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A NEW MODEL FOR INFORMATION DIFFUSION IN HETEROGENEOUS SOCIAL NETWORKS

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This paper discusses a new model for the diffusion of information through heterogeneous social networks. In earlier models, when information was given by one actor to another the transmitter did not retain the information. The new model is an improvement on earlier ones because it allows a transmitter of information to retain that information after telling it to somebody else. Consequently, the new model allows more actors to have information during the information diffusion process. The model provides predictions of diffusion times in a given network at the global, dyadic, and individual levels. This leads to straightforward generalizations of network measures, such as closeness centrality and betweenness centrality, for research problems that focus on the efficiency of information transfer in a network. We analyze in detail how information diffusion times and centrality measures depend on a series of network measures, such as degrees and bridges. One important finding is that predictions about the time actors need to spread information in the network differ considerably between the new and old models, while the predictions about the time needed to receive information hardly differ. Finally, some cautionary remarks are made about using the model in empirical research.

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1. INTRODUCTION

A considerable amount of research has been devoted recently to studying the efficiency of diffusion of different kinds of goods through networks. Models of processes such as epidemics of infectious diseases, diffusion of innovations, information diffusion, and influence of actors on others are all based on contacts between actors organized in a social network. We concentrate in this paper on the efficiency of information diffusion. The term "efficiency" refers to how fast information is expected to flow through a network with a particular structure. The major question is how efficiency of information diffusion depends on network measures such as density, degrees, centralization, and number of bridges. This question can be addressed at different levels. On the dyadic level, what is the expected time for information to go from a specific transmitter of information to a specific receiver? On the individual level, what is the expected time for an actor to receive or to diffuse information in a network? On the global level, what is the expected time for information to diffuse in a network starting from an arbitrary actor in the network? Yamaguchi explained (in)efficiency of information diffusion at the global (1994a) and dyadic (1994b) levels with the network measures mentioned above. In this paper, we compare the model used by Yamaguchi and earlier by Friedkin (1991) with a new model for the diffusion of information in heterogeneous networks.

Our new model avoids a problematic assumption by Yamaguchi (1994a) that is not made explicit and discussed substantially in the paper: namely, that information is handed from one actor to another like some kind of package. Two centrality measures that Friedkin (1991) introduced and the measure of inefficiency in information flow through networks that Yamaguchi (1994a) introduced rely on mean first passage time and the related assumption in the Markov chain model (Kemeny and Snell 1960) that the actor who transmits information loses it at the time he transmits.[1] However, this assumption is inconsistent with "[the] peculiar feature of information as a resource, in contradistinction to other sorts of resources . . . . , that it is not consumed or lost in exchange but becomes 'possessed'

[1]It has to be noted that Friedkin models opinion formation, not information diffusion. Although Friedkin's model resembles Yamaguchi's information diffusion model, Friedkin's interpretation of the elements in the model is different. Although Friedkin's model is equivalent with a Markov chain, it cannot be interpreted in the sense that influence probabilities are transition probabilities between separate states in a Markov chain.

by both the transmitter and the receiver" (Laumann and Knoke 1987:192). We believe that it is a crucial property of the transfer of information that the transmitter does *not* lose the information by giving it to somebody else.

The new model makes the information transfer assumption more realistic. This paper also searches for differences in predictions between the old and new models. In this way, we can distinguish between situations in which the unrealistic assumption is truly problematic and leads to unreliable predictions and other situations in which the predictions of the different models are comparable.

If the information diffusion process is compared with the diffusion of an infectious disease, the model used by Yamaguchi assumes that an actor is infectious only for a short time and infects at most one other actor. In the new model, it is assumed that an actor who is infectious remains infectious permanently. This is of course another extreme, and in the research on epidemics there exist intermediate models (see, for example, Bailey 1975; Bartholomew 1982). Still, in relatively small networks where information spreads in a short time, the assumption that actors do not forget information during the diffusion of the information in the network seems sensible. To distinguish between the two models compared in this paper, the model used by Yamaguchi is referred to as the "transit" model and the new model as the "contagion" model. Both are variants of diffusion models, and with the introduction of these terms the word "diffusion" is reserved for situations that do not refer to one of the two models specifically.

Among several centrality measures that Friedkin (1991) described, two of them—namely, *immediate effects centrality* and *mediative effects centrality*, which are respectively centrality measures of closeness centrality and betweenness centrality (Freeman 1979)—are related to the transit model. Friedkin did not investigate in detail how his centrality measures depend on particular existing network measures. He only showed examples of networks with five actors. Yamaguchi (1994a) used mean first passage time, thereby implicitly assuming the transit model, to derive global measures of the inefficiency of information flow through networks, and he related these inefficiency measures to descriptive measures of networks such as density and number of bridges. In another paper, Yamaguchi (1994b) introduced a new group of accelerated failure-time regression models for diffusion processes and then applied the models to simulated network-diffusion data using the logarithm of the mean first passage time between

two actors in a network (a measure based on the transit model) to estimate the determinants of information diffusion time between the actors. In both models the results were plausible. Efficiency of information diffusion increased with network density and decreased with number of bridges. Actors with more ties needed less time to reach each other than actors with fewer ties.

Thus far, the results obtained with the transit model seem plausible, and it is possible that the unrealistic assumption is not problematic. However, the investigation of the consequences of this assumption has been limited, and the results of the transit model have not been compared with those of other models. We think that more extensive investigation is needed. Therefore, some intuition is offered to demonstrate that the "transit" assumption probably has undesirable implications. For example, the role of bridges is likely to be much more important in the case of a package that is passed from one actor to another than for the case of information that is transmitted. If a package reaches one side of a bridge but does not immediately cross the bridge, it will drift back into the part of the network from which it came and it can take a long time before the package returns to the bridge. Under the new assumption, once the information reaches one side of a bridge it will stay and can cross the bridge in every subsequent time period. Even more important is the role of the crucial variable network density. In Yamaguchi's article (1994a) global network density has a negative effect on the time it takes for information to spread through the network. The negative effect of density is expected to be larger under the new assumption, because if information stays with the transmitter, the total amount of information will build up quickly in a dense network and everybody will soon be informed. If information is sent around like a package, it can "get lost" in the large number of network ties in a dense network and the package may need a long time to reach certain actors.

Section 2 describes the new model for the diffusion process in two slightly different versions. Section 3 presents formal definitions of the dependent and independent variables used in the models and states conjectures about the effects of the different variables. In Section 4, we regress diffusion times generated with the different models at the global, dyadic, and individual levels on the network measures defined in Section 3 using a set of networks with seven actors. Section 5 argues that one has to be quite cautious using the model in cases when only dichotomous data about network ties are available. This section also gives a possible generalization of the model if more continuous values for tie strengths are available. Finally, Section 6 summarizes the results.

2. THE MODEL

The wide range of models describing diffusion processes includes overviews by Bailey, (1957, 1964, 1975), Bartholomew (1982), Mahajan and Peterson (1985), and Valente (1995). Especially in research on epidemics of infectious diseases, diffusion processes are studied in considerable detail, although the attention to effects of social structure has largely been ignored until recently (see Morris 1993). During the last decade, the modeling efforts in this area have been extended considerably toward explaining epidemics using structural properties of social networks (for example, Altmann 1993; Kretzschmar and Morris 1996). For spatial diffusion, structural factors were often introduced by assuming lattice and related models (Harris 1974; Mollison 1977). More recently, lattice models have been also used in the studies of evolutionary ecology for such topics as the speed of spatial invasion and the emergence of cooperation through spatial contacts (for example, Nowak and May 1992; Ellner et al. 1998).

Furthermore, in recent years a number of studies using hazard rate models have expanded the research on diffusion models (for example, Diekmann 1989; Strang 1991; Hedström 1994; Strang and Tuma 1994; Yamaguchi 1994b; Greve, Strang, and Tuma 1995). The Strang-Tuma model, in particular, explicitly takes into account the effects of relations between transmitters and receivers of information on the rate of diffusion. While hazard rate models are very useful in general for empirical research on diffusion, most of the previous studies were not directly concerned with the effects of network structure and network positions on the rate of diffusion except for the studies of Yamaguchi (1994a, 1994b). Yamaguchi used simulated network data to derive hypotheses about the network-structural determinants of diffusion efficiency. After describing the new contagion model, which differs from the old transit model, we also employ simulated network data to compare the network-structural predictors of diffusion efficiency between the new and old models.

2.1. *Definitions*, *Assumptions*, *and Main Theorem*

Consider a network with n actors. The $n \times n$ incidence matrix of this network is given by $\boldsymbol{N}$ with $n_{ij} = 1$ if a tie exists between actor i and actor j, and $n_{ij} = 0$ otherwise; $n_{ii} = 0$.[2] Only undirected networks are considered ($n_{ij} = n_{ji}$ for all i, j), although the model can be generalized to directed networks.

[2]Boldface is used to denote matrices (uppercase) and vectors (lowercase).

A second assumption is that the network is connected: a path exists from every actor in the network to every other actor. For disconnected networks, the diffusion process can be solved by applying the model to the different connected components of the network. For the calculation of diffusion times, we need more than just the network of ties. It would be preferable if contact probabilities w_{ij} were known between every pair of actors i and j per unit of time. However, this paper is based on the situation in which only $\boldsymbol{N}$ is given. Yamaguchi (1994a) discussed theoretical considerations that allow contact probabilities to be deduced from the interests of the actors in the network. Assuming that an actor is equally interested in communicating with each of his neighbors and that he divides all his resources equally among these actors, the contact probabilities are chosen such that

$$w_{ij} = \frac{n_{ij}}{\sum_{j=1}^{n} n_{ij}} \quad . \tag{1}$$

For a connected network $\sum_{i=1}^{n} n_{ij} > 0$ and, thus, w_{ij} is well defined.[3] Note that the fact that $\boldsymbol{N}$ is symmetric does not imply that $\boldsymbol{W}$ is symmetric. At the end of the paper it will be shown that this definition of the contact probabilities is not self-evident. However, because this paper emphasizes the influence of the information transfer assumption on the implications of the model, the same definition is used for the contact probabilities as was used by Yamaguchi before, to avoid any suggestion that the implications change because of changes in this definition.

Knowing the probability that actor i communicates to another actor j in a given time period, we want to calculate expected diffusion times under different assumptions. Expected diffusion time refers to a number of distinct events in a network. These events will be discussed in detail later on. Examples are the expected time necessary to transmit information from actor i to actor j (dyadic diffusion time) or the expected time needed for information to reach every actor in a network if the information starts from one randomly chosen actor in the network (global diffusion time). Yamaguchi described how to calculate what is called *mean first passage time* (MFPT) at the global and dyadic levels for the transit model. Similarly, *mean contagion time* (MCT) will refer to the diffusion time for the con-

[3]Friedkin (1991) defined the diagonal elements of $\boldsymbol{N}$ to be equal to one in his example and then also used (1) to determine $\boldsymbol{W}$.

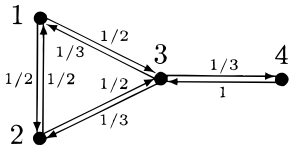
tagion model.[4] Friedkin's *immediate effects centrality* (IEC) and *mediative effects centrality* (MEC) are based on mean first passage time in a model equivalent with the transit model. For these centrality measures too, comparable measures will be introduced based on the contagion model.

