slowing rapidly to complete diffusion throughout the study population. In other words, both network models differ from the observed data by predicting diffusion curves dominated by contagion effects and evidencing the slow initial diffusion characteristic of contagion.[14]

To summarize, there is evidence of contagion in tetracycline's diffusion at the same time that contagion was far from the dominant factor driving the new drug's adoption. These results provide a useful indication of the magnitude of contagion's effects. They glaringly fail to indicate how contagion took place. Contagion remains an assumed process at this level of analysis, typically attributed to cohesion and isolated from empirical testing. To study the social structural conditions responsible for the apparent contagion effects in tetracycline's diffusion, I have to examine the interpersonal environments in which individual adoptions occurred.

CONTAGION AND THE INDIVIDUAL PHYSICIAN

The network data on advice and discussion relations make it possible to dig past the population level of analysis down to the level of social contagion's effect on the individual physician's adoption. To begin with, it is

[14] This conclusion, too, can be stated more precisely with the marketing research model given in the preceding footnote. For the diffusion curve predicted by structural equivalence, the estimated personal component in a marketing diffusion model, p, is .018 and 2.3 times its standard error, while the estimated contagion component, q, is .414 and 5.8 times its standard error. For the curve predicted by cohesion, the estimate of p is .013 and 2.6 times its standard error, while the estimate of q is .933 and 8.0 times its standard error. Thus, I reach the conclusion stated in the text: the cohesion and structural equivalence curves in fig. 3*B* illustrate diffusion processes dominated by contagion.

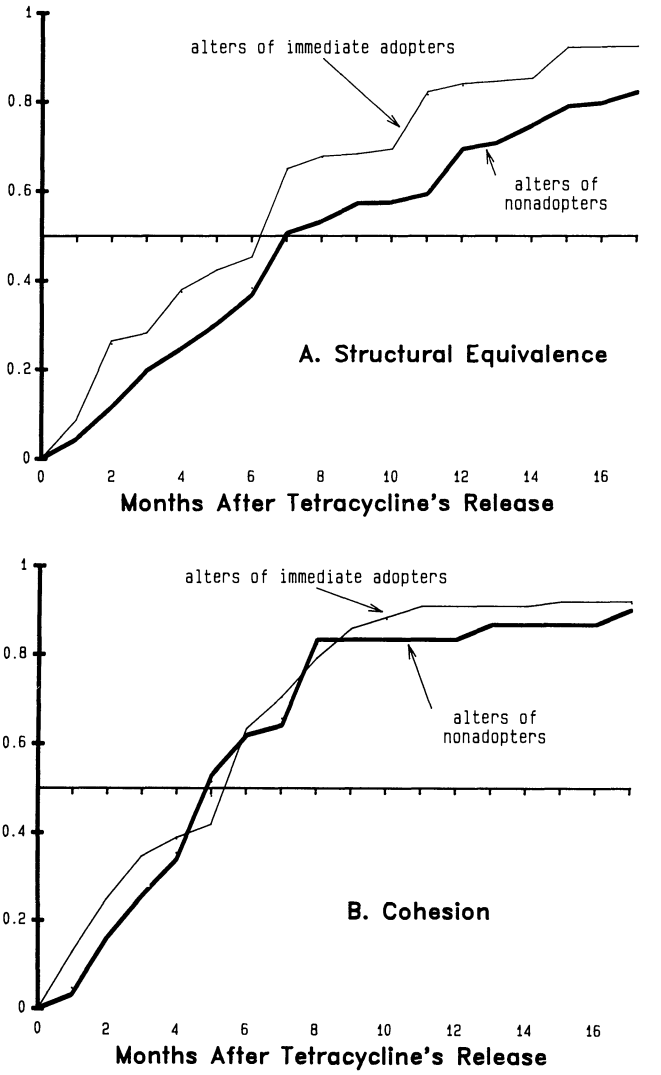

FIG. 4.—Alter adoptions for physicians adopting extremely early or extremely late (cumulative proportion of alters adopting over time).

possible to compare physicians in terms of tetracycline's diffusion among each physician's advisers and peers. In contrast to the plots in figures 2 and 3 of the cumulative proportion of physicians adopting over time, figure 4 plots the cumulative proportion of alters adopting. The alters of physicians adopting immediately (within the first two months of tetracycline's diffusion, during the slow diffusion predicted in fig. 3*B* by cohe-

sion and structural equivalence) are compared with the alters of physicians who had not adopted by the end of the study period.

It is clear from these graphs that physicians at the very beginning and very end of tetracycline's diffusion were exposed to similar adoption rates among their alters. Physicians adopting immediately were not surrounded by alters who also adopted immediately. On average, it took six months for more than half of their advisers and discussion partners to adopt (fig. 4*B*) and another month for more than half of their structurally equivalent alters to adopt (fig. 4*A*). Similarly, physicians who delayed adoption beyond the time period covered by the study were not surrounded by alters who adopted late.

At the same time, it is clear that there is more difference between the structural equivalence alters. Under structural equivalence, the alters of immediate adopters were at all times during tetracycline's diffusion more likely than the alters of nonadopters to adopt the new drug. The thin line in figure 4*A* is higher than the thick line. In contrast, there is no significant difference at any time between the cohesion alters of immediate adopters and nonadopters, and there are even times when adoptions were higher among the nonadopter alters. The thin line and thick line are intertwined in figure 4*B*. The visible difference in figure 4 between structural equivalence and cohesion is repeated in the more systematic results to follow.

The comparison in figure 4 between immediate and late adopters is extended in table 1 to all physicians. The results consistently support the conclusion that a physician's adoption was strongly determined by the behavior of his peers in the medical hierarchy (the structural equivalence column in table 1) and virtually unaffected by the behavior of the people from whom he sought advice or with whom he discussed cases (the cohesion column in table 1).

Regressing observed adoption date over the adoption dates predicted by structural equivalence and cohesion is the most obvious way to look for a social contagion effect. The results are reported in the first row of table 1. The month in which a physician began prescribing tetracycline is significantly predicted by the month in which people structurally equivalent to him began prescribing it and statistically independent of the month in which his advisers and discussion partners began prescribing it. More specifically, physicians, on average, began writing tetracycline prescriptions 3½ months after its release if the physicians with whom they were structurally equivalent adopted the new drug immediately; and they postponed their own adoption for a little more than half a month for every month that their alters delayed adopting (i.e., the .32 standardized coefficient in table 1 refers to a metric regression line with a 3.50 intercept and .58 slope).

TABLE 1

EVIDENCE OF CONTAGION IN ADOPTION

	Structural Equivalence	Cohesion
Continuous contagion effect[a]	.32	.07
	(t = 3.8)	(t = 0.7)
Independence of detailed diffusion phases[b]	68.78	29.56
	($P < .001$)	($P = .24$)
Independence of aggregate diffusion phases[c]	29.83	4.92
	($P < .001$)	($P = .30$)
Contagion effect in innovation roles[d]	1.29	.95
	(z = 2.59)	(z = −.54)
	(P = .005)	(P = .71)

[a] Standardized, ordinary least-squares estimates obtained by regressing month of adoption over normative month of adoption for the prescription-sample physicians (see App. on computing adoption-month norm). Structural equivalence adoption norms are available for 124 physicians, and cohesion norms are available for 117 physicians. Routine t-tests are presented to provide some sense of effect magnitude relative to residual variance; however, routine statistical inferences should not be made from these tests (see App.). If dates are standardized by city means and standard deviations, effects of .32 and .02 are obtained for structural equivalence and cohesion, respectively.

[b] Likelihood-ratio χ^2 statistics are reported, but inferences is difficult because of the many low frequencies in these detailed tables (see table 2). The six categories of observed and normative adoption date in table 2 create 25 df for these statistics.

[c] Likelihood-ratio χ^2 statistics are reported. As described in the text, observed and normative adoption date data are tabulated across aggregate phases in the diffusion of tetracycline (early, median, and late adopters), creating 4 df.

[d] The effect is the multiplicative interaction between observed and normative adoption in a log-linear model of the innovation roles in table 3. It equals the number of physicians conforming to alter behavior (early and late conformers) divided by the number of physicians deviating from alter behavior (eager innovators and deviant laggards), quantity to the fourth root, and so measures the tendency for physicians to conform to alter behavior.

