Opinion Leadership and Social Contagion in New Product Diffusion

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We study how opinion leadership and social contagion within social networks affect the adoption of a new product. In contrast to earlier studies, we find evidence of contagion operating over network ties, even after controlling for marketing effort and arbitrary systemwide changes. More importantly, we also find that the amount of contagion is moderated by both the recipients' perception of their opinion leadership and the sources' volume of product usage. The other key finding is that sociometric and self-reported measures of leadership are weakly correlated and associated with different kinds of adoption-related behaviors, which suggests that they probably capture different constructs. We discuss the implications of these novel findings for diffusion theory and research and for marketing practice.

Key words: diffusion of innovations; opinion leadership; social contagion; social networks
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1. Introduction

Marketers are increasingly experimenting with various forms of network marketing. In the area of new product marketing, the rationale for such strategies rests on three key assumptions: (1) social contagion among customers is at work, (2) some customers' adoptions and opinions have a disproportionate influence on others' adoptions, and (3) firms are able to identify and target those influentials or opinion leaders. These assumptions are quite reasonable, as the first two are consistent with several sociological and marketing theories, and all three have been supported in at least some studies (e.g., Godes and Mayzlin 2009, Goldenberg et al. 2006, Rogers 2003, Tucker 2008, Valente et al. 2003, Weimann 1994).

However, managers would be remiss to simply take those three assumptions for granted. For instance, Van den Bulte and Lilien (1997, 2001) have shown that contagion need not be as important as reported in prior studies, Becker (1970) and Watts and Dodds (2007) have raised doubts on the importance of opinion leaders in speeding up the acceptance of new products, and Rogers and Cartano (1962) note disagreement on whether to identify opinion leaders based on their self-reports or their centrality in social networks. More recent research by Coulter et al. (2002) and Godes and Mayzlin (2009) provides conflicting answers to the question of whether heavy

users are more influential than light users, an issue of obvious relevance to the identification and targeting of likely influentials.

The present study addresses each of the three assumptions fundamental to many network marketing practices. Specifically, we empirically assess three questions on how social contagion and opinion leadership affect new product diffusion. First, to what extent do sociometric and self-reported opinion leadership go hand in hand and have the same influence on the time of adoption? Second, is there social contagion operating over social ties such that better connected adopters exert more influence than less connected ones, over and above the effect of marketing efforts and systemwide influences that vary over time? Third, is contagion emanating from prior adopters a function of how much they use the product rather than simply whether they have adopted it?

We investigate these questions by studying the adoption of a new prescription drug by physicians. Our study combines individual-level adoption data, demographic data, a measure of self-reported opinion leadership, network data on discussion and patient referral ties among physicians, and individual-level sales call data. Hence we are able to investigate the presence of contagion dynamics over social networks in a real market in which traditional marketing efforts are also being deployed, the kind of setting

that is of greatest relevance to both practitioners and researchers (Van den Bulte and Lilien 2001, Watts and Peretti 2007).

The results are of both theoretical and managerial interest. Not only do we document the existence of contagion in new product adoption after controlling for many potential confounds, including marketing effort, but we also show that the amount of contagion is moderated by both the recipients' perception of their opinion leadership and the sources' volume of product usage. The second key finding is that sociometric and self-reported measures of leadership are weakly correlated and associated with different effects, indicating that they capture different constructs. In other words, we document important contingencies in the social contagion process as well as important differences between the two main operationalizations of opinion leadership. Although earlier studies have assessed the existence of contagion in new product adoption after controlling for marketing effort using actual network data (Hill et al. 2006, Van den Bulte and Lilien 2001) or trying to proxy for network ties by geographical propinquity (Bell and Song 2007, Grinblatt et al. 2008, Manchanda et al. 2008) or by group membership (e.g., Duflo and Saez 2003, Sacerdote 2001), or they have assessed the existence of social influence in the usage intensity or joint consumption of established products and services within dyads (e.g., Hartmann 2009, Nair et al. 2006), none of those studies has documented how sociometric leadership, self-perceived leadership, and volume of product usage interrelate in affecting contagion effects in new product diffusion.