The transit model is based on a Markov chain directly described by $\boldsymbol{W}$. This Markov chain has n states, and each state represents one actor who is able to *give* information to others. Each entry in $\boldsymbol{W}$ gives the probability that one actor will provide information to somebody else while *not* retaining the information himself. Thus the process represented by this Markov chain resembles passing a package between the actors in the network according to the probabilities in $\boldsymbol{W}$. The expected time for information to go from one actor to another is based on the expected number of steps this package needs to travel from one actor to another, including the possibility that the package will return to the original actor. Another way to observe that these models use Markov chains in a problematic way is by recalling the *Markov property*, which defines Markov chains (Kemeny and Snell 1960:24). The Markov property implies that to predict the state of a chain at time $t + 1$, one need only know the state at time t; the transition does not depend on what happened before time t. However, to predict how a diffusion process will proceed, one actually wants to know all actors who have been informed before; therefore, these Markov chains should have more than n states.

This does not imply that Markov chain theory cannot be used to describe the contagion model. On the contrary, it is a very useful theory if the necessary assumptions are made explicit. First, we assume that each actor in a network is either informed (i.e., he obtained the considered information at any time in the past) or not informed. Second, we assume that nobody ever forgets information once obtained. This assumption is not necessary to define the Markov chain, but it seems a plausible assumption for the process under study.[5] Now, a Markov chain is defined in which the states are elements of the power set of actors in the network; i.e., each subset of actors in the network that is informed forms a state in the new

[4]Although we prefer the term "mean diffusion time," we are forced to use consistent terminology to compare the different models and need the term "diffusion" in cases that do not refer to one of the two models specifically.

[5]Of course this is not a suitable assumption in, for example, the study of epidemics in which the time that an actor is infectious is short compared with the time the disease takes to spread through the population. Including the possibility that actors forget information or stop transmitting information is possible, but in that case all states have to be included and the diffusion process becomes considerably more complex.



$$\boldsymbol{W} = \begin{pmatrix} 0 & 1/2 & 1/2 & 0 \\ 1/2 & 0 & 1/2 & 0 \\ 1/3 & 1/3 & 0 & 1/3 \\ 0 & 0 & 1 & 0 \end{pmatrix}$$

FIGURE 1. Example network.

Markov chain. We consider the example of the network in Figure 1. The power set $\mathcal{S}$ for this example has $2^4 = 16$ elements,

$$\mathcal{S} = \big\{\varnothing,\{1\},\{2\},\{3\},\{4\},\{1,2\},\{1,3\},\{1,4\},\{2,3\},\{2,4\}\ , \\ \{3,4\},\{1,2,3\},\{1,2,4\},\{1,3,4\},\{2,3,4\},\{1,2,3,4\}\big\}\ , \tag{2}$$

where each element represents the set of actors who are informed in this state of the Markov chain. $\mathcal{S}$ has 2^n elements for a network with n actors. Fortunately, it is not necessary to consider all states in every analysis of such networks. For example, there is no need to consider the empty set $\varnothing$, because if nobody has information, nobody will obtain information. At least one actor has to have information to start the information process. The assumptions imply that the originally informed actor is always informed. Thus, we need only consider the states in which this actor is informed, which reduces the number of essential states to 2^{n-1}. Because the index of the actor where the information starts is only a label, this actor can always be given the number 1 without loss of generality. For special situations the number of states can be reduced even further.[6]

[6]E.g., for the network in Figure 1, if the information starts from actor 1, then actor 4 can be reached only through actor 3. Therefore, the states {1,4} and {1,2,4} will never occur during a diffusion process starting from actor 1. This implies that they do not play a role in the diffusion process and can be omitted. Furthermore, if there are groups of structurally equivalent actors—i.e., actors with identical contact probabilities with all other actors—only the number of these equivalent actors who are informed is important and not exactly which actors are informed. In the example network, actor 1 and actor 2 are structurally equivalent. This paper does not elaborate on this issue, although it is very relevant if one wants to apply the method to larger networks. Note that we assume in this paper that one actor is informed by an external source and that external sources of information do not play a role in the process of information diffusion. One can also assume that every actor with a certain probability is informed by an external source at each point in time. In that case one cannot omit, for example, the states {1,4} and {1,2,4} in the example.

To analyze the model, a transition matrix $\boldsymbol{T}$ has to be calculated that contains the transition probabilities between the states of the Markov chain at every point in time. However, some additional assumptions are necessary. Table 1 summarizes two sets of assumptions for MCT and compares them with assumptions for MFPT. In the multinomial version of MCT, we try to stay as close as possible to MFPT by allowing only one contact for an actor in every time period.[7] Except for the assumption that actors never forget information, the only additional assumption needed is that different actors act independently (*between-actor independence)*. This version of MCT implies that the sum of w_{ij} equals one where we can choose $w_{ii} = 1 - \sum_{j \neq i} w_{ij}$. Thus, in this case $\boldsymbol{W}$ is stochastic. The advantage of calculating $\boldsymbol{T}$ in this way is that the model stays as close as possible to the transit model. However, the disadvantage is the restriction that the w_{ij} add up to one. Therefore, we introduce a second version in which *within-actor independence* is assumed as well; i.e., whether an actor communicates with one of his neighbors in a certain time period does not depend on whether he communicates with one of his other neighbors. In this version, the sum of w_{ij} need not to be equal to one. This implies that if $\boldsymbol{W}$ is given, this matrix can be used directly. To compare the two versions of the contagion model a stochastic $\boldsymbol{W}$ is always used, defined with the help of $\boldsymbol{N}$, although a stochastic $\boldsymbol{W}$ is only necessary for the multinomial version.

The calculation of the transition matrix for the new Markov chain $\boldsymbol{T}$ is rather straightforward, although it becomes tedious for larger networks. We start with the assumption that an actor informs at most one other actor in each time period. Consider two states S_1 and S_2. Denote the probability that the process goes from S_1 to S_2 by $Pr(S_1 \rightarrow S_2)$. Then $Pr(S_1 \rightarrow S_2) = 0$ if $S_1 \not\subset S_2$, again because actors cannot forget information, which implies that the set of actors who have information can only increase. If $S_1 \subset S_2$ holds,

$$Pr(S_1 \rightarrow S_2) = \sum_{f: S_1 \rightarrow S_2} \prod_{i \in S_1} w_{i, f(i)} \ , \tag{3}$$

where we sum over all functions f with domain S_1 and a range $f(S_1)$ for which holds $S_2 \backslash S_1 \subset f(S_1) \subset S_2$.[8] In words, we enumerate all possible

[7] This version is called "multinomial" because the selection of receivers by the transmitter of information follows a multinomial distribution. In the second version the selection follows a product-binomial distribution.

[8] $S_2 \backslash S_1$ is the notation for the set of elements that includes all elements of S_2 that are not elements of S_1.

TABLE 1
Overview of the Key Assumptions for Different Diffusion Time Models

Mean first passage time (MFPT)	Mean contagion time, "multinomial" version (MCT^M)	Mean contagion time, "product-binomial" version (MCT^{PB})
1. The informed actor informs at most one actor in every time period with probabilities w_{ij}.[a]	1. Each informed actor informs at most one actor in every time period with probabilities w_{ij} that are independent for all i (*between-actor independence*).	1. Each informed actor informs the other actors with probabilities w_{ij} that are independent for all i and j (*within-* and *between-actor independence*).
2. For every actor i, it holds that $\sum_j w_{ij} = 1$ and w_{ii} is the probability that actor i does not inform anybody if he is informed.		2. For all i, j, it holds that $0 \leq w_{ij} \leq 1$.[b]
3. An actor who informs another actor loses his information afterward.	3. An actor who is informed at any time will never forget the information.	
4. The w_{ij} are independent of the history of the diffusion process.		

[a] Because of assumption 3, there is always exactly one actor in the network informed under this set of assumptions.

[b] To compare this set of assumptions with the other two, the w_{ij} are chosen to be the same for all three cases in this paper. However, under this set of assumptions, the w_{ij} can be chosen less restrictively to represent, for example, empirical contact probabilities in certain time periods.

ways for the informed actors in S_1 to inform exactly the uninformed actors in $S_2 \backslash S_1$. Informed actors can also transmit information again to each other. $Pr(S_1 \to S_2) = 0$ if the number of actors in $S_2 \backslash S_1$ is larger than the number of actors in S_1, because for a function (as it was assumed to be) one element in the domain is assigned to exactly one element in the range.

For the network in Figure 1 using the probability matrix $\boldsymbol{W}$, this results in the following transition matrix $\boldsymbol{T}_M$ (the subscript M indicates the multinomial version):

$$\boldsymbol{T}_M = \begin{array}{c} \\ \{1\} \\ \{1,2\} \\ \{1,3\} \\ \{1,2,3\} \\ \{1,4\} \\ \{1,2,4\} \\ \{1,3,4\} \\ \{1,2,3,4\} \end{array} \begin{array}{c} \begin{array}{cccccccc} \{1\} & \{1,2\} & \{1,3\} & \{1,2,3\} & \{1,4\} & \{1,2,4\} & \{1,3,4\} & \{1,2,3,4\} \end{array} \\ \begin{pmatrix} 0 & 1/2 & 1/2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1/4 & 0 & 3/4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1/6 & 1/2 & 0 & 0 & 1/6 & 1/6 \\ 0 & 0 & 0 & 2/3 & 0 & 0 & 0 & 1/3 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1/2 & 1/2 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1/3 & 2/3 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix} \end{array} \tag{4}$$

From this matrix and the graph that illustrates the Markov chain belonging to $\boldsymbol{T}_M$ in Figure 2, one sees that the states $\{1,4\}$ and $\{1,2,4\}$ could have been omitted. Markov chains constructed in this way have simple characteristics. As long as the network is connected, all the states are *transient* states, except the state in which everybody is informed, which is the unique *ab-*

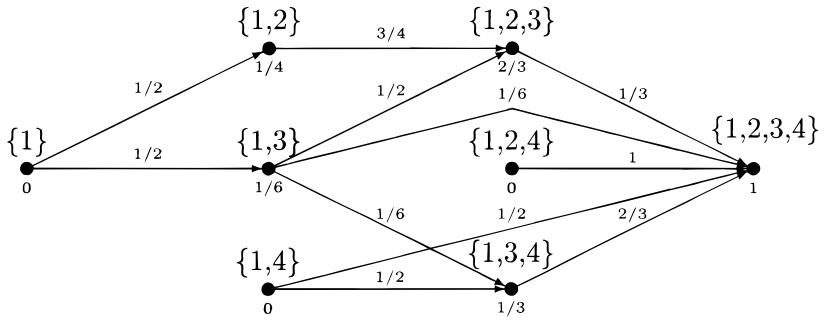


FIGURE 2. Markov chain belonging to the example network. (The probability at each node is the probability of staying at that node.)

sorbing state. Furthermore, once the process leaves a transient state, it can never return. The theory on absorbing Markov chains (Kemeny and Snell 1960:60, theorem 3.5.4.) is used to formulate the following theorem.

Theorem 1
Let $\boldsymbol{Q}$ be a matrix containing the rows and columns of $\boldsymbol{T}$ without the row and column for the absorbing state, and let actor 1 be the actor who obtained the information first and whose transition probabilities are given in the first row of $\boldsymbol{T}$. Let $\boldsymbol{I}$ be the identity matrix of appropriate order. Then the following properties hold:

1. $(\boldsymbol{I}-\boldsymbol{Q})^{-1}_{1j}$ is the expected number of times the process is in the j-th state before reaching the absorbing state.
2. $(\boldsymbol{I}-\boldsymbol{Q})^{-1}_{1+}$ is the expected total number of steps the process needs to reach the absorbing state.
3. The sum of the first row entries of $(\boldsymbol{I}-\boldsymbol{Q})^{-1}$ over all states in which a certain actor k is not informed is the expected number of steps the process needs to inform actor k.