These estimates of a continuous social contagion effect presume equal intervals between months, but the months of tetracycline's diffusion, equivalent in physical time, were not equivalent in social time. There are four months separating an adoption in the third month from an adoption in the seventh month of tetracycline's diffusion and four months separating an adoption in the thirteenth month from an adoption in the seventeenth month. However, the first difference has greater social significance than the second difference. As illustrated by the population diffusion curves in figures 2B and 3B, the first difference separates a physician who adopted during the early phase of tetracycline's diffusion—when adop-

tions were few and tentative—from a physician who adopted during the middle phase of the new drug's diffusion—when there was a great rush of physicians adopting the drug and several months of collective experience with the new drug. In other words, the four-month gap between adoptions in months three and seven spans socially distinct phases of tetracycline's diffusion. In contrast, and as again illustrated in figures 2*B* and 3*B*, the four-month difference between adoptions in months 13 and 17 is a negligible difference between adoptions during the final phase of tetracycline's diffusion, a period of very few adoptions because most of the study population had already adopted the new drug. The distinction between physical and social time suggests that it is improperly precise to evaluate contagion by the tendency for physicians and alters to have adopted tetracycline in the same month after its release. Rather, contagion should be estimated in terms of the tendency of physicians and alters to have adopted tetracycline during the same phase of its diffusion.[15]

The results in the second, third, and fourth rows of table 1 are based on alternative partitions of adoption months into diffusion phases. All support the conclusion that observed adoption was contingent on structural equivalence norms and independent of cohesion norms. For each of the three adoption variables (observed date of adoption, structural equiva-

[15] In addition, there are two important methodological reasons for estimating social contagion effects from aggregate categories of adoption dates. First, the ordinary least-squares estimates of a continuous social contagion effect in the first row of table 1 are not maximum likelihood because of variable correlations between the residuals in predicting observed from prescribed adoption. Contagion is more properly estimated as a network autocorrelation, and that, unfortunately, cannot be estimated here because of problems with missing data (see App.). In other words, one methodological reason for aggregating adoption dates is to recode autocorrelations between monthly response categories into intracategory correlations to facilitate statistical inference. Second, there is the question of how to put the small *Medical Innovation* sample to best use in studying social contagion. In order to study the form of social contagion's effect, i.e., the form of the association between observed and normative adoption, the small sample of physicians can be distributed across the cells in table 2, created when six categories of observed adoption dates are tabulated within six categories of adoption norms. But note the small frequencies in table 2. Cross-tabulating table 2 across third variables measuring personal preferences or social structural conditions further lowers cell frequencies and creates more empty cells, making estimates of effects unreliable. Aggregating adoption dates into diffusion phases increases cell frequencies in three-way tabulations. This is a second reason for aggregating adoption dates into diffusion phases: to shift analytical power from making statements about the form of social contagion to making statements about the stability of social contagion across variations in personal preference and social structural conditions. Of course, this aggregation is legitimate only if the original evidence of social contagion is preserved. Therefore, I present estimates of social contagion in table 1 for alternative aggregations to demonstrate that the relative strength of structural equivalence over cohesion is reproduced at each level of aggregation.

TABLE 2

CATEGORIES OF OBSERVED AND NORMATIVE ADOPTION DATES

OBSERVED DATE OF ADOPTION	ADOPTION DATE NORM AMONG ALTERS					
	1	2	3	4	5	6
Early adopters:						
1	7	3	6	4	2	7
	(5)	(3)	(4)	(2)	(5)	(8)
2	3	5	2	1	0	1
	(3)	(1)	(1)	(3)	(2)	(2)
Median Adopters:						
3	6	3	5	6	0	1
	(5)	(2)	(4)	(3)	(1)	(5)
4	10	0	3	1	1	6
	(8)	(4)	(3)	(3)	(2)	(1)
5	4	0	1	0	1	4
	(4)	(0)	(0)	(0)	(0)	(5)
Late adopters:						
6	0	0	5	9	6	11
	(6)	(1)	(6)	(6)	(3)	(6)

NOTE.—Frequencies for alters defined by cohesion are presented in parentheses beneath frequencies for alters defined by structural equivalence. Categories are defined by ranking physicians in each city by adoption date and aggregating adjacent physicians. Within the limits of ties between physicians adopting during the same month, category 1 contains the first 25% of physicians adopting in each city, category 2 contains subsequent adopters up to the first 33% in each city, category 3 contains subsequent adopters up to the first 50% in each city, category 4 contains subsequent adopters up to the first 66% in each city, category 5 contains subsequent adopters up to the first 75% in each city, and the remaining 25% of physicians, consisting of very late adopters and the nonadopters in each city, fall into category 6.

lence date, and cohesion date), phases in tetracycline's diffusion have been defined by ordering physicians in each city by adoption date and aggregating adjacent physicians. Table 2 presents the distribution of physicians across six adoption categories defining three and four phases in tetracycline's diffusion. For each city, categories 1 and 2 contain the first third of physicians adopting, categories 3 and 4 contain the second third, and categories 5 and 6 contain the last third (including physicians for whom no tetracycline prescriptions were found). Alternatively, category 1 contains the first quarter of physicians adopting, categories 2 and 3 contain the second quarter of adopters, categories 4 and 5 contain the third quarter of adopters, and category 6 contains the final quarter. Note two things. First, the frequencies are very low in this table. The data must be aggregated into broader diffusion phases in order to study effects in tables with additional variables. Second, even at this level of detail, the χ^2 statistics in the second row of table 1 show that observed adoption is strongly associated with structural equivalence (less than a .001 probability of independence) and is statistically independent of cohesion (.24 prob-

ability of independence). Three aggregate phases in tetracycline's diffusion are distinguished in table 2: early, median, and late.

The first third of a city's physicians adopting the new drug were early adopters. These 41 physicians adopted during the first four months of tetracycline's diffusion in each city[16]—a period when adoptions were rare and tentative (as illustrated in fig. 3*B;* see Coleman et al. 1966, p. 32) and the only period in which evidence of interpersonal influence from advisers and discussion partners was observed in the original study.[17] Further, there is no significant difference in the adoption norms to which physicians in the first two rows of table 2 were exposed (4.88 and 2.94 χ^2 statistics with 5 *df* for structural equivalence and cohesion, respectively).

The final fourth of a city's physicians remaining after the city's other physicians had begun prescribing tetracycline were late adopters. Half of these 31 physicians were nonadopters in that they wrote none of the sampled tetracycline prescriptions. The other half of the late adopters began prescribing tetracycline a year or more after the new drug was released. As illustrated in figure 3, the rate of tetracycline's diffusion during this period was very slow, almost nonexistent, except among the

[16] One early adopter, the last in Galesburg, adopted during the fifth month, but tetracycline did not begin diffusing among the sampled Galesburg physicians until the third sampling period, so that this last adoption is well within the first four months of tetracycline's diffusion within Galesburg.

[17] Interpersonal influence from advisers and discussion partners was reported in *Medical Innovation* for the first five months of tetracycline's diffusion (Coleman et al. 1966, pp. 114–30). This suggests that the lack of a cohesion effect in table 1 might be a consequence of estimating contagion across the entire time period covered by the study rather than focusing on the first five months when cohesion had its effect. Such is not the case. The evidence of interpersonal influence reported in the original study depends on censoring the adoption data and is duplicated here if the adoption data are similarly censored. Answering the question of when networks had their effect, Coleman et al. (1966, pp. 117–20) estimated interpersonal influence by comparing the date on which a physician adopted with the dates of adoption by his advisers and discussion partners who had already adopted. Advisers and discussion partners adopting later, or not adopting at all, were deleted from the estimation. Thus, variation in the adoption behavior of physicians adopting early was censored, giving the appearance of simultaneous adoptions early in tetracycline's diffusion. The later the month was in which a physician adopted, the greater the acknowledged variation in the dates on which advisers and discussion partners adopted and the lower the likelihood of finding evidence of contagion. Similarly, the date on which an early adopter began prescribing tetracycline is strongly predicted here by structural equivalence and cohesion if physicians whose alters adopted later are ignored ($r = .72$, 4.16 t-test for 18 early adopters under structural equivalence; $r = .56$, 2.16 t-test for 12 early adopters under cohesion). However, expanding the calculations to all 41 early adopters regardless of when their alters adopted shows that there is no association between the month in which an early adopter began prescribing and the month in which his alters, on average, began prescribing ($r = .06$ for structural equivalence; $r = .03$ for cohesion).

physicians not cited as advisers or discussion partners by anyone in the study.[18]

Finally, physicians adopting during the intervening months of tetracycline's diffusion were median adopters. As illustrated in figure 3, these adoptions occurred at a time when tetracycline was spreading rapidly, the principal shift occurring during the tenth month to a slower spread of the new drug among physicians generally. In addition, there are no significant differences in the adoption norms to which physicians in rows 3, 4, and 5 of table 2 were exposed (15.66 and 11.65 χ^2 statistics with 10 *df* for structural equivalence and cohesion norms, respectively).