The findings reported here are of interest to researchers seeking to better understand the relations between opinion leadership, sensitivity to contagion, and time of adoption, a set of issues that recent research has shown to be more complex than was previously thought (Van den Bulte and Joshi 2007, Watts and Dodds 2007). The findings are also of interest to practitioners seeking to identify effective opinion leaders. That contagion is at work, and sociometric leadership is more strongly associated with early adoption than self-reported leadership, for instance, implies that the former metric is more effective in identifying early seeding points to jump-start the diffusion process. Another key implication is that the practice of targeting heavy users, common in the pharmaceutical industry and elsewhere, is justifiable based not only on their higher "stand-alone" customer lifetime value but also on their higher "network value" because they exert more social contagion. However, because the correlation between prescription volume and sociometric leadership is only moderate, just focusing on heavy users will fail to leverage all potential influential seeding points.

We proceed as follows. We first develop the three research questions. We then describe our research setting and research design. Next, we specify the variables created for analysis and present the results. We conclude with a discussion of implications for theory and research and for marketing practice.

2. Research Questions

2.1. Social Contagion
The most fundamental assumption of network marketing is that social influence or social contagion among customers is at work. Although this assumption is often taken for granted, it need not always be warranted. For instance, several studies have documented inflated evidence of contagion as a result of estimation problems and the use of theoretically overdetermined models. Unless one analyzes individual-level adoption data and network data, contagion can easily be confounded with other mechanisms generating temporal changes in adoption speed (Van den Bulte and Stremersch 2004). Another source of upward bias in prior evidence of contagion has been the failure to appropriately control for marketing effort and other changes in the market environment, as shown most compellingly in reanalyses of the classic *Medical Innovation* study (Marsden and Podolny 1990, Van den Bulte and Lilien 2001).

The second assumption underlying network marketing strategies, that some customers' adoptions and opinions have a disproportionate influence on others' adoptions, should not be taken for granted either. It is likely to hold when some customers have a much more central position in the social network than do others or when potential adopters look for advice from experts (e.g., Goldenberg et al. 2006). In contrast, when the social network structure is not very centralized and when what spreads is simply information about the product's existence rather than information that mitigates perceived risk—conditions typically associated with so-called buzz marketing campaigns—then there is not much variation among customers' relative influence (e.g., Van den Bulte and Wuyts 2007, Watts and Peretti 2007).

Thus, we assess whether new product adoption is subject to social contagion operating through network ties such that better connected adopters exert more influence than do less connected ones and whether such contagion operates over and above the effect of targeted marketing efforts and systemwide influences that vary over time. To our knowledge, the network contagion effect has never survived such a stringent test because prior studies did not use social network data (e.g., Bell and Song 2007), did not account for degrees of connectivity (e.g., Hill et al. 2006), did not control for marketing effort or time-varying shocks

(e.g., Coleman et al. 1966, Strang and Tuma 1993), or did control for marketing effort or time-varying shocks but found no evidence of contagion (Marsden and Podolny 1990, Van den Bulte and Lilien 2001).

2.2. Sociometric vs. Self-Reported Opinion Leadership

The third key assumption underlying many network marketing strategies is that firms are able to identify and target influentials or opinion leaders. Rogers and Cartano (1962) discuss three ways to identify such people: (1) self-designation, i.e., asking survey respondents to report to what extent they perceive themselves to be influential; (2) sociometric techniques, i.e., computing network centrality scores after asking survey respondents to whom they turn for information or advice, or after observing interactions through other means (e.g., citations among scientists); and (3) the key informant technique, where selected people are asked to report their opinion about who the influentials are. Whereas self-designation is the most popular technique among marketing academics, the sociometric technique has been more popular among social network analysts. The latter technique is also gaining popularity among marketing practitioners to identify influential scientists, physicians, and engineers (e.g., Dorfman and Maynor 2006) and among some consumer network marketing firms, such as Procter & Gamble's Vocalpoint, that target people with demographic characteristics associated with having a central network position.