Proof
Proofs for parts 1 and 2 can be found in Kemeny and Snell (1960, theorem 3.5.4). Part 3 is not true for absorbing chains in general, but it is true in this case because returning to any state is impossible. We will not present a formal proof here. The following intuition can be translated in a formal proof quite easily. To calculate the time needed to inform actor k, one considers the set of states in which actor k is informed as the new absorbing state. This implies that only a part of $(\boldsymbol{I}-\boldsymbol{Q})$ has to be inverted. However, because the probability of going from a state in which k is informed to a state in which k is not informed is always 0, the inverse of the suitable part of $(\boldsymbol{I}-\boldsymbol{Q})$ is exactly equal to the corresponding part of $(\boldsymbol{I}-\boldsymbol{Q})^{-1}$. ■

For the example network the first row of $(\boldsymbol{I}-\boldsymbol{Q}_M)^{-1}$ using $\boldsymbol{W}$ becomes

{1}	{1,2}	{1,3}	{1,2,3}	{1,4}	{1,2,4}	{1,3,4}.
1	$\frac{2}{3}$	$\frac{3}{5}$	$\frac{12}{5}$	0	0	$\frac{3}{20}$

(5)

This implies that the expected time to reach actor 2 from actor 1 equals $1+\frac{3}{5}+\frac{3}{20}=1\frac{3}{4}$; to reach actor 3 equals $1\frac{2}{3}$; to reach actor 4 equals $4\frac{2}{3}$; and to reach all actors equals $4\frac{49}{60}$.

The diffusion process as described above differs only in one aspect from MFPT—i.e., that information conveyed by the transmitter is also kept by the transmitter. Furthermore, actors communicate at most (exactly, if $w_{ii} = 0$) to one actor in every time period. This was a necessary assumption to treat $\boldsymbol{W}$ directly as a Markov chain. We can change that assumption now. If it is assumed that one actor i communicates with the other actors in one time period independently, $\boldsymbol{T}_{PB}$ can be calculated for every $\boldsymbol{W}$ with $0 \leq w_{ij} \leq 1$ for all i and j.

Consider two states: S_1 and S_2. Denote the probability that the process goes from S_1 to S_2 by $Pr(S_1 \rightarrow S_2)$. Then $Pr(S_1 \rightarrow S_2) = 0$ if $S_1 \not\subset S_2$, because actors cannot forget information, which implies again that the set of actors who have information can only increase. For $S_1 \subset S_2$,

$$Pr(S_1 \rightarrow S_2) = \prod_{j \in S_2 \setminus S_1} \left(1 - \prod_{i \in S_1} (1 - w_{ij})\right) \prod_{j \notin S_2} \left(\prod_{i \in S_1} (1 - w_{ij})\right) . \qquad (6)$$

This is quite easy to understand. The first part expresses the probability that for each actor j who will be one of the newly informed, at least one of the already informed actors informs him. The second part expresses the probability that none of the informed actors in S_1 informs any actor not in S_2. Because all these events are independent and must be true at the same time, their respective probabilities have to be multiplied. Furthermore, it is important to see that the diagonal elements of $\boldsymbol{W}$ are not used to calculate $\boldsymbol{T}_{PB}$. Thus the extent to which an actor "talks to himself" does not influence the diffusion process.

For the network in Figure 1, this results in the following transition matrix:

$$\boldsymbol{T}_{PB} = \begin{array}{c} \\ \{1\} \\ \{1,2\} \\ \{1,3\} \\ \{1,2,3\} \\ \{1,4\} \\ \{1,2,4\} \\ \{1,3,4\} \\ \{1,2,3,4\} \end{array} \begin{array}{c} \begin{array}{cccccccc} \{1\} & \{1,2\} & \{1,3\} & \{1,2,3\} & \{1,4\} & \{1,2,4\} & \{1,3,4\} & \{1,2,3,4\} \end{array} \\ \left(\begin{array}{cccccccc} 1/4 & 1/4 & 1/4 & 1/4 & 0 & 0 & 0 & 0 \\ 0 & 1/4 & 0 & 3/4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 2/9 & 4/9 & 0 & 0 & 1/9 & 2/9 \\ 0 & 0 & 0 & 2/3 & 0 & 0 & 0 & 1/3 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1/2 & 1/2 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1/3 & 2/3 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{array} \right) \end{array} \qquad (7)$$

Theorem 1 applies also to this case. For the example network, the first row of $(\boldsymbol{I} - \boldsymbol{Q}_{PB})^{-1}$ becomes

$$\begin{array}{cccccccc} \{1\} & \{1,2\} & \{1,3\} & \{1,2,3\} & \{1,4\} & \{1,2,4\} & \{1,3,4\}. \\ \frac{4}{3} & \frac{4}{9} & \frac{3}{7} & \frac{18}{7} & 0 & 0 & \frac{1}{14} \end{array} \tag{8}$$

According to Theorem 1, this implies that the expected time to reach actor 2 from actor 1 equals $\frac{4}{3} + \frac{3}{7} + \frac{1}{14} = 1\frac{5}{6}$; to reach actor 3 equals $1\frac{7}{9}$; to reach actor 4 equals $4\frac{7}{9}$; and to reach all other actors equals $4\frac{107}{126}$. Thus the expected contagion time is somewhat larger for the product-binomial selection of receivers than for the multinomial selection.

2.2. *Some Analytic Results for the New Model*

With the methods and theorem described above, it is possible to calculate contagion times for the contagion model. To find relations between contagion times and simple network measures, the first approach is to calculate these relations analytically. In this subsection, we give some illustrations showing that analytic results are complex and hard to interpret even for small networks.

The first analytic results are related to the comparison between the multinomial and product-binomial versions of the contagion model. In Section 4, it will be shown that the two models hardly differ in their implications. In all the networks we calculated, contagion time in the multinomial version is equal to or marginally smaller than it is in the product-binomial version. We could not prove that this result holds for all networks, but two related results are presented below. First, for trees—i.e., connected graphs with n nodes and $n - 1$ vertices (for example, see Wasserman and Faust 1994:119–20), the two versions result in exactly the same contagion times. The reasoning behind the proof is the following. In a tree exactly one possible path exists for information to travel between a pair of nodes. We can disregard paths in which information goes back and forth between actors because that does not occur in the information diffusion process as defined in the contagion model. For a certain tie in the network, say between actor i and actor j, the expected time needed for information to cross that tie after information has reached actor i equals $1/w_{ij}$ for the multinomial as well as for the product-binomial selection of receivers. The expected time needed for information to be transferred between i and j does not depend on which other actors are or have been informed during the diffusion process. Therefore, the expected time needed to traverse a

certain path is the sum of the expected times for each tie in that path. Consequently, the expected time needed for information to travel from actor *i* to actor *j* in a tree is equal for both versions of the contagion model

$$\mathrm{MCT}_{ij} = \frac{1}{w_{ik_1}} + \frac{1}{w_{k_1k_2}} + \cdots + \frac{1}{w_{k_tj}} \quad , \tag{9}$$

where $k_1, \ldots, k_t$ are the nodes that necessarily have to be passed to reach *j* from *i*.[9] This is an appealing result because it closely fits the intuition that the diffusion process is straightforward if only one path for information diffusion exists. Results are not this simple for the transit model because information can go back and forth, even in trees.

For networks more complicated than trees, analytic expressions become fairly complex. For a three-actor network in which information starts from actor *i*, the expected time to reach actor *j* equals for the product-binomial distribution

$$\mathrm{MCT}_{ij}^{PB} = \frac{1}{1-(1-w_{ij})(1-w_{ik})} + \frac{w_{ik}(1-w_{ij})}{(1-(1-w_{ij})(1-w_{ik}))(1-(1-w_{ij})(1-w_{kj}))} \quad , \tag{10}$$

where *k* is the third actor in the network. For the multinomial distribution, the expected time equals

$$\mathrm{MCT}_{ij}^{M} = \frac{1}{w_{ij}+w_{ik}} + \frac{w_{ik}}{(w_{ij}+w_{ik})(1-(1-w_{ij})(1-w_{kj}))} \quad . \tag{11}$$

Straightforward calculation shows that $\mathrm{MCT}_{ij}^{PB} \geq \mathrm{MCT}_{ij}^{M}$ and that equality holds if and only if $w_{ij} = 1$ or one of the ties in the triad does not exist—i.e., if the graph is a tree.

The two foregoing analytic results enable us to calculate directly expected contagion times for the network in Figure 1. For example, because the only way for information to reach actor 4 is through actor 3, MCT from actor 1 to actor 4 equals MCT from actor 1 to actor 3 plus the time needed for information to go from actor 3 to actor 4. Both contagion

[9]This implies that if we would approximate contagion time for $\boldsymbol{W}$ in a "neighborhood" of $\boldsymbol{I}$ with the help of linearization, we obtain similar expressions using the shortest paths, because probabilities of longer paths tend to zero. Note also that these expressions do not hold if information may start with more than one actor at the same time.

times are independent and are deduced from contagion times for the smaller subnetworks. This implies that MCT from actor 1 to actor 4 in the example network for the version using the multinomial distribution equals

$$\mathrm{MCT}_{14}^{M} = \frac{1}{w_{12} + w_{13}} + \frac{w_{12}}{(w_{12} + w_{13})(1 - (1 - w_{13})(1 - w_{23}))} + \frac{1}{w_{34}} \quad . \tag{12}$$

Thus for networks without loops other than triads, we can calculate contagion times easily. However, for a cycle with four actors the situation is complex enough to refrain from presenting it here. For a complete four-actor network, the expressions exceed one page; it resisted and discouraged attempts at interpretation.

The examples given above show that analytic expressions for contagion times become complex for small networks. An additional step is needed to link these expressions to simple network measures, such as density or degree. Therefore, in Section 4 we will use an approach that has been used before by Yamaguchi (1994a, 1994b) and Buskens (1998). We will calculate contagion times and network measures for a given set of networks. Then, with the help of statistical models, we will try to find comparative statics for the relations between contagion times and network measures.

3. VARIABLES AND MODEL COMPARISONS

In this section we formalize the dependent and independent variables to compare the different models. Conjectures are presented about the effects of the independent variables on the diffusion time and about the differences in these effects between the different diffusion models.[10]

3.1. *Dependent Variables*

We introduce comparable dependent variables in this section to compare the models distinguished in Section 2. The dependent variables will be defined at three levels: (1) global, (2) dyadic, and (3) individual.

[10]We use the term "conjecture" here because we discuss intuitions about the mathematical implications of theoretical models. This is in contradistinction to the term "hypothesis," which is used for a theoretical prediction about an empirical situation. Only if the analyses below show that the conjectures actually follow from the model do the conjectures become hypotheses.