The results in the third row of table 1 show that the distinction between early, median, and late phases in tetracycline's diffusion preserves the strong evidence of contagion predicted by structural equivalence and continues to reveal no evidence of contagion by cohesion. The hypothesis of no contagion effect has less than a .001 probability of being true in structural equivalence predictions and a .30 probability of being true in cohesion predictions.

Enriching these results, the graphs in figure 5 show that the contagion effect predicted by structural equivalence operated continuously over the entire course of tetracycline's diffusion. The figure presents the proportion of adopters in each month who had early adopters as alters, median adopters as alters, and late adopters as alters.[19] Consider the physicians who began writing tetracycline prescriptions four months after its release. Under structural equivalence (fig. 5*A*), 64% of these physicians had alters adopting early in tetracycline's diffusion, 27% had alters adopting during the median phase, and 9% had alters adopting late or not adopting at all. In other words, physicians adopting during the fourth month tended to have alters who also adopted early. In the same graph, notice how the tendency for early adopters to have had alters adopting early shifts smoothly to late adopters exposed to alters who adopted late. Contrast this with the graph for cohesion norms in figure 5*B*. There is no shift from early to late adoption among the alters. Physicians adopting at the beginning and end of tetracycline's diffusion were exposed similarly to alters

[18] Categories 5 and 6 are not combined to define the last 33% of physicians as late adopters because there is a significant difference in the kinds of adoption norms to which the late adopters (category 6) and late median adopters (category 5) were exposed. The hypothesis that observed and normative adoption are independent in rows 5 and 6 does not fit the data ($\chi^2 = 12.59$, with 4 *df* for structural equivalence, $P = .01$, and the usual negligible result with cohesion: $\chi^2 = 7.01$ with 5 *df*).

[19] In order to highlight trends over time in fig. 5, moving averages are plotted. The proportion of physicians at time t exposed to alters adopting early, e.g., is the average of the proportion exposed to early adopting alters at time $t - 1$, t, and $t + 1$. For this figure, the proportions observed at the end of the first month have been extended backward to time 0, the initial release of tetracycline.

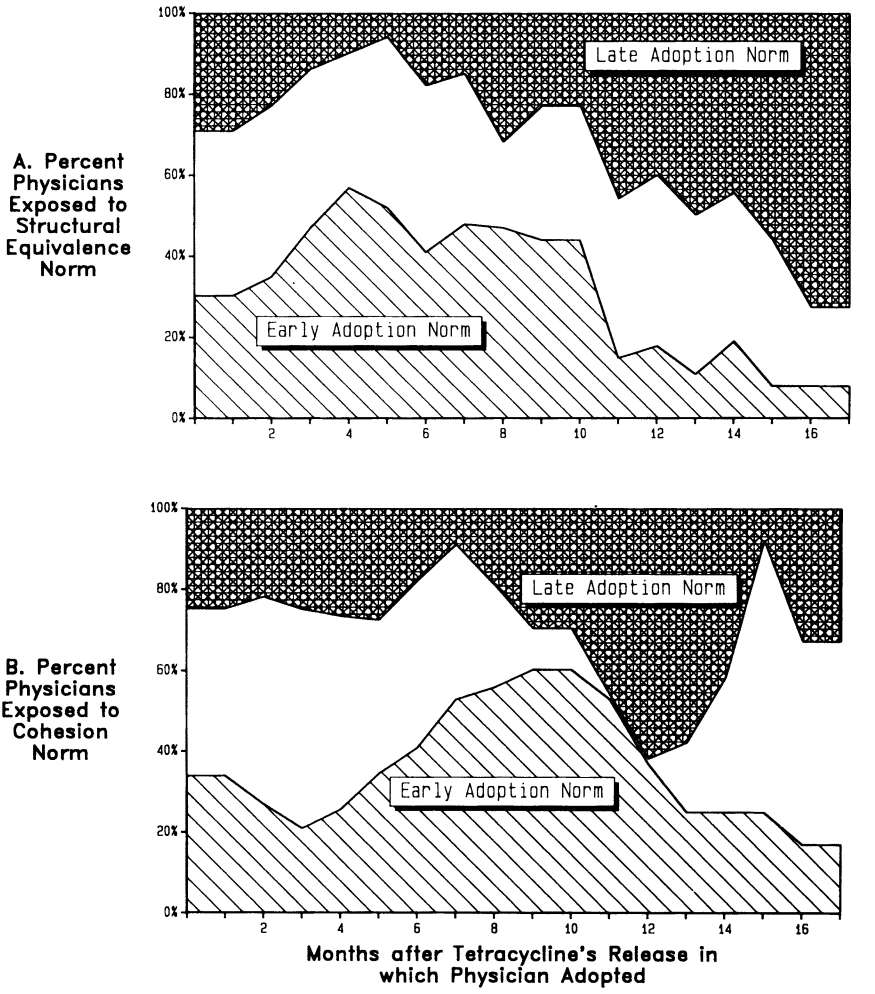

FIG. 5.—Adoption norms over time

adopting early and alters adopting late. Evidence of contagion is not only stronger under structural equivalence, it is also more consistent over time.

Further, there are lags in the contagion effects among early, median, and late adoptions that highlight four innovation roles played by the physicians, roles that capture the most basic evidence of social contagion in tetracycline's diffusion. The following tabulation of physician adoptions (rows) by structural equivalence alters (columns) is taken from

table 2:

early	18	15	8
median	23	18	11
late	0	20	11,

and the following multiplicative interaction effects are taken from a log-linear model of the frequencies (increased by .5 to eliminate the zero frequency):

early	2.4	0.7	0.6
median	2.4	0.6	0.7
late	0.2	2.4	2.3.

The third column describes physicians whose alters postponed adopting tetracycline until very late in its diffusion or never adopted it at all. Physicians exposed to such alters tended not to adopt during the early or middle phases of tetracycline's diffusion. The number of them postponing adoption, as did their alters, until late in the diffusion process is 2.3 times the number that would be expected if physician adoptions were independent of alter adoptions. The second column describes physicians exposed to alters adopting during the middle phase of tetracycline's diffusion. These physicians too were likely to delay writing prescriptions for the new antibiotic until late in its diffusion. The first column describes physicians whose alters adopted tetracycline soon after it was available. The number of such physicians who themselves adopted early is 2.4 times the number that would be expected if physician adoptions were independent of alter adoptions. This effect continues into the middle of tetracycline's diffusion. The number of physicians exposed to early-adopting alters and themselves adopting during the middle period of tetracycline's diffusion is 2.4 times the number that would be expected under independence. What physicians exposed to early-adopting alters were unlikely to do was postpone their adoption to the late phase of tetracycline's diffusion. In sum, contagion had a direct and lagged effect. Physicians exposed to alters adopting during a given phase of tetracycline's diffusion tended to adopt during that phase or in the subsequent phase. They tended not to adopt in the phase preceding the one in which their alters adopted and tended not to postpone adoption for more than a phase after the one in which their alters adopted.[20]

[20] This lag could be an artifact of the way in which prescriptions were sampled. Recall that prescriptions were audited for three working days per month. It is quite possible for a physician to have begun prescribing tetracycline and have had his prescriptions filled during the days for which prescription records were not audited, but such sampling errors could only occur in one direction. It is possible for a physician to have begun prescribing tetracycline earlier than the prescription data would indicate, but he

TABLE 3

INNOVATION ROLES

	Alters Adopt Early	Alters Do Not Adopt Early
Physician adopts early	Early conformer	Eager innovator
	27	34
	(19)	(38)
Physician adopts late or not at all ..	Deviant laggard	Late conformer
	14	49
	(23)	(37)

NOTE.—Frequencies for alters defined by cohesion are presented in parentheses beneath frequencies for alters defined by structural equivalence, and both are combinations of the frequencies in table 2. With 1 *df*, the likelihood-ratio χ^2 statistic for independence is unacceptable for the structural equivalence frequencies (6.89, $P < .01$) and quite acceptable for the cohesion frequencies (.32, $P = .57$).