Doubts exist about the value of both self-reports and sociometric measures. It is likely that self-reported opinion leadership is biased upward and that it also reflects self-confidence rather than actual influence. Conversely, doubts about marketers' ability to effectively identify influentials using sociometric methods have arisen recently following a simulation study by Watts and Dodds (2007) showing that the customers critical in generating a sudden burst in the speed of diffusion need not necessarily be the best connected. Although this possibility was already long known to network and diffusion researchers (e.g., Becker 1970, Locock et al. 2001), the recent simulation results have created a heated debate among marketing practitioners (Thompson 2008). Much of that debate seems to ignore that the study by Watts and Dodds (2007) was only a simulation demonstrating a possibility, not an empirical study providing actual evidence in support of that possibility. Still, the simulation results bring to the fore potential difficulties marketers may face in identifying key influentials using sociometric methods.

To gain a deeper understanding of the issues at hand, we test several hypotheses. Because little is known about whether different methods actually identify the same influentials (convergent validity), as indicated by a recent review of the literature (Valente and Pumpuang 2007), we first assess to what extent their leadership scores are correlated. Apart from three studies (Jacoby 1974, Kratzer and Lettl 2009, Rogers and Svenning 1969), none of which used the validated (Childers 1986) scale of self-reported leadership standard in marketing nowadays, we are not aware of any evidence on this fundamental issue.

Even less is known about whether the two leadership constructs have the same association with adoption behavior (nomological validity). People who are often nominated by their peers as someone they turn to for expertise or discussion are likely to be true sources of influence. People who perceive themselves to be influential, in contrast, may simply have an inflated sense of self-importance. To the extent that true expertise drives early adoption, sociometric leadership may be more strongly associated with early adoption than self-reported leadership is. On the other hand, early adoption may be affected more by how one perceives oneself than by one's true status. These arguments suggest that sociometric leaders and self-reported leaders need not adopt equally early but leave open the question which type of leader adopts before the other.

Whereas many studies have reported evidence that one of these two measures of opinion leadership is associated with early adoption, others have found no effect (e.g., Goldenberg et al. 2009, Van den Bulte and Lilien 2001) or even negative effects (Becker 1970, Leonard-Barton 1985). More importantly, there is no evidence to date of the effect of one after controlling for the other. That is, there is no evidence that both have an *independent* effect on the speed of adoption. Such evidence is critical to the claim that both measures capture different constructs.

The distinction between sociometric and self-reported leadership may also affect how sensitive one is to input from one's peers. Following the original two-step flow hypothesis, several studies have documented that the information flow between opinion leaders and followers is not unidirectional. True experts rarely ignore whatever user experience or other information less prestigious actors have to share (e.g., Strang and Tuma 1993, Weimann 1994). This suggests that sociometric leaders may be as responsive to contagion as non-leaders are (assuming leaders do not adopt too early to ever experience peer influence, of course). Self-reported leaders, in contrast, may be more or less sensitive to contagion than their peers. On the one hand, several theories of social identity and status imply that people with a high sense of self-importance may deem it below their dignity to take into consideration, let alone imitate, the behavior of lower-status actors (Berger and Heath

2007, Van den Bulte and Joshi 2007). On the other hand, status competition implies that people who think of themselves as having above-average status might be driven to adopt quickly once they see others of lower status adopting, out of fear that being outpaced will lead their own status advantage to erode (Burt 1987). Taken together, these arguments imply that self-reported leaders are differentially (either less or more) sensitive to social contagion compared with non-leaders, whereas sociometric leaders are not more or less sensitive than non-leaders.

Empirical support for the clear distinction between sociometric and self-reported leadership would be of theoretical importance because it would imply that they are not different measures of the same construct, as advanced by Rogers and Cartano (1962) and Jacoby (1974), but are distinct theoretical constructs. Investigating the distinction is also of obvious value to marketers seeking to identify whom to target as seeding points in their campaigns.