3.1.1. *Dependent Variables for the Transit Model*
We define dyadic MFPT from actor i to actor j: MFPT_{ij}. This is the expected time it takes to transmit information from actor i to actor j in the transit model. For the exact way to calculate MFPT_{ij}, we refer to Yamaguchi (1994a). By definition $\mathrm{MFPT}_{ii} = 0$ for all i. *Individual mean first passage time* is the average of MFPT_{ij} over all j:

$$\mathrm{MFPT}_i = \frac{\sum_{j=1}^{n} \mathrm{MFPT}_{ij}}{n-1} \quad . \tag{13}$$

If $\boldsymbol{W}$ is not symmetric, MFPT_i is not equal to the average time needed for actor i to receive information, which we call *individual mean first receiving time*. Formally,

$$\mathrm{MFRT}_i = \frac{\sum_{j=1}^{n} \mathrm{MFPT}_{ji}}{n-1} \quad . \tag{14}$$

Friedkin (1991) defines *immediate effects centrality* (IEC_i), which equals the inverse of MFRT_i. Furthermore, Friedkin defines a betweenness centrality measure based on the transit model called *mediative effects centrality* (MEC_i), which "indicates the extent to which an actor transmits the total effects of other actors" (p. 1490). We will define a similar measure for the contagion model later on. To obtain *global* MFPT, we average all MFPT_{ij} over i and j:

$$\mathrm{GMFPT} = \frac{\sum_{i=1}^{n} \sum_{j=1}^{n} \mathrm{MFPT}_{ij}}{n(n-1)} = \overline{\mathrm{MFPT}_i} = \overline{\mathrm{MFRT}_i} \quad . \tag{15}$$

3.1.2. *Dependent Variables for the Contagion Model*
We now turn to the contagion model. For the multinomial and product-binomial versions, the definitions of contagion times are the same. We call the expected time it takes to diffuse information from actor i to actor j in the contagion model *dyadic mean contagion time* (MCT_{ij}), which is a measure of the distance between two actors in the network. The exact way to calculate MCT_{ij} is presented in Section 2. The calculation of MCT_{ij} implies that actor i is the first actor to be informed and then the expected time to reach actor j is determined. Because other starting situations, in

which more actors are informed initially, are possible, one might ask whether this is the best starting position. For the transit model, only one actor is informed at any time; therefore, starting with one informed actor seems to be most closely related to dyadic mean first passage time. Moreover, it is the best building block for constructing individual and global contagion times. Namely, for these contagion times, we are interested in what happens with the information if it starts from a particular or a random actor.

For an actor i, one can average MCT_{ij} over all other actors. This is called *individual mean contagion time*:

$$\mathrm{MCT}_i = \sum_{j=1}^{n} \frac{\mathrm{MCT}_{ij}}{n-1} \quad . \tag{16}$$

A straightforward alternative to this diffusion measure is the expected time needed for information to reach the absorbing state in which everyone is informed, when information starts from a certain actor. The disadvantage of this alternative measure is that one cannot determine whether many actors are informed early in the diffusion process and few at the end or the other way round. The intuition is that individual mean contagion time should be smaller for an actor who informs many actors early in the diffusion process. This problem could be cured by weighting a period of time before absorption with the expected number of actors not informed in that time period. Thus, a "second" definition of individual mean contagion time would be the following:

$$\widehat{\mathrm{MCT}}_i = \sum_{j=1}^{2^{n-1}-1} \frac{u_j q_{1j}}{n-1} \quad ,$$

where q_{1j} is the j-th element of the first row of the matrix $\boldsymbol{Q}$ and u_j is the number of actors who are not informed in the state that corresponds to the j-th column in the matrix $\boldsymbol{Q}$. However, it can be proven that this alternative definition is equivalent to the first one—i.e., $\mathrm{MCT}_i = \widehat{\mathrm{MCT}}_i$. The reason is that each $q_{1j}/(n-1)$ occurs exactly u_j times in MCT_{ij}. Because of the parallel with the transit model, we refer mostly to the first definition of MCT_i.

Again, if $\boldsymbol{W}$ is not symmetric, the expected time for transmitting information is not equal to the expected time for receiving information, and we again need a reciprocal concept, *individual mean receiving time*:

$$\mathrm{MRT}_i = \sum_{j=1}^{n} \frac{\mathrm{MCT}_{ji}}{n-1} \quad . \tag{17}$$

Global diffusion time based on the contagion model is *global mean contagion time* and is the average of all dyadic contagion times:

$$\mathrm{GMCT} = \frac{\sum_{i=1}^{n}\sum_{j=1}^{n}\mathrm{MCT}_{ij}}{n(n-1)} = \overline{\mathrm{MCT}_i} = \overline{\mathrm{MRT}_i} \ . \tag{18}$$

Individual mean contagion time and individual mean receiving time are measures of a certain actor's ability to transmit and receive information quickly through the network. We use these measures directly to introduce related centrality measures, *contagion transmitter centrality* and *contagion receiver centrality*:

$$\mathrm{CTC}_i = \frac{1}{\mathrm{MCT}_i} \ , \tag{19}$$

$$\mathrm{CRC}_i = \frac{1}{\mathrm{MRT}_i} \ . \tag{20}$$

Both centrality measures are closely related to closeness centrality as defined by Freeman (1979) and immediate effects centrality as introduced by Friedkin (1991).[11]

The concept of betweenness centrality is somewhat more difficult to address via the contagion model. We define $\boldsymbol{W}^{-i}$ as the contact probability matrix in which actor i does not communicate with any of the other actors. Thus $w_{ii}^{-i} = 1$ and $w_{ij}^{-i} = 0$ if $i \neq j$. In all other rows $\boldsymbol{W}^{-i}$ equals $\boldsymbol{W}$.[12] We now define MCT_{jk}^{-i} as dyadic mean contagion time for $\boldsymbol{W}^{-i}$ from j to k. The difference between contagion time if actor i does not inform others and contagion time if actor i does inform others is a measure of the dyadic betweenness of actor i. We define

$$p_{ijk} = \frac{\mathrm{MCT}_{jk}^{-i} - \mathrm{MCT}_{jk}}{\mathrm{MCT}_{jk}^{-i}}, \quad i \neq j \neq k \tag{21}$$

[11]The new closeness centrality measures can be considered generalizations of Freeman's closeness centrality, because CTC_i and CRC_i are exactly Freeman's measure if $\boldsymbol{W} = \boldsymbol{N}$. Note that Freeman defines his closeness measure only for symmetric networks, which means that CTC_i and CRC_i are identical in that case.

[12]Although $\boldsymbol{W}^{-i}$ is not related to a symmetric $\boldsymbol{N}$, it is related to a network in which the ties between actor i and the others are directed toward actor i.

as the extent to which actor i affects the flow of information from actor j to actor k; $p_{ijk} \in [0,1]$. It is possible that actor j can no longer reach actor k if actor i ceases to transmit information. In that case we define $p_{ijk} = 1$. Now we define *contagion betweenness centrality* as the mean dyadic betweenness of actor i,

$$\mathrm{CBC}_i = \frac{\sum_{j \neq i} \sum_{k \neq j, k \neq i} p_{ijk}}{(n-2)(n-1)} \quad . \tag{22}$$

CBC_i has some nice properties. For the central actor in a star network, this betweenness measure equals one. The betweenness measure is zero for an actor who has only one tie and thus does not take a position between any two actors. All other actors will obtain values between zero and one. Table 2 presents an overview of the dependent variables.

3.2. *Independent Variables*

This subsection discusses the independent variables used in the different analyses. All the independent variables are relatively simple network measures. We will make conjectures about the effects expected from the network measures on the different dependent variables. The independent variables are all based on $\boldsymbol{N}$, such that almost all network measures correspond to the measures used by Yamaguchi (1994a, 1994b). Because the network measures are explicitly defined in terms of n_{ij}, it is possible to generalize them in terms of w_{ij}. The discussions in Section 5 will show why that can be preferable. We present not only conjectures about effects of the independent variables on the different diffusion times but also conjectures about the differences between the transit model and the contagion model.

The independent variables can be divided in three levels: (1) dyadic, (2) individual, and (3) global.

3.2.1. *Independent Variables at the Dyadic Level*

At the dyadic level we have only one independent variable—namely, the *distance* between the two actors involved. The distance between two actors is the shortest path length between these two actors. We assumed that all actors are connected. Formally, the distance from actor i to actor j can be defined in terms of the product-binomial version of the contagion model:

$$\mathrm{Distance}_{ij} = \mathrm{MCT}_{ij}^{PB} \text{ if we choose } \boldsymbol{W} = \boldsymbol{N}.^{13} \tag{23}$$

[13]Note that this independent variable is not equal to dyadic contagion time, which is calculated with a $\boldsymbol{W}$ that is certainly not equal to $\boldsymbol{N}$.

TABLE 2
Overview of Diffusion Times at Different Levels

MEAN FIRST PASSAGE TIME		
Global level	Global mean first passage time	$\mathrm{GMFPT} = \frac{\sum_i \sum_j \mathrm{MFPT}_{ij}}{n(n-1)}$
Dyadic level	Dyadic mean first passage time	MFPT_{ij}
Individual level	Individual mean first passage time	$\mathrm{MFPT}_i = \frac{\sum_j \mathrm{MFPT}_{ij}}{n-1}$
	Individual mean first receiving time	$\mathrm{MFRT}_i = \frac{\sum_j \mathrm{MFRT}_{ji}}{n-1}$
	Immediate effects centrality	$\mathrm{IEC}_i = \frac{1}{\mathrm{MFRT}_i}$
	Mediative effects centrality	MEC_i
MEAN DIFFUSION TIME		
Global level	Global mean contagion time	$\mathrm{GMCT} = \frac{\sum_i \sum_j \mathrm{MCT}_{ij}}{n(n-1)}$
Dyadic level	Dyadic mean contagion time	MCT_{ij}
Individual level	Individual mean contagion time	$\mathrm{MCT}_i = \frac{\sum_j \mathrm{MCT}_{ij}}{n-1}$
	Contagion transmitter centrality	$\mathrm{CTC}_i = \frac{1}{\mathrm{MCT}_i}$
	Individual mean receiving time	$\mathrm{MRT}_i = \frac{\sum_j \mathrm{MCT}_{ji}}{n-1}$
	Contagion receiver centrality	$\mathrm{CRC}_i = \frac{1}{\mathrm{MRT}_i}$
	Contagion betweenness centrality	$\mathrm{CBC}_i = \sum_j \sum_k \frac{\mathrm{MCT}_{jk}^{-i} - \mathrm{MCT}_{jk}}{(n-1)(n-2)\mathrm{MCT}_{jk}^{-i}}$

The reason is that by setting all contact probabilities equal to one, information will traverse all ties connected to informed actors in all time periods; therefore, the expected dyadic contagion time for information transmission from actor *i* to actor *j* equals the minimal number of steps needed between *i* and *j*.

This variable applies only in analyses where the dependent variable is at the dyadic level. The conjecture about the effect of distance does not need extensive elaboration. The farther two actors are apart, the longer it will take to convey information from one to the other. For both models, we expect distance to be a predominant predictor of dyadic diffusion time. Nevertheless, in the contagion model the probability that the shortest path will be used for diffusion is relatively high, while in the transit model this probability is relatively low, for in the transit model every deviation from the shortest path generates a completely different path to reach an actor. Therefore, we predict that the effect of distance is larger for the contagion model than for the transit model.

Conjecture 1 *Dyadic diffusion time increases with the distance between the two actors involved.*[14]
Conjecture 2 *The positive effect of distance on dyadic diffusion time is larger in the contagion model than in the transit model.*

3.2.2. *Independent Variables at the Individual Level*
We use four individual independent variables. For the dyadic dependent variables, these network measures form pairs of independent variables, because the measures for both actors are relevant. These independent variables do not apply to global diffusion times. The first variable is the *degree* of the actor. Degree is defined as the proportion of actual ties among possible ties:

$$\text{Degree}_i = \frac{\sum_{j=1}^{n} n_{ij}}{n-1} \quad . \tag{24}$$

Because the networks used are undirected, indegree and outdegree cannot be distinguished. For directed networks, the above formula gives the outdegree, and the indegree can be obtained by summing over i. For both transmitting and receiving information, we think that a large degree is advantageous.