Four innovation roles, which show the ways in which the physicians responded to interpersonal influence, can be distinguished in these effects. Physicians adopting tetracycline in the diffusion phase ahead of their alters were *eager innovators*, pioneers in their reference groups. These eager innovators went against the tendency for physicians to avoid adopting the new drug a phase ahead of their alters. Physicians exposed to alters adopting during the middle and late phases of tetracycline's diffusion and themselves adopting during these phases were *late conformers*. These physicians fell within the bounds of the typical response to middle- and late-adopting alters. Physicians in the first half of their city's adopters and exposed to alters adopting early were *early conformers*. They were exposed to a norm of adopting early and responded with their own early adoption, as was typical of physicians exposed to early-adopting alters. Finally, physicians postponing their own adoption until over half of their city's physicians had begun using tetracycline, despite the early adoptions of their alters, were *deviant laggards*. These physicians contradicted the tendency toward early or median adoption in response to alters adopting early.

In this final aggregation, displayed in table 3, contagion is a tendency for early and late conformers to outnumber eager innovators and deviant laggards. The diagonal frequencies in table 3 exceed the off-diagonals under structural equivalence and are about equal to the off-diagonals

had definitely begun prescribing by the time one of his prescriptions for it was located. This means that an unexpectedly late adoption could be a sampling error but an unexpectedly early adoption could not. The physicians seeming to adopt in the phase after their alters adopted, in other words, could easily have written earlier tetracycline prescriptions that went unnoticed in the prescription sampling.

TABLE 4

ATTRIBUTES PREDISPOSING PHYSICIANS TO TETRACYCLINE

ATTRIBUTES	MEAN	REGRESSION COEFFICIENTS PREDICTING ADOPTION DATE		
		Metric	Standardized	*t*-test
Belief in science	1.13	−2.31	−.33	−3.9
Professional age	.25	3.53	.28	3.3
Many journal subscriptions	1.94	−2.63	−.29	−3.5
Prescription-prone medical practice	.23	−3.30	−.25	−2.9
Detail-man contact	.85	−2.50	−.16	−2.0

NOTE.—Adoption date is the first sampling period in which a physician's prescriptions for tetracycline were found. Effects are estimated with pairwise deletion for the 125 physicians in the prescription sample, yielding an intercept of 17.65 and a .348 squared multiple correlation. Belief in science is a three-category variable weighing the relative importance of keeping up with scientific developments vs. spending time with patients (0 for physicians stressing patients, 1 for those stressing both, and 2 for those stressing science; data missing on four physicians). Professional age distinguishes (0) physicians graduating from medical school in 1930 or later (physicians roughly under 40 years old at the time of the study) from (1) those graduating before 1930 (data missing on one physician). Journal subscriptions is a three-category variable indicating extensive subscriptions to professional journals (1 for those subscribing to two of three journals, 2 for those subscribing to four to seven journals, and 3 for those subscribing to more than seven journals). Prescription-prone medical practice is a dichotomy based on a physician's frequency of house calls and office visits (see n. 22; data missing on 12 physicians). Detail-man contact distinguishes physicians who (1) remembered having been contacted by a detail man about the drug they most recently began using vs. (0) those not recalling such contact (data missing on 13 physicians).

under cohesion. The results in the bottom row of table 1 show that the evidence of physicians conforming to alter behavior is strong when alters are defined by structural equivalence (2.59 z-score, $P = .005$) and continues to be negligible when alters are defined by cohesion (-0.54 z-score, $P = .71$). These results are in proportion to the results obtained with more detailed adoption data, but for the four innovation roles highlight the critical distinctions in adoption dates that provide the evidence of social contagion in tetracycline's diffusion. This reduction in the number of distinctions made among adoption dates makes it possible to see evidence of contagion more clearly and to study contagion more reliably under varying conditions of personal predispositions toward adoption and varying social structural conditions.

CONTAGION, PERSONAL PREFERENCE, AND PROMINENCE

Many background attributes are discussed in *Medical Innovation*, but few show an independent effect on adoption when other attributes are held constant. The most significant are presented in table 4.[21] On aver-

[21] Data on the attributes highlighted in *Medical Innovation* are available in the data set cited in n. 8. For the purposes of this study, attributes were combined to maximize

age, physicians in the four cities began prescribing tetracycline about eight months after its release. Varying from this mean, young physicians began prescribing the new antibiotic 3.5 months before old physicians. Physicians who remembered being contacted by a drug company salesman ("detail man") began prescribing the new antibiotic 2.5 months before those not remembering such contact. In addition, early adopters tended to subscribe to many professional journals, to make many house calls, to have a moderate number of office visits,[22] and to emphasize the importance of keeping up to date with scientific developments in medicine as opposed to emphasizing the importance of devoting time to patients.[23] In sum, physicians predisposed toward adopting the new anti-

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their association with adoption. The mean adoption date was computed for physicians, with each attribute identified as having an effect on adoption in the original study. Attribute categories were combined to maximize differences in mean adoption date across attributes. Many of these aggregate categories were taken from the original study; e.g., the major differences between physicians, by the year in which they completed medical school, are between those who received their degree before 1930 and those who received their degree afterward. Therefore, a professional age attribute was coded as "young" (obtaining the degree in 1930 or after) versus "old" (obtaining the degree before 1930; see Coleman et al. 1966, p. 165). The many attribute variables, coded to have as clear and strong an association with adoption as possible, were then specified as simultaneous predictors of adoption in a multiple-regression equation. Those having a strong independent effect were retained for a final regression model, reported in table 4. In theory and method, this process of scaling and selecting predictors is inelegant. However, the purpose of the exercise was not to specify a structural equation model of the attributes affecting adoption; the purpose was to sort physicians by their predisposition toward adopting tetracycline so that preference could be held constant in order to reveal evidence of social contagion better.

[22] The number of tetracycline prescriptions that a physician wrote was strongly associated with early adoption (Coleman et al. 1966, p. 39) and so held constant in many graphs presented as evidence in the original study. This association seems reasonable, inasmuch as those physicians first adopting had a longer period of time in which to write tetracycline prescriptions. In order to measure prescription behavior in a manner more independent of the dependent variable, I considered several attributes of a physician's medical practice: number of office visits, number of house calls, his perception of his tendency to prescribe drugs, and his perception of this tendency relative to other physicians'. The strongest association between these attributes was obtained with a combination of office visits and house calls, indicating a physician's opportunities to prescribe tetracycline. Physicians making more than 15 house calls per week tended to begin prescribing tetracycline early. Those receiving a moderate number of office visits, 26–100 per week, tended to begin prescribing earlier than those receiving an extreme number, more than 100 or fewer than 26. Physicians whose medical practice made them prone to early adoption (many house calls and a moderate number of office visits) are coded "1" on the "prescription-prone medical practice" variable in table 4, and othe physicians are coded "0". This variable is more strongly associated with adoption than either office visits or house calls alone.

[23] Detailed information on these effects can be obtained in the original study (Coleman et al. 1966, pp. 164–66 on age, pp. 47–48, 182–85 on orientation toward science in medicine, and pp. 44–46 on subscriptions to professional journals). No association is

biotic were young (professionally), kept up to date with scientific developments in medicine, and believed that such behavior was important to being a good physician. To measure each physician's predisposition toward adoption, an aggregate personal preference variable has been constructed by computing the adoption date expected of a physician from his attributes listed in table 4.[24]

Although personal preferences had an obvious effect on adoptions, there is equally strong evidence of contagion, regardless of personal preference. Summary results from an analysis of contagion and personal preference are presented in table 5. Three conclusions can be drawn. First, the evidence of contagion observed with personal preference held constant is nearly identical to the evidence observed without controls for personal differences among the physicians. Regressing a physician's observed adoption over his adoption norm and the five background variables in table 4 yields: (*a*) no evidence of contagion from his advisers and discussion partners and (*b*) strong evidence of contagion between the month in which he adopted and the month in which the physicians to whom he was structurally equivalent adopted. This evidence is presented in the first row of table 5, corresponding to the zero-order effects reported in the first row of table 1. Similarly, the evidence of contagion between adoptions in aggregate phases of tetracycline's diffusion is unaffected by controls for personal preference. The results in the second row of table 5 correspond to the results in the fourth row of table 1, with the dominant effect being the tendency for physicians to have conformed to the adoption behavior of physicians to whom they were structurally equivalent (2.61 z-score, $P = .005$).