2.3. Social Contagion Through Central Actors and Heavy Users

A key assumption underlying many network marketing strategies is that some customers are not only better connected but also more influential than are others. Who these customers are is critical for selecting initial targets or "seeding points" in network marketing campaigns. We investigate whether customers who are heavy rather than light users, a characteristic that is easier and cheaper to determine than opinion leadership, are disproportionately influential among those to whom they are connected. Whereas prior research indicates that centrality in the network and usage volume tend to be associated with early adoption (Coulter et al. 2002, Taylor 1977, Weimann 1994), we investigate whether centrality and usage volume also affect how effective one is as a source of influence *after* one has adopted.

The answer to that question is far from obvious. Standard theoretical arguments based on (i) the link between repeat buying behavior and satisfaction or (ii) the link between experience and source credibility imply that someone who adopted a while ago but is not using the product anymore is likely to be less enthusiastic and less credible than someone who is still using the product. Conversely, Godes and Mayzlin (2009) note that heavy users may tend to be connected mostly to people already predisposed to be early adopters. The latter idea implies that heavy users are less likely to generate new adoptions.

Whether usage volume enhances or depresses the amount of social contagion exerted is likely to depend on whether contagion operates by boosting either awareness or evaluation, the two key stages in the adoption process (e.g., Lin and Burt 1975). For products that do not benefit from marketing communication and that present little perceived risk or ambiguity such that little additional information is required in the evaluation stage, Godes and Mayzlin's (2009) argument implies that light users will be very effective sources of influence. For products that are supported by a fair amount of standard marketing communication but pose significant perceived risk or ambiguity to potential adopters, in contrast, contagion fosters adoption by operating at the evaluation stage rather than at the awareness stage, so heavy users are likely to be more effective sources of influence. Testing the heavy-user hypothesis for a product with significant perceived risk and ambiguity complements the study by Godes and Mayzlin (2009) of a low-risk product enjoying very little marketing support and for which contagion operated most likely by boosting awareness rather than evaluation.

Whether usage volume moderates the amount of social contagion exerted might conceivably also depend on the stage of the diffusion cycle. For a firm-created word-of-mouth campaign started well after the product's introduction, such as the one studied by Godes and Mayzlin (2009), it is conceivable that all heavy users have already engaged their network members (either successfully influencing them or not), making heavy users less effective seeding points than light users, who may still have many opportunities left to convert network members. This alternative explanation implies not a reversal but a corroboration of the light-users-are-better finding by Godes and Mayzlin for high-risk versus low-risk products.

3. Research Setting

To provide valid answers to our research questions and an informative assessment of the assumptions underlying many network marketing efforts, the research setting should ideally satisfy several conditions. First, the newly launched product should have characteristics making it theoretically justified to expect contagion to be at work. Second, one must be able to collect data on self-reported leadership and sociometric leadership for each person whose behavior is analyzed. Third, one must have data on who can influence whom. Fourth, one must have data not only on the adoption of each person whose behavior is analyzed but also on the adoption and postadoption usage of others in their network. Fifth, key marketing efforts deployed must be observed or otherwise controlled for.

We secured the cooperation of a pharmaceutical company to meet those stringent conditions. For reasons of anonymity agreed upon with the company, we do not report its identity nor the drug's name, treatment category, or launch date. Like many other firms

in its industry, the company was keen on identifying the physicians with the most central and influential positions and on using that information in its medical education and detailing programs. Managers realized, however, that their premises were in doubt; they were therefore keen on facilitating a study about the importance of social networks, opinion leadership, and marketing effort.

3.1. The Product

The product is a newly launched prescription drug used to treat a specific type of viral infection. There are both short-term (acute) and long-term (chronic) forms of the disease. The chronic form can cause severe damage to internal organs and—if left untreated—sometimes can even lead to patient death. The product we study is the third entry in the category of drugs for treating the chronic condition. No later entries occurred during the observation window.