Conjecture 3 *Individual and dyadic diffusion time decrease with, respectively, the degree of the focal actor and the degree of an actor of the dyad.*

[14]If we write "diffusion time" without direct reference to a comparison between the contagion model and the transit model, we refer to diffusion time in general, including mean contagion time and mean first passage time.

The second individual variable is *individual local density*. This variable is defined for each actor as the extent to which ties exist between two actors connected to the focal actor:

$$\text{Local density}_i = \frac{\sum_{j=1}^{n} \sum_{k \neq j} n_{ij}\, n_{ik}\, n_{jk}}{\sum_{j=1}^{n} \sum_{k \neq j} n_{ij}\, n_{ik}} \quad . \tag{25}$$

Individual local density is only defined by this formula if actor *i* has more than one tie. If an actor has only zero or one tie, individual local density is defined to be zero. There is one important effect of local density. High local density has the potential to trap information in a "corner" of the network and can therefore hinder information diffusion to other parts of the network. Because of the difference between the transit model, in which information really travels around, and the contagion model, in which information builds up, we expect that the effect of trapping information will be more severe for the transit model than for the contagion model. We expect that trapping information in the region of the transmitter will increase diffusion time, while the possibility of trapping information in the region of the receiver will decrease diffusion time. The following conjectures are consequences of this reasoning.

Conjecture 4 *Individual mean contagion time, individual mean first passage time, and dyadic diffusion time increase with the individual local density of the transmitter of information.*
Conjecture 5 *Individual mean receiving time, individual mean first receiving time, and dyadic diffusion time decrease with the individual local density of the receiver of information.*
Conjecture 6 *All predicted effects of individual local density are larger (in the absolute sense) for the transit model than for the contagion model.*

The third individual variable is *degree quality*, which is closely related to prominence measures (Burt 1976). The measure is formally defined for actor *i* as the covariance of whether actor *i* has a tie with actor *j* and the degree of actor *j* over all other actors *j*:

$$\text{Degree quality}_i = \sum_{j \neq i} \frac{n_{ij}\, n_j}{n-1} - \sum_{j \neq i} \frac{n_{ij}}{n-1} \sum_{j \neq i} \frac{n_j}{n-1} \quad . \tag{26}$$

Because degree quality is an indication of the number of actors an actor can reach in two steps, relative to his own number of ties, we think that high degree quality is also advantageous for information diffusion.

Conjecture 7 *Individual and dyadic diffusion time decrease with, respectively, the degree quality of the focal actor and the degree quality of an actor of the dyad.*

The final independent variable at the individual level is the number of bridges at which an actor is located. A bridge is a tie in the network that satisfies two conditions. First, if this tie is removed, the network is not connected anymore; thus, information can no longer be communicated between these two parts of the network. Second, the two parts that are created by removing the tie both contain at least two actors. Because actors at bridges have direct access to different parts of the network, they will be faster at transmitting information. Moreover, actors at bridges have to be passed in order for information to travel from one part of the network to another; therefore, they will receive information earlier than others. In the introduction we already argued that this effect is larger for the transit model than for the contagion model.

Conjecture 8 *Individual and dyadic diffusion time decrease with the number of bridges at which the focal actor(s) is located.*
Conjecture 9 *The negative effect of the number of bridges at which an actor is located on individual and dyadic diffusion time is larger (in the absolute sense) for the transit model than for the contagion model.*

Of course, the number of bridges at which an actor is located is closely related to the concept of betweenness centrality and structural holes (Burt 1991), although Burt emphasizes essential actors, while we emphasize essential ties. We did not develop any conjectures about the effects of other independent variables on betweenness centrality measures, because for most variables it is not straightforward. For the number of bridges at which an actor is located it is almost obvious.

Conjecture 10 *Betweenness centrality increases with the number of bridges at which an actor is located.*

3.2.3. *Independent Variables at the Global Level*
Finally, we use five independent variables at the global level. The first one is *global density*, the proportion of ties in the network:

$$\text{Global density} = \sum_{i=1}^{n} \sum_{j=1}^{n} \frac{n_{ij}}{n(n-1)} \quad . \tag{27}$$

If all ties have equal probability of transferring information, the conjecture that global density increases efficiency of information transfer is straightforward. However, for $\boldsymbol{W}$ in which the expected number of information transfers for each actor is one in each time period, this conjecture is not self-evident. The choice of $\boldsymbol{W}$ implies that global density is not a measure of the amount of information circulating through the network but of the number of possible ways for information to be transferred between all the actors in the network. Nevertheless, Yamaguchi (1994a, 1994b) obtained negative effects for global density. However, because Yamaguchi (1994b) did not include distance, it is questionable whether we will replicate his result. We expect that global density promotes diffusion if we take the average over all diffusion times—i.e., for GMCT and GMFPT. But for individual and certainly dyadic diffusion times the effects are less clear. More ties provide additional possibilities for the transmission of information, and information can choose paths other than the shortest path. This effect will be larger for the transit model than for the contagion model, because in the transit model deviating from the shortest path really means taking a detour, while in the contagion model it only means waiting for the moment when the shortest path can be continued.

Conjecture 11 *Global, individual, and dyadic diffusion time decreases with global density.*
Conjecture 12 *The negative effect of global density on global, individual, and dyadic diffusion time decreases (in the absolute sense) with the level of aggregation of diffusion time—i.e., going from global diffusion time via individual diffusion time to dyadic diffusion time.*
Conjecture 13 *The negative effect of global density on global, individual, and dyadic diffusion time decreases (in the absolute sense) if we move from the contagion model to the transit model, especially for individual and dyadic diffusion time.*

The second global variable is *transitivity*—the proportion of all ordered triads i, j, k for which the ties (i, j), (i, k), and (j, k) exist among all ordered triads for which the ties (i, j) and (i, k) exist.[15] Formally,

[15]Unfortunately, this definition is different from the two definitions for system-level local density given by Yamaguchi, although it correlates highly (.975 in the data

$$\text{Transitivity} = \frac{\sum_{i=1}^{n}\sum_{j=1}^{n}\sum_{k=1}^{n} n_{ij}\, n_{ik}\, n_{jk}}{\sum_{i=1}^{n}\left(\sum_{j=1}^{n}\sum_{k\neq j} n_{ij}\, n_{ik}\right)} \quad . \tag{28}$$

In a network in which no actor has more than one tie (for connected networks this can only be a network with two actors), transitivity is zero. By an argument similar to that for individual local density, transitivity will cause trapping of information in some parts of the network. Therefore, transitivity will slow down information diffusion for global diffusion time, and the effect will be larger for the transit model. Similar effects are expected for individual and dyadic diffusion time.

Conjecture 14 *Global, individual, and dyadic diffusion times increase with transitivity.*
Conjecture 15 *The positive effect of transitivity on global, individual, and dyadic diffusion times is larger for the transit model than for the contagion model.*

The third global variable is the *number of bridges* in the whole network. Because bridges form bottlenecks for information transfer through the network, we expect diffusion times to increase with the number of bridges in the network. As we explained in the introduction, we expect this effect to be larger for the transit model than for the contagion model.

Conjecture 16 *Global, individual, and dyadic diffusion time increase with the number of bridges in the network.*
Conjecture 17 *The positive effect of bridges on global, individual, and dyadic diffusion time is larger for the transit model than for the contagion model.*

used later in the paper) with the "1994b" definition. One advantage of this definition is that it is more easily expressed in terms of the n_{ij}. A second advantage is that it is a weighted sum of the individual local densities, which will be defined later, and in this sense it is an improvement on Yamaguchi's "1994a" definition because it weights the individual local densities in such a way that all ordered triads contribute equally to transitivity. All these definitions lead to virtually the same results in the analyses. Because the new definition differs slightly from earlier definitions of system-level local density and to avoid confusion with individual local density, we use the term "transitivity," which is certainly appropriate.

The fourth global variable is *diameter*, which is defined as the maximum of the distances between all pairs of actors in the network:

$$\text{Diameter} = \max_{i,j} \text{distance}_{ij} \ . \tag{29}$$

We use the diameter only to explain individual and global diffusion measures. It does not add explanatory power beyond that provided by the distance between two actors for dyadic diffusion times. We expect that for larger diameters diffusion times are larger because the actors are more dispersed. By an argument similar to that for number of bridges, we expect that especially for the transit model information has many possible ways to wander around if the diameter is large.

Conjecture 18 *Global and individual diffusion time increase with the diameter of the network.*
Conjecture 19 *The positive effect of diameter on global and individual diffusion time is larger for the transit model than for the contagion model.*

The last global variable is *coefficient of variation in centrality* (CVC) used by Yamaguchi (1994a). CVC is formally defined as

$$\text{CVC} = \frac{\sqrt{\sum_{i=1}^{n} \left(n_i - \sum_{i=1}^{n} \frac{n_i}{n-1} \right)^2}}{\sum_{i=1}^{n} \frac{n_i}{n-1}} = \frac{\sqrt{V(n_i)}}{E(n_i)} \ , \tag{30}$$

where $n_i = \sum_{j=1}^{n} n_{ij}$ is the number of ties of actor i, and $E(n_i)$ and $V(n_i)$ are the mean and variance of n_i over actors in the network. CVC, rather than the standard deviation of n_i, is employed because the latter is affected by the density. CVC is closely related to the degree variance defined by Snijders (1981). Conjectures about CVC are not straightforward, but we will include this variable because of its relevance in Yamaguchi's papers.

4. THE SIMULATION

4.1. *Design*

As discussed in Section 2, it seemed infeasible to obtain relations between diffusion times and network measures analytically. First, diffusion times can be expressed in terms of w_{ij}, but for most networks these are complex

expressions even for quite simple networks. Second, we did not define the network measures in terms of w_{ij} but in terms of n_{ij}. Thus, an additional step is needed to express diffusion times as a function of these network measures.[16]

Therefore, we use a simulation method that has also been used by Yamaguchi (1994a, 1994b) and, in a slightly adjusted way, by Buskens (1998) to compare the different diffusion models. We use the set of 218 networks containing all nonisomorphic connected networks with 7 actors and 6, 7, 8, or 9 ties. We computed the expected diffusion times and the network measures discussed in Section 3 for each of these networks. In the next step, we used ordinary statistical methods to "explain" the diffusion times from the network measures. With the linear regression models, we estimate diffusion times as a linear function of the network measures. This linear function will not fit the diffusion times perfectly, but because any "smooth" function can be approximated by a linear function, the coefficients found in the regression are estimates for the partial derivatives of the involved network measures. The explained variance of these models provides an indication for how well the diffusion times can actually be approximated by such a linear function.

There are two main reasons why we chose this particular set of networks. First, the variance of the network measures is large enough. Choosing denser networks—i.e., with more than 9 ties—causes higher correlations between the network measures we want to use in the analyses. With fewer than 6 ties, a network with 7 actors cannot be connected. Second, diffusion times are still computable in a relative short time for networks with 7 actors.[17] The population of networks for which we want to estimate the regression analyses is the population of all networks with 7 actors and 6, 7, 8, or 9 ties. This population consists of considerably more than 218 networks; however, there are only 218 different networks that are nonisomorphic. Some groups of isomorphic networks are larger than other groups. Because isomorphic networks lead to equivalent calculations, we calculate diffusion times for each group of isomorphic networks only once. In the analyses, we weight each network in proportion to the number of

[16]Buskens (1998) expressed the network measures in terms of the w_{ij} and was able to deduce with linearization some first-order results for the relation between a comparably complex dependent variable and density and outdegree.