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reported between adoption and the number of detail men seen (p. 180), but the effect reported here is between some vs. no contact. A strong association is reported in the original study between adoption and attending specialty meetings (p. 45), but the physicians who attended many such meetings also subscribed to many professional journals, and the latter is more strongly associated with adoption. Attending specialty meetings, along with several other attributes associated with adoption in the original study, has a negligible effect on adoption when the attributes in table 4 are held constant.

[24] Specifically, the five attribute variables have been aggregated by using the metric regression coefficients in table 4: $p_j = 8 + \Sigma_i b_i(x_{ji} - \bar{x}_i k)$, where p_j is physician j's personal preference (expected adoption date), x_{ji} is his score on the ith attribute having effect b_i on adoption and mean $\bar{x}_i$, summation is across the five attribute variables i, and 8 is the mean adoption date. Where x_{ji} is missing, it is set equal to its mean value $\bar{x}_i$, so that physician j's preference is neither increased nor decreased relative to other physicians' by the missing attribute. A preference score is available on each prescription-sample physician, representing his predisposition toward adopting tetracycline relative to the other prescription-sample physicians'.

TABLE 5

CONTAGION AND PERSONAL PREFERENCE

	Structural Equivalence	Cohesion
Continuous contagion effect, holding preference constant[a]	.24 (t = 3.0)	.05 (t = .5)
Contagion effect in innovation roles across personal preferences[b]	1.31 (z = 2.61) (P = .005)	.96 (z = −.42) (P = .66)
Personal preference effect across adoption norms[c]	1.20 (z = 1.81) (P = .04)	1.21 (z = 1.89) (P = .03)
Adoption norms independent of personal preferences[d]	1.57 (P = .46)	6.59 (P = .04)

[a] Standardized, ordinary least-squares estimates of b_s in eq. (1) are reported for multiple-regression models predicting observed month of adoption from a continuous adoption-norm variable (see App.) and the five variables in table 4 indicating a physician's predisposition toward adoption. Routine t-tests are presented to provide some sense of effect magnitude relative to residual variance; however, routine statistical inferences should not be made from these results (see App.). Results are based on those prescription-sample physicians on whom complete data are available (101 under structural equivalence, 96 under cohesion). The same results are obtained if missing values of variables in table 4 are set equal to means (N increases to 124 under structural equivalence and to 117 under cohesion). Standardizing adoption dates by city means and standard deviations yields coefficients of .21 and .05 for structural equivalence and cohesion, respectively.

[b] The effect is the multiplicative interaction between observed and normative adoption in a log-linear model of the three-way binary tabulation of observed adoption, adoption norm, and personal preference (the aggregate variable constructed from the five variables in table 4 and dichotomized at the mean). The structural equivalence table is based on 124 physicians. The cohesion table is based on 117 physicians. The effect measures the tendency for physicians to have been conformers rather than deviants, across personal preference categories (cf. effects in the fourth row of table 1).

[c] The effect is the multiplicative interaction between personal preference and adoption in a log-linear model of the three-way binary tabulation in n. b. The effect measures the tendency for high-preference physicians to have adopted early and low-preference physicians to have adopted late, regardless of the dates at which their alters adopted.

[d] Likelihood-ratio χ^2 statistics are reported for the three-way binary tabulation eliminating both interactions between adoption norms and personal preferences and so have 2 *df*. Corresponding χ^2 statistics of 3.07 (P = .80) and 7.44 (P = .28) with 6 *df* are obtained if adoption date is categorized into early, median, and late adoption, as in the third row of table 1.

Second, a physician's personal situation strongly determined his adoption, regardless of adoptions by the physicians around him. This is illustrated by the results in the third row of table 5. Physicians predisposed toward adoption tended to adopt early, and those predisposed against adoption tended to adopt late, regardless of adoptions by their structurally equivalent peers (1.81 z-score, $P = .04$) or adoptions by their advisers and discussion partners (1.89 z-score, $P = .03$). More specifically, the belief in science, professional age, and journal subscription variables strongly predicting adoption date in table 4 remain strong predictors, with continuous adoption norms held constant (t-tests of -4.3, 3.9, and -3.3, respectively, with structural equivalence norms held constant).

Third, contagion and personal preference can be treated as independent factors in tetracycline's diffusion. The χ^2 statistics in the bottom row of table 5 show that there is no direct or three-way interaction between personal preference and the tendency for a physician's structurally equivalent alters to have adopted early ($\chi^2 = 1.57$, $P = .46$). The data on cohesion are slightly more complex because of a significant three-way interaction, but there is no direct tendency for high-preference physicians to have had advisers and discussion partners adopting early in tetracycline's diffusion. The 6.59 χ^2 statistic with 2 *df* in table 5 is the sum of two χ^2 statistics with 1 *df*, a negligible 2.16 χ^2 statistic ($P = .16$) created by eliminating the direct interaction between personal preferences and alter behavior, and a significant 4.43 χ^2 statistic ($P = .04$) created by eliminating the three-way interaction from the table.

The significant three-way interaction created by the cohesion model can be traced to systematic bias in the model. High-preference physicians tended to be prominent within the network of advice and discussion relationships, and cohesion broke down in predicting adoptions by prominent physicians. This is illustrated by figures 6 and 7.

Associations among prominence, personal preference, and adoption date are illustrated in figure 6. Four categories of prominence are distinguished. A physician not named by anyone as an adviser or discussion partner was at the bottom of the medical hierarchy, and a physician named by four or more other physicians was at the top. Someone named by a single other physician was below average, and someone named by two or three was about average.[25]

[25] These four categories are based on the adoption behavior of physicians at each level of choice status. Those receiving no citations were quite different from those receiving one citation, and both were different from physicians receiving two or more citations. Physicians receiving two or three citations were more similar to one another than to physicians receiving four or more, and no further categories were apparent among physicians receiving more than four citations (bear in mind the very small number of physicians in higher categories). The four categories roughly correspond to categories

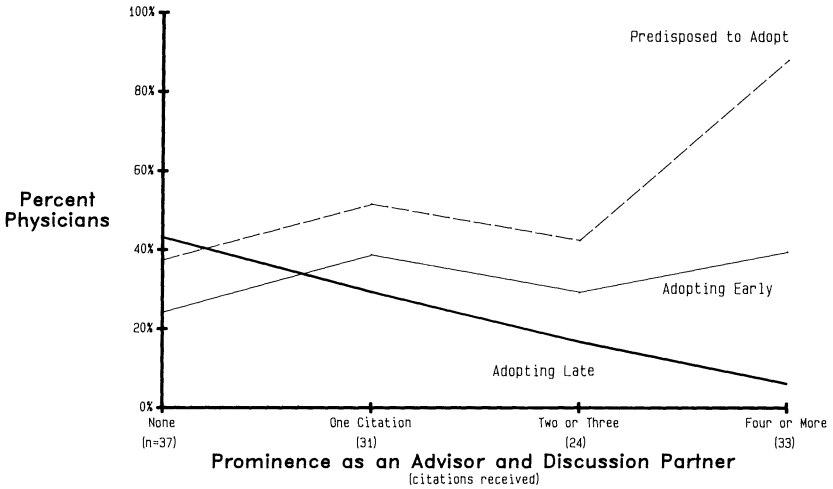

FIG. 6.—Adoption across levels of prominence

There is a sharp tendency for the most prominent physicians to have been predisposed toward adopting the new antibiotic. There are few differences among the low- and average-prominence physicians on the dichotomous personal preference variable ($\chi^2 = 1.43$, 2 *df*, $P = .49$). Among the physicians of low to average prominence, 43% were predisposed toward adopting the new antibiotic. This more than doubles, to 88%, for the most prominent physicians, creating a strong overall association between prominence and predisposition toward adoption ($\chi^2 = 22.93$, 3 *df*, $P < .001$).