Because the condition is chronic, physicians cannot observe drug efficacy quickly and adjust a patient's therapy if necessary. There is uncertainty in the medical community regarding the best treatment because there exists little comparative information about the three drugs' long-term efficacy. In an issue of a prestigious medical journal featuring two separate studies documenting the focal drug's effectiveness, an editorial by a director of one of the National Institutes of Health warned that even though the new drug seemed like an excellent treatment option given its low rate of resistance and outstanding potency, the drug's use should—for the time being—be tempered because the medical condition requires long-term therapy.

In short, the drug treats a potentially lethal condition, but there is considerable ambiguity and risk in making the decision to adopt. In such situations characterized by high risk, high complexity, and low observability of results, both theory and research suggest that contagion is likely to be a significant driver of adoption behavior (e.g., Hahn et al. 1994, Rogers 2003).

Those product characteristics also determined how the product was marketed. The complex nature of the treatment decision made detailing (personal selling) the main marketing instrument. There was only very limited medical journal advertising and no direct-to-consumer advertising. There was no sampling either. Given the chronic nature of the therapy, physicians cannot assess effectiveness rapidly. This drastically limits the effectiveness of free samples in triggering the decision whether to use the drug as part of a treatment plan. Even more important is the concern that patients develop resistance when they take a sample but do not continue on the drug.

3.2. The Physicians and Their Local Network

Given the specific medical condition the new drug is treating, the company defined the relevant population as those physicians who had prescribed at least one of the earlier two drugs in the two years prior to the focal drug's launch. Based on American Medical Association membership records, IMS prescription data, and the company's internal records, the company supplied us with a list of such physicians practicing in three large U.S. cities: San Francisco (SF), Los Angeles (LA), and New York City (NYC). Hence, the relevant networks were bounded based on both a positional criterion, being a physician practicing in one of a specific set of five-digit zip codes, and an event criterion, having prescribed at least one of two drugs in the past (Laumann et al. 1989).

When studying opinion leadership and social contagion among physicians, it is important to take into account their localized character. The importance of local as opposed to national opinion leaders is well documented in the modern medical literature (e.g., Doumit et al. 2007, Keating et al. 2007, Kuo et al. 1998). Whereas nationally reputed "expert opinion leaders" may be respected for their research, to most physicians they are much less representative than local "peer opinion leaders" who are members of their own community and face similar patients and working conditions (Locock et al. 2001). The pharmaceutical industry is keenly aware of the importance of such social dynamics at the local level. Better understanding of local opinion leadership dynamics was the main motivation of the pharmaceutical company to make our study possible.

Because the three cities we study are major metropolitan areas, the local networks also contain several national opinion leaders. That the physicians who the company considered to be national opinion leaders also emerged as opinion leaders within their city made the network data fully credible to the managers, who were also quite interested in the identity of prominent local opinion leaders they had overlooked so far.

4. Data Sources

Our data sources consist of a survey of physicians, a commercial data vendor providing physician prescription data, and company records on sales calls to each physician.

4.1. Physician Survey

We used a mail survey to collect data on the physicians' social network ties and characteristics such as patient volume and self-reported opinion leadership. The survey was mailed twice during a two-month period, separated by a reminder postcard. There was also an online link provided for physicians who

Table 1   Response Rates Across the Three Cities

	SF	LA	NYC
Mailing	187	273	372
Returned to sender	37	76	88
Return to sender (%)	19.8	27.8	23.7
Valid addresses	150	197	284
Surveys completed	67	57	69
Response rate (%)	44.5	28.9	24.3

wanted to complete the survey online; about 10% of participants did so. A $75 honorarium was promised for completing the survey within two weeks of receiving it. In SF, the first mailing took place two months before the U.S. product launch; in LA and NYC, it took place 10 months after the U.S. launch.