[17]At the moment we can handle networks of size at most 12 and certainly up to size 10. Diffusion times can be calculated in a few seconds for networks of size 7, but computation time increases rapidly to minutes and hours for larger networks. The Pascal and FORTRAN computer programs used are available from the authors.

isomorphic networks it represents. The weighting is done in such a way that the total number of networks still adds up to 218.[18] We do not claim that we use in this way a representative set of networks as they occur in the real world. How the results depend on the specific choice of the set of networks will be a subject of later investigations.

4.2. *Analyses*

4.2.1. *Global Diffusion Time*

We start by presenting the analyses for global mean contagion time compared with global mean first passage time. For these global diffusion times, we apply ordinary linear regression. There were no strong indications that we could obtain a considerably better fit by transforming the dependent variable by the logarithm transform (Yamaguchi 1994a) or using another transformation of the dependent variable.[19]

Table 3 (Panel A) shows the correlations between the different global diffusion measures. We use superscripts *M* and *PB* to distinguish between the multinomial and product-binomial version of the contagion models. It becomes immediately clear that the contagion times of these two versions are extremely highly correlated. Furthermore, although all measures are highly correlated, the measures of the transit and contagion models are certainly not perfectly correlated.

[18]In fact, the weighting is based on a representation of social networks as *labeled* graphs. If networks would be considered as *unlabeled* graphs, such weighting should not be used. We think that the representation by labeled graphs is preferable because actors are clearly distinguishable in a social network. Because substantive results do not differ whether or not the weights are used in the analyses, this choice is not essential for our results.

[19]More precisely, we did Box-Cox regression (Box and Cox 1964) in Stata 5.0 around the mean and the origin, estimating one parameter for the transformation of the dependent variable. Although the transformation parameter was significant in many analyses, it was not consistent, depending on whether the transformation was done around the origin or around the mean. Moreover, the substantial consequences did not differ from those obtained from the untransformed regression in all analyses. Also, the logarithm transform was not chosen. The largest differences with the untransformed dependent variables occurred for dyadic diffusion times. However, equation (13) shows that the distance between the two actors has an additive effect on the dependent variable. Because the distance is the most important explanatory variable, the logarithm transform is less suitable. Furthermore, the substantial differences between the untransformed and logged dependent variables were marginal at the global and individual levels. Thus, we preferred to keep all dependent variables comparable and did not use any transformation.

TABLE 3
Global Diffusion Variables: Weighted Correlations and Weighted Linear Regression with Huber Standard Errors and Standardized Coefficients ($N = 218$)

A. Correlations		GMCT ^{M}	GMCT ^{PB}	GMFPT
GMCT^{M}		1		
GMCT^{PB}		0.996	1	
GMFPT		0.916	0.911	1
B. Regression results		Contagion		Transit
	Conjecture	GMCT ^{M}	GMCT ^{PB}	GMFPT
Global density	−	−0.51**	−0.48**	−0.35**
Transitivity	+	0.36**	0.41**	0.40**
Number of bridges	+	0.46**	0.43**	0.48**
Diameter	+	0.01	0.03	0.26**
CVC	?	0.36**	0.38**	0.14**
Explained variance		0.95	0.94	0.95

** and * represent significance at respectively $p < .001$ and $p < .01$ (two-sided tests).

Table 3 (Panel B) presents the regression results for the global dependent variables. We present β-coefficients to compare the sizes of similar effects in the different models. The standard errors are Huber standard errors that are consistent under heterogeneity in the residual variables.[20] These can be generalized for clustered observations (Rogers 1993), which we will need to do for the other dependent variables.

The density effect is negative for all dependent variables as was expected in Conjecture 11. As a matter of fact, the effects of all independent variables are in the expected directions. Only the effect of diameter is not significant for the contagion models. The differences or similarities between the transit model and the contagion models are more interesting to look at. As Conjecture 13 stated, the effect of density is indeed larger for the contagion models than for the transit model. In the contagion models, it is the largest effect of all, and that is certainly not the case for the transit model. Furthermore, we expected in Conjecture 15 that the effect of transitivity would be smaller for the contagion models. However, the coeffi-

[20]The Huber estimator of variance was independently discovered by Huber (1967) and White (1980) and is also called the White, sandwich, or robust estimator of variance. Note that the standard errors do not have an interpretation in the classical sense because we analyze a complete population and not a sample. Nevertheless, we provide the standard errors to indicate the "importance" of the effects.

cients are very much alike. What can be noted is that GMCT ^{M}, which was considered to be closest to the transit model because under the multinomial selection of receivers only one actor at a time could be informed, has indeed a smaller coefficient. This coefficient becomes larger again if we change to GMCT ^{PB}. From the analytic results, we know that GMCT ^{PB} is larger than GMCT ^{M} for the three-actor network. Because transitivity measures the number of "closed" triads compared with "open" triads, this is an indication that the difference between the contagion times for the multinomial and the product-binomial versions depends on the number of closed triads in the network.

The number of bridges has a larger effect on GMFPT than on GMCT, although the difference is marginal. Thus, we do not obtain strong confirmation of Conjecture 17, which stated that bridges would hinder information diffusion more severely in the transit model. On the other hand, similar reasoning holds for diameter, which led to Conjecture 19. In the transit model, information is much more able to wander around especially if the diameter of the network is large. In the contagion model, diameter does not affect information flow, while it has a strong effect on GMFPT. In addition, number of bridges and diameter are the two dependent variables with the highest correlation (.66). If we drop diameter from the analyses, the results for GMCT hardly change, but for GMFPT the coefficient of the number of bridges increases sharply. All these observations confirm that GMFPT increases much faster than GMCT with number of bridges and/or size of diameter.

The results for global diffusion times show that substantive predictions do not change between the transit and contagion models—i.e., all predicted directions of effects are the same. The only difference is that the contagion model does not predict an effect of diameter in addition to an effect of number of bridges, while the transit model predicts that diameter has a positive effect on global diffusion time above and beyond the positive effect of number of bridges.

4.2.2. *Dyadic Diffusion Time*

In the previous section, we already found differences in the magnitude of effects of the network measures between the transit model and the contagion model at the global level. However, we will now see that the differences become larger at the more detailed dyadic level.

Table 4 (Panel A) shows the correlations between the dyadic diffusion measures. Again, the correlation between the two different versions of the contagion model is nearly one. The correlations between the contagion

TABLE 4

Dyadic Diffusion Variables: Weighted Correlations and Weighted Linear Regression with Standardized Coefficients and Huber Standard Errors Modified for Observations Clustered Within the Networks ($N = 9156$, number of clusters is 218)

A. Correlations		MCT_{ij}^{M}	MCT_{ij}^{PB}	$MFPT_{ij}$
MCT_{ij}^{M}		1		
MCT_{ij}^{PB}		0.999	1	
$MFPT_{ij}$		0.884	0.875	1
B. Regression results		Contagion		Transit
	Conjecture	MCT_{ij}^{M}	MCT_{ij}^{PB}	$MFPT_{ij}$
Global network measures				
Global density	−	0.03*	0.03*	0.08**
Transitivity	+	0.06**	0.06**	0.12**
Number of bridges	+	−0.03*	−0.04**	0.10**
CVC	?	0.10**	0.09**	0.10**
Individual network measures				
Degree transmitter	−	0.19**	0.20**	0.19**
Degree receiver	−	−0.26**	−0.24**	−0.48**
Local density transmitter	+	0.02**	0.03**	−0.02**
Local density receiver	−	−0.07**	−0.06**	−0.06**
Degree quality transmitter	−	0.05**	0.05**	0.12**
Degree quality receiver	−	0.02**	0.03**	−0.23**
Bridges transmitter	−	0.03**	0.04**	0.05**
Bridges receiver	−	0.03**	0.03**	−0.05**
Dyadic network measure				
Distance	+	0.88**	0.90**	0.55**
Explained variance		0.93	0.93	0.90

** and * represent significance at respectively $p < .001$ and $p < .01$ (two-sided tests).

models and the transit model are a little bit lower than for the global diffusion variables.

Table 4 (Panel B) presents the regression results for the dyadic dependent variables. The dyadic dependent variables have the problem that the cases are nested within networks. For each network we have forty-two cases. Therefore, a straightforward generalization of the Huber standard errors is used that takes explicitly into account the clustering of observations and the expected correlations between residuals (Rogers 1993).

Arguments to explain the effects on dyadic diffusion time have to start with distance. In the contagion model, distance is by far the most important explanatory variable, and in the transit model it is, with the

degree of the receiver, one of the most important variables. The interpretations of all the other variables are based on whether they cause information to make detours away from the shortest path or whether they help information reach the receiver. In comparing the models, the reasoning will be analogous to that outlined earlier. Possibilities for detours increase diffusion time to a larger extent in the transit model than in the contagion model. Possibilities for trapping information in the neighborhood of the receiver decrease diffusion time to a larger extent in the transit model than in the contagion model.

Distance has the effect expected in Conjecture 1 and is indeed more important in the contagion model than in the transit model, as was stated in Conjecture 2. Global density clearly does not have the effect expected in Conjecture 11. High global density causes information to go in many directions, and most of them turn out to be wrong. This implies that the argument for Conjecture 12 about the decreasing effect of density for dependent variables at lower aggregation levels is even stronger than expected. The effect of information drifting away to unfavorable parts of the network is larger than the opportunity provided by information paths; and, therefore, the effect turns in the other direction. In correspondence with Conjecture 13, this effect is even stronger for the transit model. In the contagion models, the effect of density is in the wrong direction but hardly significant. Related to this effect is also the definition of $\boldsymbol{W}$, which implies that increasing density does not increase the amount of information going through the network but only the number of possible ways to transfer information.

Transitivity has the effect of trapping information away from the receiver, and this effect is stronger in the transit model (Conjectures 14 and 15). In the transit model, number of bridges has the effect expected in Conjecture 16, which reasons that bridges hinder information flow. However, we find a different prediction for the contagion model—namely, that number of bridges decreases diffusion time. This shows clearly that information diffusion turns out to be hindered less by bridges in the new model, as stated in Conjecture 17. The fact that bridges force information to take the shortest path is even more important in the contagion model resulting in a negative effect of number of bridges on contagion time. CVC has similar effects in all models.

The effect of degree is in the direction expected in Conjecture 3 for the receiver. Larger receiver degree reduces diffusion time. However, larger transmitter degree increases diffusion time. Comparable with the argument explaining why a higher density could have a positive effect on dif-

fusion time, transmitter degree has a positive effect on diffusion time because the transmitter has more possible ways to transmit the information in the *wrong* direction. The origin of this effect also lies in the definition of $\boldsymbol{W}$. We will be more specific about that problem in the next section. We did not formulate conjectures about differences between the effect of degree for the transit model and the contagion model. Still, degree is more important in the transit model than in the contagion model. This can be seen most clearly if the degree qualities of the transmitter and receiver are taken into account. Degree quality indicates the "second-order" degree—namely, the number of contacts that can be reached in two steps. High transmitter degree quality increases diffusion times. Again, in this situation information has a higher probability of taking routes other than the direct route to the receiver, and this effect is stronger in the transit model. Moreover, receiver degree and degree quality help considerably to accelerate information diffusion toward the receiver in the transit model, but in the contagion model, receiver degree quality increases contagion time slightly.