There is also a strong association between prominence and adoption date. This association was emphasized in the original study (see, e.g., Coleman et al. 1966, pp. 79–112) and has become an often-replicated finding in diffusion research (Rogers 1983, p. 277 ff.). Here, city-standardized choice status (the number of citations a physician received, standardized for city-specific means and standard deviations) is strongly associated with adoption date, with prominent physicians adopting early in tetracycline's diffusion (-2.6 *t*-test, $P = .01$).[26]

constructed from city-standardized choice status: low (*z*-score of -1 or less), below average (*z*-scores from -1 to 0), above average (*z*-scores from 0 to 1), and high (*z*-scores greater than 1). I have used the citation categories in figs. 6 and 7 because they are more obviously tied to the observed sociometric data.

[26] In an interesting analogy to Homans's (1961, p. 352 ff.) and Cancian's (1967, 1979) descriptions of middle-class conservatism, this association is kinked in the middle. Mean adoption dates decrease from 10.4 months for physicians receiving no citations

More precisely, figure 6 illustrates the uneven association between prominence and adoption date. There is no association between prominence and early adoption, but a strong negative association with late adoption. The thin solid line in figure 6 shows the proportion of physicians at each level of prominence who were early adopters (rather than median or late adopters; see table 2). City-standardized choice status has a .10 correlation with early adoption, and early adoption is statistically independent of the four choice-status categories in figure 6 ($\chi^2 = 2.53$, 3 *df*, $P = .47$). In contrast, city-standardized choice status has a $-.24$ correlation with late adoption (-2.7 *t*-test, $P = .004$), and late adoption varies significantly across the categories in figure 6 ($\chi^2 = 15.35$, 3 *df*, $P = .002$). The bold solid line in figure 6 shows the proportion of physicians at each level of prominence who were late adopters. The line decreases linearly across levels of prominence, from 43% of physicians receiving no citations to 6% of physicians receiving four or more citations. In keeping with the structural equivalence conception of contagion, the strong association between early adoption and prominence is not a result of prominent physicians rushing to have been the first to adopt. It is created by their tendency to have avoided being the last to adopt.

The prominence effect is virtually unaffected by the behavior of a physician's advisers and discussion partners. Regardless of adoptions by their advisers and discussion partners, marginal physicians tended to be late adopters (3.10 *z*-score, $P = .001$), and prominent physicians tended not to be (-2.51 *z*-score, $P = .006$). Aggregating across prominence levels, the direct association between late adoption and prominence is equally significant before and after cohesion norms are held constant ($\chi^2 = 15.35$, 3 *df*, $P = .002$, before and 18.90, 6 *df*, $P = .004$, after).

Nevertheless, the prominence effect is in one sense spurious. It disappears when contagion by structural equivalence is held constant. Merely holding constant the distinction between early-adopting alters and median- or late-adopting alters (the distinction used to define innovation roles in table 3) eliminates any association between late adoption and prominence ($\chi^2 = 5.54$, 6 *df*, $P = .48$).

This difference between cohesion and structural equivalence in eliminating the prominence effect is illustrated in figure 7. Contagion is estimated within prominence categories and measured on the vertical axis by a *z*-score expressing the tendency for conforming physicians to outnumber deviants (see contagion among innovation roles in tables 1 and 5). The

to 7.9 months for physicians cited by one physician. Physicians receiving two and three citations adopted at about the same time as the physicians receiving one citation (7.4 months). Above three citations, mean adoption date again decreases to 5.8 months among physicians receiving four or more citations.

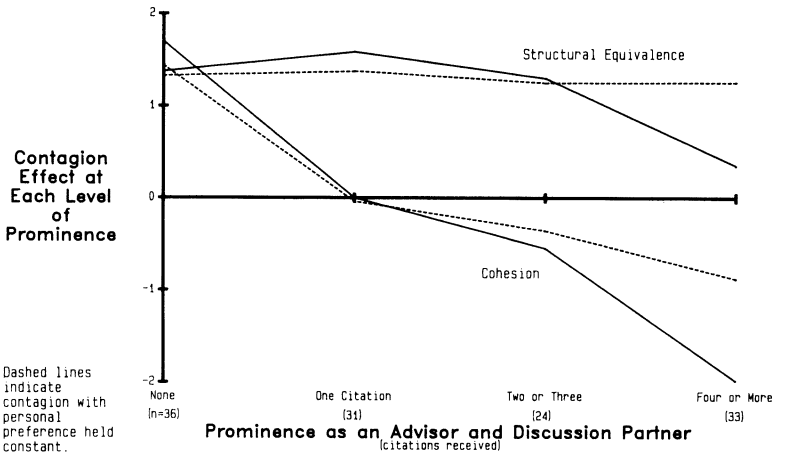

FIG. 7.—Social contagion within levels of prominence

contagion effect is positive to the extent that a physician and his alters adopted during the same phase of tetracycline's diffusion. A zero effect indicates that physician adoptions were not contingent on alter adoptions, and a negative effect indicates that physicians adopted during a diffusion phase other than that in which their alters adopted.[27]

[27] The conclusions illustrated in fig. 7 for categorical adoption dates are also supported by analysis-of-covariance models describing continuous contagion effects. With observed ($\mathbf{X}$) and normative ($\mathbf{X}^*$) adoption dates standardized by city means and variances (so that interaction effects measure slope adjustments for the average physician in a city), ordinary least-squares estimates of parameters in the following analysis-of-covariance model have been obtained (see App.):

$$\mathbf{X} = (a_1 + a_2P_2 + a_3P_3 + a_4P_4) + (b_1 + b_2P_2 + b_3P_3 + b_4P_4)\mathbf{X}^* + \mathbf{E},$$

where P_2, P_3, and P_4 are dummy variables corresponding to the second, third, and fourth categories of network prominence in fig. 7 (respectively, equal to one for a physician receiving one, two or three, or four or more citations). Terms in the first parentheses define the equation intercept, and terms in the second parentheses define the equation slope. The continuous contagion effect among uncited physicians is measured by b_1. The effect among physicians cited by one other physician is measured by $b_1 + b_2$. The effect among physicians cited by two or three physicians is measured by $b_1 + b_3$, and the effect among the most prominent physicians is measured by $b_1 + b_4$. Routine t-tests for b_1, b_2, b_3, and b_4 indicate the magnitude of these effects relative to residual variation but are uncertain indicators of statistical significance (see App.). When $\mathbf{X}^*$ is defined by cohesion, the t-tests for b_1, b_2, b_3, and b_4 are 2.7, -2.1, -3.1, and -2.2, respectively. The same pattern of effects is observed if the aggregate personal preference variable constructed from table 4 is entered into the above equation (t-tests of 2.4, -2.4, -2.4, and -1.7, respectively). When $\mathbf{X}^*$ is defined by structural equivalence, in contrast, the slope adjustments are all negligible. The t-tests are .7, .9, $-.1$, and $-.1$, respectively, before holding personal preference constant and .9, .4, $-.2$, and $-.3$, respectively, after holding personal preference constant.

The two lines at the top of figure 7 illustrate the stability of contagion under structural equivalence. There is a slight tendency for contagion to decline among the most prominent physicians, but that decline is statistically negligible ($\chi^2 = 2.16$, 3 *df*, $P = .54$) and completely eliminated by holding dichotomous personal preferences constant (see dashed and solid lines in fig. 7). Finally, the evidence of contagion by structural equivalence is significant across the four prominence categories.[28] In other words, all physicians—marginal and prominent—began adopting tetracycline at about the same time as other physicians occupying similar positions in the social structure of medical advice and discussion.

Physicians clearly were not following the behavior of the people from whom they sought advice or with whom they discussed cases. Across the levels of network prominence, there is the now-familiar lack of support for contagion by cohesion.[29] The two lines at the bottom of figure 7 illustrate the instability of this aggregate effect. Contagion by cohesion is evident among physicians never cited as an adviser or discussion partner (1.70 *z*-score, $P = .04$). Among the most prominent physicians, in contrast, the negligible aggregate contagion effect is negative. Prominent physicians actually deviated from their advisers and discussion partners more than they conformed to them (-1.98 *z*-score, .05 two-tail probability). In sum, the interaction in table 5 between preference and contagion by cohesion reflects cohesion's failure to predict adoption by prominent physicians. Prominent physicians were predisposed to adopting tetracycline early and appear more willing to deviate from the adoption norms of their advisers and discussion partners. The same physicians, however, conformed to the adoption norms of their structurally equivalent peers.