The response rate in SF was markedly higher than in LA or NYC (Table 1). That may be due to a higher interest in the treatment options in the SF area, where several national thought leaders are based and a sizable population group lives with an above-average risk of contracting the medical condition. It may also be due to the higher quality of the mailing list. For instance, there were a number of instances in LA and NYC where two entries in the list had the same name but different addresses. In any case, the response rates in all three cities (24%–45%) are quite high by both industry and social science standards and do not generate problems for our network-based covariates. We discuss this in more detail in §7.

Physician characteristics. Following Coleman et al. (1966), we collected data on the type of primary practice and physician specialty. We also asked about the number of patients seen and the number referred to other physicians because physicians treating many patients are more likely to prescribe new drugs. To measure self-reported opinion leadership, we adapted the scale of Childers (1986) to our particular research setting. We used six items pertaining to the likelihood and frequency of a physician to interact with other physicians on issues related to the chronic disease. All items were measured on a scale of 1 to 7.[1]

Network ties. Following Coleman et al. (1966), we collected network data using a sociometric survey. We asked each physician to name up to eight physicians with whom he or she feels comfortable discussing the clinical management and treatment of the disease (discussion ties) and up to eight physicians to whom he or she typically refers patients with the disease (referral ties). Both lists may but need not overlap. Within the network boundary, the 67 respondents in SF generated 37 unique nominees for discussion and 24 unique nominees for referral. In LA, the 57 respondents generated 38 unique nominees for discussion and 24 for referral, and in NYC the 69 respondents generated 43 unique nominees for discussion and 22 for referral. Again following Coleman et al. (1966), we excluded physicians who were nominated by survey respondents but who were not part of the original network boundary (e.g., a physician cited by an LA physician but practicing in Irvine, California, or a fellow LA physician who had never prescribed in the category prior to launch).[2] Physicians who were within the network boundary but did not respond to the survey, in contrast, were included in the network. We then built a "discussion" and a "referral" network matrix for each city, with respondents as rows and all network members as columns and with the (i, j)th cell being one when i cited j and zero otherwise. We also constructed "total" network matrices by adding the referral and network matrices in each city. The SF matrices were of size 67×150, those for LA were 57×197, and those for NYC were 69×284. Including all physicians who were part of the network boundary as columns allows us to take into account the contagion emanating from everyone within the network boundary even if they did not respond to the survey.

As has long been known to researchers of contagion in social and spatial networks, symmetry of ties and extradyadic cycles (e.g., a triad with ties from node a to node b, from b to c, and from c to a) can create an endogeneity or reflection problem (e.g., Ord 1975). Our data, however, do not exhibit such a structure. Of the 204 discussion ties among survey respondents, only three are symmetric. Of the 138 referral ties among survey respondents, none is symmetric. Of the 234 "total" ties, only three are symmetric. Also, our data include only one instance of an extradyadic

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[1] In consultation with industry experts, and consistent with findings by Flynn et al. (1994), we excluded one item from the seven-item Childers scale as it was not relevant to our research context. The six items included in our survey were these: In general, do you talk to others doctors about ——? (Never/Very often); When you talk to your colleagues about —— do you...(Offer very little information/Offer a great deal of information); During the past six months, how many physicians have you instructed about ways to treat ——? (Instructed no one/Instructed multiple physicians); Compared to your circle of colleagues, how likely are you to be asked about ways to treat ——? (Not at all likely to be asked/Very likely to be asked); In discussions of ——, which of the following happens more often? (Your colleagues tell you about treatments/You tell your colleagues about treatments); In general, when you think about your professional interactions with colleagues, are you...(Not used as a source

of advice/Often used as a source of advice). In these items, "——" stands for the medical condition treated by the focal drug.

[2] Robustness checks reported in the electronic companion to this paper, available as part of the online version that can be found at http://mktsci.pubs.informs.org/, provide no evidence that this exclusion affects our results.

cycle: the three symmetric ties just mentioned pertain to a triad in the NYC discussion network.[3] Thus, reflection does not constitute a likely threat to internal validity of our contagion analyses.