Concerning individual local density, we run into collinearity problems that affect the analyses. Correlations between independent variables are often low, but in some cases they are as high as 0.6—for example, between number of bridges and global density. Fortunately, in most cases this does not seem to be a problem. Nevertheless, it is a problem for degree quality and individual local density. The correlation is about 0.52 and the two measures seem to explain about the same part of the variation in the dependent variable. If we include only one of the two pairs of variables, the explained variance decreases only marginally. In that case, Conjecture 7 is confirmed for receiver degree quality but rejected for transmitter degree quality. The trapping effects of individual local density as stated in Conjectures 4 and 5 are confirmed for the contagion model. Only if we add the coefficients of degree quality and individual local density is some confirmation found for the assertion that trapping of information is more important in the transit model than in the contagion model (Conjecture 6). This is indicated especially by the fact that receiver degree quality has a large negative effect on the dyadic diffusion time for the transit model.

The effect of number of bridges at which an actor is located mostly rejects Conjecture 8. The conjecture is only confirmed for receiver bridges in the transit model. All effects are relatively small, and we are still puzzled about what explains these effects. Nevertheless, the effects of receiver bridges moves in different directions for the transit model and the contagion model. Thus we do find essential differences between the transit and contagion models in this section. Not only is the effect of distance

between two actors much more important in the contagion model, but also opposite effects were found most prominently for number of bridges in the network and for degree quality of the receiver.

4.2.3. *Individual Diffusion Measures*

All analyses in this paper were done for both the multinomial and the product-binomial selection of receivers. The correlations between the corresponding diffusion times were always higher than 0.995, and substantive implications did not change. A plausible reason for this is that at every time point, the "marginal" probability of information being provided to actors is the same in both versions of the model: only probabilities for simultaneously informing others differ. We have already shown in Section 2 that the assumptions led to exactly the same results for trees. In the last two subsections, we presented results for both versions for dyadic and global diffusion time to show how small the differences are. From now on we use only the product-binomial version because this version places less stringent restrictions on $\boldsymbol{W}$.

In considering the efficiency of information transfer in a network from the actor's point of view, there are actually three things that matter. First, if an actor obtains information, how quickly can he transmit that information throughout the network? Second, if information enters the network at some random place, how long will it take before the information reaches the focal actor? Third, if somebody wants to transmit information to a specific other actor in the network, to what extent can a third actor influence the time it takes to transfer information between these two actors? For all three concepts, we developed diffusion and/or centrality measures. In this subsection, diffusion times for transmitting and receiving and the centrality measure for betweenness are analyzed. We did not analyze closeness centrality measures related to the diffusion times for transmitting and receiving because they are the inverses of the diffusion times.

Table 5 (Panel A) presents the correlations between the dependent variables of this section. Note that we expect negative correlation between diffusion times and centrality measures. In Table 5 (Panel A), we are immediately struck by the correlations in the second column. All diffusion measures should give an indication of the lack of centrality of a certain actor. However, individual mean first passage time correlates negatively with the other diffusion times, while it correlates positively with the betweenness centrality measures. The implications of the model are not convincing for mean first passage time: for example, in a chain of seven actors the most peripheral actors (those at the ends of the chain) reach the other

TABLE 5

Individual Diffusion Variables: Weighted Correlations and Weighted Linear Regression with Standardized Coefficients and Huber Standard Errors Modified for Observations Clustered within the Networks ($N = 1526$, number of clusters is 218)

A. Correlations		MCT_i^{PB}	$MFPT_i$	MRT_i^{PB}	$MFRT_i$	CBC_i	MEC_i
MCT_i^{PB}		1					
$MFPT_i$		0.52	1				
MRT_i^{PB}		0.63	−0.10	1			
$MFRT_i$		0.60	−0.17	0.95	1		
CBC_i		−0.21	0.38	−0.63	−0.54	1	
MEC_i		−0.44	0.34	−0.93	−0.90	0.81	1
B. Regression results		Transmitting		Receiving		Betweenness	
		Contagion	Transit	Contagion	Transit	Contagion	Transit
	Conj.[a]	MCT_i^{PB}	$MFPT_i$	MRT_i^{PB}	$MFRT_i$	CBC_i	MEC_i
Global network measures							
Global density	−	−0.23**	−0.43**	0.12**	0.19**	−0.27**	−0.29**
Transitivity	+	0.18**	0.32**	0.14**	0.12**	0.13**	0.04**
Number of bridges	+	0.51**	0.45**	0.21**	0.21**	−0.19**	−0.11**
Diameter	+	0.06*	0.24**	0.01	0.06**	0.04**	−0.01*
CVC	?	0.45**	0.04	0.19**	0.18**	0.10**	−0.08**
Individual network measures							
Degree	−	−0.27**	0.31**	−0.88**	−0.78**	0.73**	0.94**
Individual local density	+/−	0.34**	0.07**	−0.01	−0.03	−0.11**	−0.10**
Degree quality	−	−0.38**	0.23**	−0.18**	−0.37**	−0.16**	0.21**
Individual bridges	−	−0.31**	−0.05**	−0.12**	−0.14**	0.52**	0.19**
Explained variance		0.88	0.91	0.94	0.89	0.89	0.97

** and * represent significance at respectively $p < .001$ and $p < .01$ (two-sided tests).

[a]The conjectures hold only for transmitting and receiving information. If there are two signs, the first holds for transmitting information and the second for receiving. For betweenness, the only conjecture we had is that the number of bridges at which an actor is located increases betweenness centrality.

actors in the shortest time and should be considered central. These kinds of results do not follow from the contagion model. There are two factors responsible for these peculiar results. First, the transit model implies that if an actor in the center of the chain starts the diffusion process in the wrong direction, he has to wait until the information comes back to him before he can transmit to the other half of the network. The actors at the end of the chain will always transmit their information in the right direction. Second, the definition of $\boldsymbol{W}$ implies that everybody communicates with others to the same extent. The definition is built on the assumption that every actor has the same amount of resources to transmit information and divides these resources equally among his contacts. Thus, an actor at the end of the chain will communicate at every time period to his neighbor and as soon as he obtains information the neighbor will know it at the next time period. In this way, actors are restricted by their own contacts, but they are hardly restricted by the resources of their neighbors; in other words, neighbors will always "listen." On the other hand, if we examine the time it takes before an actor obtains information, he is restricted by his own contacts as well as by the time other actors invest in communicating with him. Therefore, for the diffusion time of an actor it seems better to define $\boldsymbol{W}$ in such a way that actors divide their resources among the actors who are communicating with them. Formally, this would imply that we transpose $\boldsymbol{W}$ and interpret w_{ij} as "actor i receives information from actor j." The resulting diffusion times are exactly MRT_i and MFRT_i. Furthermore, as was mentioned before, $\mathrm{MFRT}_i = 1/\mathrm{IEC}_i$, for which the results are still reasonable.

Table 5 (Panel B) shows that most of the conjectures about MCT_i are supported. Density, degree, degree quality, and the number of bridges at which the transmitter is located promote information diffusion. Local density, bridges, diameter, and centralization (CVC) inhibit information diffusion. Note that this does not imply that centralization of the network is disadvantageous for information diffusion in general. It is only disadvantageous on average if each actor is equally likely to be the starting point of the diffusion process. However, due to the degree effect, a centralized network will diffuse information fast if the central actor always receives information first.

MFPT_i is clearly a problematic measure. The coefficients for degree and degree quality imply that peripheral actors often turn out to be the most central actors. This is a good example of how a number of plausible assumptions can combine to lead to an implausible conclusion. Further-

more, it is the clearest example that the contagion and transit models can have different implications in certain situations.

In contrast with the measures for transmitting information, it is also remarkable that the results for receiving time for the transit and contagion models are so similar. To receive information soon, degree and degree quality are the important indicators, which confirms Conjectures 3 and 7. Density is again a disturbing factor. Large density increases information diffusion time in both models, refuting Conjecture 11. Again, the background of this effect lies in the definition of $\boldsymbol{W}$, which implies that larger density only influences the number of possible ways to transfer information, not the amount of information in the network. Trapping information in the area around the focal actor is, as stated by Conjecture 6, more important in the transit model. The similarity between the transit and contagion models in this case indicates that the immediate effects centrality introduced by Friedkin (1991) is less sensitive with respect to the information transfer assumption in this situation.

We did not make many conjectures about the betweenness measures. The only conjecture about betweenness centrality we formulated is confirmed: actors located at more bridges have higher betweenness centrality. It is not in accordance with our intuition that this effect is larger in the contagion model than in the transit model. Nevertheless, we observe some other interesting results. In dense networks, actors will find other routes relatively easily if one actor stops diffusing information. As a result betweenness centrality is smaller, on average, in dense networks. In a network with areas that are locally dense, there will be actors with high betweenness centrality between these areas, while actors that are in the locally dense areas will have lower betweenness centrality. An actor who has more ties will also be more influential in the diffusion of information through the network. Moreover, contagion betweenness centrality is much more strongly related to number of bridges at which an actor is located than is mediative effects centrality. Two effects point in opposite directions for the two models. Namely, in the transit model degree quality has a positive effect on betweenness centrality, while in the contagion model it has a negative effect. And CVC has a positive effect on betweenness in the contagion model, while it has a negative effect in the transit model.

Summarizing, we encountered a number of essential differences related to the assumptions made in the models under research. At the highest aggregation level, the models do not differ that much. However, at the individual and dyadic levels, we find that the implications of the transit

model differ from those of the contagion model. First, we obtained predictions of effects of variables that point in opposite directions for some variables—for example, for the effect of number of bridges on dyadic diffusion time and for the effects of degree and degree quality on individual diffusion time for transmitting information. For betweenness centrality, two effects of network measures were found that went in different directions. Second, effects of some variables were found to be consistently smaller for one model than for the other. In the transit model the effect of bridges and/or diameter is much larger than in the contagion model. Also, the effect of trapping information in certain parts of the network is larger in the transit model. These effects are due to the unrealistic information transfer assumption in the transit model; and, therefore, it can be stated that the transit model actually overestimates these effects. Third, we discovered that the implementation of $\boldsymbol{W}$ can have large effects on centrality measures. For example, the positive effects of density on dyadic and individual diffusion times are related to the assumption that all actors invest the same amount in communicating with others. We found especially undesirable results for the combination of the transit model and the $\boldsymbol{W}$ chosen for individual mean first passage time. The results turned out to be less problematic for individual mean contagion time using the improved information transfer assumption. Thus we found differences between different centrality measures not only because they have different substantive meanings but also because of assumptions we do not want to have such effects—among others, the assumption that information is transmitted between actors as a kind of package.

5. USING THE MODELS IN EMPIRICAL ANALYSES

Discussions about the choice and use of $\boldsymbol{W}$ in the model would have shifted the focus of the paper too much away from the key assumption we wanted to discuss—namely, the information transfer assumption. However, the influence of the choice of $\boldsymbol{W}$ seems to be essential in the empirical use of the model. Therefore, we summarize some points of that discussion without new analyses in this section.