CONCLUSIONS

To summarize in a sentence, two factors drove the diffusion process: personal predispositions and contagion by structural equivalence. Cohe-

[28] This constant is based on three-way tabulations of observed adoption (early, late), adoption norm (early, late), and a measure of network structure (low, below average, above average, high). The likelihood-ratio χ^2 statistic for observed and normative adoption being independent across the four levels of prominence in fig. 7 is 7.13, with 4 *df*. This is the sum of a negligible tendency for contagion to decline with prominence ($\chi^2 = 2.16$, 3 *df*, $P = .54$) and a significant tendency for physicians to have conformed to rather than deviated from the adoption behavior of alters ($\chi^2 = 4.97$, 1 *df*, $P = .03$).

[29] This conclusion is based on the three-way tabulation described in the preceding note, here by using cohesion instead of structural equivalence to define adoption norms. The likelihood-ratio χ^2 statistic for observed and normative adoption being independent across the four levels of prominence in fig. 7 is 7.10, with 4 *df*. This is the sum of a tendency for contagion to decline with prominence ($\chi^2 = 6.95$, 3 *df*, $P = .07$) and negligible tendency for physicians to have conformed to rather than deviated from the adoption behavior of alters ($\chi^2 = 0.15$, 1 *df*, $P = .90$).

sion and structural equivalence have been compared for their adequacy as the driving mechanism in social contagion. The alternative models have been defined, compared, and applied to behavioral data on the diffusion of a new antibiotic, tetracycline, among physicians in four midwestern cities during the mid-1950s. Four conclusions have been drawn from the analysis: (*a*) Putting the effects into perspective, it is clear that contagion was not the dominant factor driving tetracycline's diffusion. The slow initial diffusion, characteristic of social contagion, is missing altogether. Where there is evidence of contagion, there is evidence of personal preferences at work. (*b*) Where contagion occurred, however, there is strong evidence of contagion through structural equivalence and virtually no evidence of contagion through contagion. (*c*) Regardless of contagion, adoption was strongly determined by physicians' personal preferences, but these preferences did not dampen or enhance contagion. Personal preference and social contagion can be treated as independent components in tetracycline's diffusion. (*d*) There is no evidence of a physician's network position influencing his adoption when contagion is properly specified in terms of structural equivalence. Ostensible evidence of a prestige effect is spurious, resulting from biases created when cohesion is used to model contagion. In short, the product of reanalyzing the *Medical Innovation* data in light of recent developments in network theory is clearer, stronger evidence of social contagion and a redefinition of the social structural conditions responsible for contagion.

There is a central message here. When one studies either contagion in the diffusion of an innovation or, more generally, informal social pressures on subjective opinions, principles of cohesion or structural equivalence can be used to guide the analysis. Structural equivalence is a recent development; cohesion is typically the principle assumed to generate social pressure. The central message of this analysis is that the ordering of cohesion and structural equivalence should be reversed. Structural equivalence is the principle more likely to generate social pressure. The results of this analysis show that cohesion is much weaker in the aggregate than structural equivalence and is systematically biased against correct predictions in certain social structural conditions. Burt and Doreian (1982; Burt 1982, chap. 6) reach the same conclusion in their study of perceptions of journal significance among elite sociological methodologists in the mid-1970s. As in the results presented here, structural equivalence is more accurate than cohesion in predicting expert perceptions of journal significance, and cohesion is systematically biased in certain social structural conditions. Unlike the results of this analysis, cohesion did have some effect on expert perceptions, and the social structural conditions in which cohesion broke down could be identified more easily because statuses in the social structure of elite methodological advice were

much more evident than is true of advice and discussion among the *Medical Innovation* physicians. Cohesion has little to recommend it in the results of these studies. The results suggest that evidence of contagion and social pressure that has in the past been attributed to cohesion is probably evidence of structural equivalence obtained in social structural circumstances where the two models make identical predictions. The results further suggest that stronger evidence of contagion and social pressure in the earlier studies could be obtained by reanalyzing their data with network models based on structural equivalence, as has been done here with the *Medical Innovation* data.

In closing, a note of caution. The *Medical Innovation* data concern highly trained technical professionals evaluating the risk of prescribing a new drug. The elite methodology data concern highly trained technical professionals evaluating the value of publishing their work in alternative journal outlets. In addition to the similarities, obvious in these two sentences, people in both study populations were overexposed to information on the objects being evaluated. The marketing campaigns of drug companies leave few physicians ignorant of the latest releases. Eighty-five percent of the prescription-sample physicians recalled a visit from a drug company salesman (detail man) advocating the drug that they had most recently begun prescribing. Similarly, no social scientist pretends to keep up with all the latest developments in statistical models, mathematical models, and research designs. The barrage of information on methodological developments is simply crushing. Any of these similarities between the *Medical Innovation* physicians and the elite methodology experts could be responsible for the obvious failure of cohesion. Perhaps, cohesion is weaker in predicting responses to excessive information. Perhaps, it is weaker in predicting the perceptions of highly trained technical professionals. These are cautions but no more than speculations. What is fact is that cohesion yields predictions that are near random in the aggregate and systematically biased in certain social structural conditions, while structural equivalence yields strong, stable predictions with the same data.

APPENDIX

Estimating Contagion Effects

It is convenient to discuss equation (1) as a network autocorrelation model expressed in matrix terms:

$$\mathbf{X} = b_p\mathbf{P} + b_s\mathbf{W}\mathbf{X} + \mathbf{E},$$

where $\mathbf{X}$ is a vector of adoption dates, $\mathbf{P}$ is a vector of personal preference data, $\mathbf{E}$ is a vector of residuals, and $\mathbf{W}$ is a matrix of the network weights

defined in equation (2). The product $\mathbf{WX}$ defines a vector of adoption-date norms, $\mathbf{X}^*$; b_p measures the effect of personal preference on adoption; and b_s—the network autocorrelation effect—is a linear-slope coefficient measuring the contagion effect of alters' adoptions on ego's adoption. In the *Medical Innovation* data, $\mathbf{W}$ is a square, block-diagonal matrix with network weights for each of the 216 study respondents defining a row of the matrix: nonzero w_{ji} within cities and zero w_{ji} between cities. There is a rich statistical literature on estimation problems posed by such models (see Ord 1975; Davis and McCullagh 1975; Tobler 1975; Haining 1980; Ripley 1981; and Cliff and Ord 1981 for review and references), and the general problem has been brought into sociology by network analysts (see, e.g., Doreian 1980, 1981; Dow, Burton, and White 1982; Dow et al. 1984; Dow 1984). The main point here is that an ordinary least-squares estimate of b_s is unsatisfactory; correlations between the residuals in $\mathbf{E}$ make the ordinary least-squares estimate inefficient, and (excluding triangular $\mathbf{W}$) correlation between $\mathbf{E}$ and the predictor $\mathbf{WX}$ makes the ordinary least-squares estimate inconsistent (see, e.g., Ord 1975, pp. 121–22). Maximum-likelihood estimates of b_s are obtained numerically.

Unfortunately, maximum-likelihood estimates of b_s cannot be obtained with the *Medical Innovation* data. Of the 216 elements in $\mathbf{X}$, adoption date is only known for the 125 physicians in the prescription sample. Available estimation procedures cannot be used with missing data and so cannot be used here, although they are useful in imputing missing data, as described below.

I have adopted the following guidelines in presenting evidence of social contagion: (1) Ordinary least-squares coefficients are presented because of their general familiarity, with the proviso that routine statistical inference cannot be used to interpret their significance. (2) The response variables $\mathbf{X}$ and $\mathbf{X}^*$ are collapsed into broad categories. To the extent that the network weights defining $\mathbf{X}^*$ are correctly specified, most of the correlation between residual terms will occur within categories. Inferences are then made through generalized least squares in the form of log-linear statistics. As discussed in the text, this aggregation of responses has a substantive rationale in the difference between social and physical time and a methodological rationale in shifting precision from describing the form of contagion's effect to describing the circumstances under which contagion operates. (3) Effects are not studied too closely for their statistical significance. Contagion effects are interpreted only where the null hypothesis is extremely unlikely, with a .01 or less probability. Fortunately, the evidence of social contagion is strong under structural equivalence and very nearly random under cohesion.