4.2. Prescription Data

For each physician within the network boundary (not only respondents), the time of adoption is measured using monthly individual-level prescription data from IMS Health, a data provider whose role and reputation in the pharmaceutical industry is similar to that of ACNielsen and IRI in consumer package goods. For the focal drug, the data start from the month the drug was introduced. Prescriptions were tracked for the next 17 months—incidentally, the same duration as that in *Medical Innovation* (Coleman et al. 1966). Data on postadoption prescriptions are available as well.

Of the 193 doctors across the three cities who responded to the survey, 68 adopted within 17 months. This adoption rate of 35% is markedly lower than the 87% rate for tetracycline in *Medical Innovation* over the same length of time, consistent with the notion that the present drug poses a greater risk to physicians than tetracycline did.

We also have prescription data for the two other drugs in the category for two years prior to the launch of the focal drug. This allows us to identify the heavy prescribers in the category before the focal drug was introduced and to avoid problems that might occur if the firm targeted heavy prescribers with a higher level of marketing effort. By including the variable available to the decision maker in the model, we avoid an endogeneity bias in the effect of marketing variables included in the model (sales calls) and control for other targeted marketing efforts excluded from the model (e.g., direct mail).

4.3. Sales Call Data

From the company's internal records, we obtained data on the number of sales calls (detailing efforts) pertaining to the focal drug for each of the physicians and in each of the 17 months we track. Free samples were not distributed, and the price did not vary over time.

5. Data Analysis Approach

We use hazard modeling as the main statistical approach to analyze the data and test the hypotheses. We operationalize the time of adoption as the time of first prescription (e.g., Coleman et al. 1966). Because

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[3] Because only ties between physicians who both responded to the survey can be shown to be symmetric or to form cycles, this specific analysis is limited to ties among respondents and excludes ties between respondents and nonrespondents.

no samples were distributed, the first recorded prescription corresponds to the actual time of adoption. For each physician-month, we create a binary adoption indicator variable y_{it} that is set to zero if physician i has not adopted by period t and is set to one if he has. The discrete-time hazard of adoption is then modeled as

$$P(y_{it} = 1 \mid y_{it-1} = 0) = F(x_{it}\beta), \qquad (1)$$

where x_{it} is a row vector of covariates, β is a column vector of parameters to be estimated, and F is a cumulative distribution function (e.g., logistic or standard normal). Because the population of interest consists only of physicians who had prescribed within the category at least once in the two years prior to the focal drug's launch, we consider each and every physician to be at risk of adopting the new drug. Because we observe all physicians from the time of launch, left-censoring does not exist in our data. Hence, we use the standard log-likelihood function for discrete-time hazard processes, which appropriately handles right-censoring and can be expressed as

$$LL = \sum_{i=1}^{N} \sum_{t=1}^{T_i} y_{it} \ln[F(x_{it}\beta)] + [1 - y_{it}] \ln[1 - F(x_{it}\beta)], \quad (2)$$

where T_i is the number of monthly observations on physician i and N is the total number of physicians.

6. Covariates

6.1. Opinion Leadership

Indegree centrality. Indegree the number of other physicians who nominate or "send network ties to" a particular physician; it is computed for each physician separately in the referral, discussion, and total network (in the latter network, *Indegree* is the sum of the *Indegree* in the referral and discussion networks). Indegree centrality is the most basic measure of status or prestige in a network (e.g., Van den Bulte and Wuyts 2007). Because we measured the social ties as pertaining to patient referral and discussion of the treatment of a medical condition, physicians with high *Indegree* are, in the parlance of Goldenberg et al. (2006), both "social connectors" having many ties and recognized "experts" with expertise and good judgment.

Self-reported Leadership. The reliability of the six-item scale was quite high (Cronbach $\alpha = 0.88$), and factor analysis confirmed the metric validity of the scale. We construct the *Self-reported Leadership* variable by taking the average of the six items.