Some cautionary remarks about using the model discussed in this paper in empirical analyses are necessary. Network data often are dichotomous, and, therefore, we made the model applicable to cases in which only dichotomous data are available. We "estimated" contact probabilities in $\boldsymbol{W}$ from the dichotomous data based on theoretical considerations (see

Yamaguchi 1994a). In Section 4, we argued that the way we obtained these contact probabilities would probably be less suitable if MCT_i or $MFPT_i$ were the dependent variable. The mere fact that we have to pay attention to this discussion, because the definition of $\boldsymbol{W}$ can have large effects on the implications of the model, shows that one has to be careful using the model when only dichotomous network data are available. Moreover, we highlighted in Subsection 4.2.3 only one part of the problem—namely, that the theoretical reasoning behind the choice of contact probabilities does not correspond directly to the dependent variable involved. The other side of the problem is that we use only the dichotomous data to define the network measures in the analyses. One can argue that we are analyzing a valued network and should deduce network measures that are related to the values of the ties and not only to whether a tie does or does not exist. For example, one can ask who has a larger degree: an actor with many friends whom he sees rarely or an actor with fewer friends whom he sees frequently. According to the former definition, the actor with many friends has a larger degree, but it is certainly arguable that degree depends not only on the number of contacts but also on the frequency.

It is relatively easy to change the definitions of most network measures to make them applicable to valued networks. In fact, almost all definitions were formulated in a way that would make this change straightforward. All the network measures that were formulated in terms of n_{ij} can be reformulated by changing n_{ij} to w_{ij}. One point that needs some more attention is that in some of the formulations of the network measures we used the fact that $n_{ii} = 0$ so we could sum over all i and j without having to exclude the cases where $i = j$. If we want to choose w_{ii} to be different from zero, we need to be more explicit about how these diagonal elements have to be treated. Another point that needs attention is that when $\boldsymbol{W}$ is used the networks are not necessarily symmetric. For instance, indegree and outdegree need not have the same value and should be calculated separately, but these generalizations are straightforward. The only measures that need some adjustment are bridges, distance, and diameter. For bridges one can define the "bridgeness" of a valued graph as

$$\text{Bridgeness} = \sum_{\text{bridges}} \frac{1}{w_{ij}} , \tag{31}$$

which implies that a bridge that is very "small" adds more to the bridgeness than a bridge between two actors with a strong tie. Note that this

definition is in fact a generalization of the number of bridges in the dichotomous network; namely, changing w_{ij} to n_{ij} results in a value for bridgeness that is equal to the number of bridges. The number of bridges at which an actor is located can be generalized in a similar way by summing over the bridges connected to that actor. The new distance measure we propose is inspired by the new diffusion measure:

$$\text{Distance}_{ij} = \min_{R} \sum_{(k,l)\in R} \frac{1}{w_{kl}} \ , \tag{32}$$

where R contains all paths that go from actor i to actor j and (k,l) are all dyads on these paths. This definition implies that for all trees, $\text{MCT}_{ij} = \text{distance}_{ij}$ (see Section 2). The diameter was defined in terms of distances in the network, and this definition can still be used.

Further research is necessary to investigate how the results of this paper change if the network measures are based on valued networks. Nevertheless, using the model based on contact frequencies that are measured as such in empirical research will lead to more convincing results due to the model's sensitivity to an indirect theoretical estimation of $\boldsymbol{W}$ based on dichotomous network data.

6. CONCLUSIONS AND DISCUSSION

This paper introduced a contagion model for information diffusion in heterogeneous networks and compared that model with the transit model used by Yamaguchi (1994a). The main difference between the two models is that, information is given from one actor to another as a kind of package in the transit model, while in the contagion model every actor who ever obtained information will have that information at any time in the future. With analyses of networks with seven actors, we compared the implications of the two models for substantive questions, such as efficiency of information diffusion in networks and centrality of actors in networks.

The analyses show that the implications of the transit model and the contagion model are partially different. Bridges hindered global information diffusion more in the transit model than in the contagion model. Moreover, trapping of information in certain parts of the network was more severe in the transit model than in the contagion model. We assert that these difference are due to the unrealistic information transfer assumption in the transit model and, consequently, that the transit model overestimates

these effects. Individual mean first passage time based on the transit model turned out to have undesirable implications when taken in combination with the contact probabilities used. Individual mean contagion time based on the contagion model seemed to be less sensitive to the choice of the contact probabilities; at least the results for individual mean contagion time made much more sense.

For individual mean first passage time, increases in degree and degree quality had large positive effects on the time needed to transmit information through the network, while these effects were negative and, consequently, much more plausible for individual mean contagion time. Furthermore, concerning centrality measures, this paper shows again that the determination of who is central in a network depends on whether centrality is about transmitting, receiving, or betweenness. Somebody who can transmit information quickly in a network is not necessarily a good receiver of information in the network. Still, we assert that contagion receiver centrality and contagion betweenness centrality measure similar concepts as Friedkin (1991) measured, respectively, with immediate effects centrality and mediative effects centrality. Both pairs of measures are defined for valued networks $\boldsymbol{W}$. However, comparing the measures using the same $\boldsymbol{W}$ showed that there were considerable differences. As said before, for receiving information the effects of the different network measures varied only in the size of effects; therefore, the new model does not provide different hypotheses about the effects of certain network measures on centrality. This result makes it questionable whether it is necessary to use the more complex model to estimate closeness centrality. On the other hand, betweenness centrality predictions of two network measures went in opposite directions, which increases the value of using our more complex model for this centrality measure.

Besides the differences found in the regression analyses, we studied the ordering of centrality inside the networks. For connected networks with fewer than six actors there were hardly any differences in the ordering,[21] but for networks of size 7, changes in the ordering were omnipresent, especially as can be expected from the foregoing results, for betweenness centrality. We expect that these differences will increase with the network size and, therefore, investing in the increasing calculation effort of the new model for larger networks is reasonable. Of course, only

[21]This set of networks consists of only 30 networks and could easily be checked by hand.

empirical tests can provide evidence about whether the models produce different predictions in empirical situations and which one is better in a particular situation.

This brings us directly to a discussion about the use of the new model for larger networks. For larger networks, it is essential that only reachable states are included in describing the diffusion process. Also, network equivalence concepts can be used to reduce complexity. We do not exclude the possibility that there are far more efficient methods to calculate the new diffusion times, because there seems to be a lot of structure in the Markov chain that we could not use directly to reduce computation time. For example, in the case of trees, the contagion times turned out to be simple, but this was a result of the discovery of a straightforward way to calculate these contagion times. If we had constructed the Markov chain and calculated contagion times that way, these relations would be hard to reproduce. Besides trying to find faster methods to calculate contagion times exactly, one can develop approximation methods. A first example is what we did in this paper: finding relations between simple network measures and contagion times. For the networks with seven actors, we were able to explain about 90 percent of the variance in diffusion times with relatively simple network measures. One could use the regression equations to approximate contagion times in other networks. The problem is that the accuracy of these equations for larger networks is highly uncertain. Another way of approximating contagion times is to summarize a large network structure in a simpler stochastic block design. Then expected contagion times can be calculated within and between the blocks. Thereafter, these results can be brought together to estimate contagion times between actors in different blocks.

REFERENCES

Altmann, Michael. 1993. "Reinterpreting Network Measures for Models of Disease Transmission." *Social Networks* 15:1–17.

Bailey, Norman T. J. 1957. *The Mathematical Theory of Epidemics*. London: Charles Griffin.

———. 1964. *The Elements of Stochastic Processes with Applications to the Natural Sciences*. New York: Wiley.

———. 1975. *The Mathematical Theory of Infectious Diseases and Its Applications*, 2nd ed. London: Charles Griffin.

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

Box, G. E. P., and D. R. Cox. 1964. "An Analysis of Transformations." *Journal of the Royal Statistical Society*, ser. B, 26:211–43.

Burt, Ronald S. 1976. "Positions in Networks." *Social Forces* 55:93–122.

———. 1991. *Structural Holes*. Cambridge, MA: Harvard University Press.

Buskens, Vincent. 1998. "The Social Structure of Trust." *Social Networks* 20:265–89.

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

Ellner, Stephen P., Akira Sasaki, Yoshihiro Haraguchi, and Hirotsugu Matsuda. 1998. "Speed of Invasion in Lattice Population Models: Pair-Edge Approximation." *Journal of Mathematical Biology* 36:469–84.

Freeman, Linton C. 1979. "Centrality in Social Networks: Conceptual Clarification." *Social Networks* 1:215–39.

Friedkin, Noah E. 1991. "Theoretical Foundations for Centrality Measures." *American Journal of Sociology* 96:1478–504.

Greve, Henrich R., David Strang, and Nancy B. Tuma. 1995. "Specification and Estimation of Heterogeneous Diffusion Models." Pp. 377–420 in *Sociological Methodology 1995*, edited by Peter V. Marsden. Washington, DC: American Sociological Association.

Harris, T. E. 1974. "Contact Interactions in a Lattice." *Annals of Probability* 2:969–88.

Hedström, Peter. 1994. "Contagious Collectivities: On the Spatial Diffusion of Swedish Trade Unions, 1890–1940." *American Journal of Sociology* 99:1157–79.

Huber, Peter J. 1967. "The Behavior of Maximum Likelihood Estimates under Non-Standard Conditions." *Proceedings of the Fifth Berkeley Symposium in Mathematical Statistics and Probability* 1:221–33.

Kemeny, John G., and J. Laurie Snell. 1960. *Finite Markov Chains*. New York: D. Van Nostrand.

Kretzschmar, Mirjam, and Martina Morris. 1996. "Measures of Concurrency in Networks and the Spread of Infectious Disease." *Mathematical Biosciences* 133:165–95.

Laumann, Edward O., and David Knoke. 1987. *The Organizational State: Social Choice in National Policy Domains*. Madison: University of Wisconsin Press.

Mahajan, Vijay, and Robert A. Peterson. 1985. *Models for Innovation Diffusion*. Beverly Hills, CA: Sage.

Mollison, Denis M. 1977. "Spatial Contact Models for Ecological and Epidemic Spread." *Journal of the Royal Statistical Society*, ser. B, 39:283–326.

Morris, Martina. 1993. "Epidemiology and Social Networks: Modeling Structured Diffusion." *Sociological Methods and Research* 22:99–126.

Nowak, Martin A., and Robert M. May. 1992. "Evolutionary Games and Spatial Chaos." *Nature* 359:826–29.

Rogers, W. H. 1993. "Regression Standard Errors in Clustered Samples." *Stata Technical Bulletin* 13:19–23.

Snijders, Tom A. B. 1981. "The Degree of Variance: An Index of Graph Heterogeneity." *Social Networks* 3:163–74.

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

Strang, David, and Nancy B. Tuma. 1994. "Spatial and Temporal Heterogeneity in Diffusion." *American Journal of Sociology* 99:614–39.

Valente, Thomas W. 1995. *Network Models of the Diffusion of Innovations*. Cresskill, NJ: Hampton Press.

Wasserman, Stanley, and Katherine Faust. 1994. *Social Network Analysis: Methods and Applications*. Cambridge, England: Cambridge University Press.

White, Halbert. 1980. "A Heteroscedasticity-Consistent Covariance Matrix Estimator and a Direct Test for Heteroscedasticity." *Econometrica* 48:817–30.

Yamaguchi, Kazuo. 1994a. "The Flow of Information through Social Networks: Diagonal-Free Measures of Inefficiency and the Structural Determinants of Inefficiency." *Social Networks* 16:57–86.

———. 1994b. "Some Accelerated Failure-Time Regression Models Derived from Diffusion Process Models: An Application to a Network Diffusion Analysis." Pp. 267–300 in *Sociological Methodology 1994*, edited by Peter V. Marsden. Washington, DC: American Sociological Association.