Weighting Alters

Alters have been defined with the weights in equation (2), defined by the sociometric advice and discussion choices made by the 228 interviewed physicians. The 18 uninterviewed physicians cited by two or more prescription-sample physicians are included in the sociometric calculations because they define points of similarity in the relation patterns of the physicians citing them and so contribute to variation in the structural equivalences among them. The pooled advice and discussion data define a choice matrix in which cell (j,i) is one if j cited i as an adviser or discussion partner and zero otherwise. The choice matrix is 117 × 117 in Peoria, 50 × 50 in Bloomington, 34 × 34 in Quincy, and 32 × 32 in Galesburg. Path distances have been computed and normalized to define relation variables z_{ji} varying between zero and one, with the minimum number of choices required to reach physician i from physician j, where zero indicates that there is no chain of intermediary advisers or discussion partners through which physician j can reach physician i. Structural equivalence has been defined by the Euclidean distance d_{ij} between the positions of physicians i and j in the network of medical advice and discussion,

$$d_{ij} = [(z_{ij} - z_{ji})^2 + \Sigma_k(z_{ik} - z_{jk})^2 + \Sigma_k(z_{ki} - z_{kj})^2]^{1/2},$$

where summation is across all physicians k other than i and j. These are standard network distance measures and are reviewed elsewhere (see, e.g., Burt 1982, pp. 42–49).

Cohesion adoption norms have been computed from raw choice data and normalized path distances among the 216 study respondents (excluding the 12 physicians interviewed only as informants). Inserting the normalized path distances for proximity in equation (2) yields:

$$w_{ji} = (z_{ji})^\nu / \Sigma_k(z_{jk})^\nu, \; k \neq j.$$

This cohesion weight, based on path distance, generates negative correlations between observed and normative adoption for integer values of ν from one to six. Better results are obtained with raw choice data (z_{ji} equal to one or zero)—the limiting case of w_{ji} for infinite ν. The correlation between observed and normative adoption reported in table 1 is negligibly positive across the four cities. Using raw choice data to measure relation strength, we can drop ν from the equation (because $z_{ji} = z_\nu^{ji}$), and the above network weight for cohesion is as follows: w_{ji} is $1/K$ if j cited i as adviser or discussion partner and zero otherwise, where K is the number of the 216 study respondents that j cited as advisers or discussion

partners. Note that w_{ji} varies from zero to one and sums to one across all physicians i.

Structural equivalence adoption norms have been computed from the above-defined Euclidean distances among the 216 study respondents in each city. These distances capture the similarity of direct and indirect relations in which physicians i and j were involved. Given the largest distance between j and any other physician in his community, $d\max_j$, the proximity of some physician i to j can be expressed by the extent to which d_{ji} is smaller than $d\max_j$, i.e., $d\max_j - d_{ij}$, which can be inserted in equation (2) to define a structural equivalence network weight (see Burt 1982, pp. 176–77; Burt and Doreian 1982, p. 117):

$$w_{ji} = (d\max_j - d_{ij})^{\nu}/\Sigma_k(d\max_j - d_{kj})^{\nu},\ k \neq j.$$

This weight varies from zero to one, measuring the extent to which i was structurally equivalent to j and sums to one across all physicians i. Structural equivalence norms have been computed for integer values of ν ranging from one to 15. In Galesburg, the maximum contagion effect is obtained with ν equal to two. The association is negligibly positive for ν equal to six or more in Quincy, so the exponent has been left at six. In the more complex social structures of Bloomington and Peoria, the association between observed and normative adoption increases with increasing values of ν but changes little past 12 in Bloomington and 10 in Peoria, so norms have been defined at these values.

The method used to select a value of ν is exceedingly crude, but the lack of an estimator for ν and the cost of computing norms for alternative values of ν severely constrain any practical search. What is clear from the high values of ν for cohesion and structural equivalence is that physicians only relied on the closest of alters as a social frame of reference for their own evaluations of adopting tetracycline (see Burt 1982, p. 234; Burt and Doreian 1982; and Friedkin 1983 for similar conclusions). It is reassuring to note that this finding is consistent with the standard practice of using direct sociometric citations to test cohesion effects and ignoring indirect connections through intermediaries (see, e.g., Coleman et al. 1966, p. 113 ff.).

Missing Data on Alter Adoptions

The preceding calculations involve all 216 study respondents, but adoption date is only available on the 125 prescription-sample physicians. Unfortunately, there is no allowance for missing data in the class of network models under consideration. Consider a five-person system in

which adoption data are missing on the fourth person (i.e., $x_j = x_1, x_2, x_3, ?, x_5$) and alters are defined by the following network weights:

$$\begin{matrix} .0 & .5 & .5 & .0 & .0 \\ .5 & .0 & .0 & .6 & .0 \\ .0 & .4 & .0 & .0 & .3 \\ .5 & .5 & .0 & .0 & .0 \\ .3 & .4 & .0 & .3 & .0, \end{matrix}$$

so that adoption norms for the first, third, and fourth persons can be computed from equation (1) ($x_1^* = .5x_2 + .5x_3$, $x_3^* = .4x_2 + .3x_5$, and $x_4^* = .5x_1 + .5x_2$), but adoption norms for the second and fifth persons cannot be computed because of the missing data on the fourth person's adoption. If we sum the network weights to alters whose adoption is unknown, 60% of x_1^* is undefined and 30% of x_5^* is undefined.

Doing the same computation for physicians in the prescription sample reveals that 40% of the average physician's adoption-date norm is undefined under structural equivalence and 37% is undefined under cohesion. In other words, a large proportion of physician reference groups extended beyond the limits of the prescription sample.

Deleting observations having missing data does not seem wise here. Ignoring the physicians beyond the prescription sample would seriously distort the social situations in which prescription-sample physicians evaluated tetracycline. Limiting the analysis to prescription-sample physicians on whom alter data are complete, however, would leave too few physicians to estimate effects: one physician under structural equivalence and 28 under cohesion.

The missing adoption data on physicians outside the prescription sample have been imputed. The network model defining a norm is used to make a best guess of how missing alters responded to tetracycline adoption. A missing alter's adoption was imputed from his alters'. Imputation was carried out as part of the process generating adoption norms. Given physician j in the prescription sample and some physician i in or beyond the prescription sample, for whom w_{ji} is nonzero and on whom adoption data are missing, impute x_i from x_k for a physician k who can speak as a surrogate alter for i: (1) Locate the physician k best defining an adoption norm for the missing alter i, that is, locate the largest w_{ik} for all k, $k \neq j$. If two or more physicians are equally the strongest weights in defining i's adoption norm, then one of them is selected at random to speak for i. (In order to minimize regression toward the mean on the norm variable and keep alter responses as close as possible to observed response data, this seems preferable to averaging their responses.) (2) Impute x_i from x_k. (3) If the data on k's adoption are also missing, then locate the physician with the next strongest weight in defining an adoption norm for the missing

alter i. Continue until the missing data on i are imputed. If no surrogates for i are found (e.g., if cohesion norms are being computed and i cited no advisers or discussion partners), then delete i from respondent j's alters and increase the remaining weights to sum to one. If more than half of j's alters cannot be imputed, then delete his adoption norm as missing data.

Consider the five-person example given above. In order to compute an expected adoption date for person 5, person 4's adoption has to be imputed. Persons 1 and 2 are equally the strongest weights in defining an adoption norm for person 4; person 1 is selected at random. The value of x_4 is set equal to x_2. An adoption norm for person 5 is now defined: $x^*_5 = .3x_1 + .4x_2 + .3x_2$.

Tests were made against the possibility that imputing alter responses affected the study conclusions. As described above, the level of imputation on physician j was computed as the sum of nonzero w_{ji}, where data on i's adoption were imputed. The results of these tests have been deleted to conserve space but only support a negative conclusion: imputation is uncorrelated with the adoption, network, and preference variables.

The final point to note is that failing to eliminate ego from the imputation can bias the evidence of social contagion. In the above example, suppose that person 4's missing adoption data were imputed from data on person 2. This poses no problem in predicting person 5's adoption, but it would create a problem in predicting person 2's adoption; x_2 would be used to predict x_2, and an erroneously inflated measure of social contagion would result. More generally, failure to eliminate ego from imputation would result in the strongest evidence of social contagion being found in study populations of nonoverlapping cohesive or structurally equivalent dyads with data missing on one member of each dyad. In sum, imputation must be, and has been, carried out independently for the alters of each prescription-sample physician.

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