6.2. Social Contagion

We operationalize exposure to prior adopters through social ties using lagged endogenous autocorrelation terms. The extent to which physician i is exposed at time t to prior adoptions is captured through the term $\Sigma_j w_{ij} z_{jt-1}$, where w_{ij} captures how relevant each physician j is to i and z_{jt-1} is a variable capturing the behavior of j at time $t-1$.[4]

The social network weight w_{ij} can be constructed in various ways (e.g., Valente 1995). We use a simple weighting scheme of contagion through direct ties: in both the referral and the discussion network, w_{ij} equals one if i nominates j, and it equals zero otherwise. In the total network, the weights are simply the sum of the referral and discussion weights. Because $Indegree_j = \Sigma_i w_{ij}$, the number of colleagues that a physician can influence directly through discussion or referral ties equals his *Indegree*. The number he can influence indirectly is of course greater.

We capture the behavior of fellow physicians in three different ways.

(1) *Adoption.* In this variant, $z_{jt-1} = y_{jt-1}$, i.e., the lagged adoption indicator. The resulting contagion variable assumes that people start influencing once they have adopted and that they continue doing so. This operationalization is the one commonly used in models of network contagion in the adoption of innovations.

(2) *Use.* In this variant, $z_{jt-1} = s_{jt-1}$, where s_{jt-1} is set to 1 if j wrote at least one prescription at time $t-1$ and is set to 0 if he did not. The resulting contagion variable assumes that only recent prescribers exert peer influence (e.g., for reasons of enthusiasm or credibility).

(3) *Volume.* In this variant, $z_{jt-1} = q_{jt-1}$, i.e., the number of prescriptions written by j at time $t-1$. The resulting contagion variable assumes that one's influence is proportional to one's recent prescription volume (e.g., again for reasons of enthusiasm or credibility).

Having operationalized both the social network weights w_{ij} and the various kinds of behavior z_{jt-1}, we calculate the extent of social network exposure physician i is experiencing at time t and create the following variables: *Adoption Contagion*, *Use Contagion*, and *Volume Contagion*. To assess to what extent sociometric and self-reported leadership moderates the effect of these contagion variables, we also create the necessary interaction terms.

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[4] Lagging avoids endogeneity problems, unless (1) people are forward-looking not only about their own behavior but also that of others, *and* (2) social ties over which influence flows are symmetric. The first condition is quite unlikely in large networks, and the second condition does not hold in our data. Of course, if the contagion in the data-generating process is contemporaneous, lagging creates misspecification bias. We find no such evidence (see the electronic companion for details).

6.3. Marketing Effort

We use monthly physician-level detailing (sales calls) as our measure of marketing effort. To allow for effects spanning multiple months, we construct a depreciation-adjusted stock measure. Let D_{it} be the amount of detailing (number of sales calls) received by physician i in month t. The detailing stock of physician i for month t (DS_{it}) is then defined as follows:

$$DS_{it} = D_{it} + \delta DS_{it-1} = \sum_{\tau=1}^{t} \delta^{t-\tau} D_{i\tau}, \qquad (3)$$

where δ is the monthly carryover rate bounded between zero and one, and detailing stock in month 1 is the amount of detailing in that month.[5] To control for a potential confound in the interaction between contagion and the leadership variables, we allow the effect of marketing effort to be moderated by *Indegree* and *Self-reported Leadership*.

6.4. Control Variables

Physician characteristics. We control for several physician characteristics that industry experts and prior research suggest may be associated with early adoption (e.g., Coleman et al. 1966, Rogers 2003). Identifying systematic heterogeneity in adoption time is also of practical interest to managers seeking to identify and target likely early adopters. *University/Teaching Hospital* is a dummy variable indicating whether the physician works in or is affiliated with a university or teaching hospital. *Solo Practice* is a dummy variable capturing whether the doctor is in solo practice or not. Although this variable was important in the original *Medical Innovation* study, it is not clear a priori whether practicing solo is a useful predictor once one takes into account actual network exposure to previous adopters. *Early Referral* is a dummy variable taking the value one if the physician reports sometimes referring patients to other doctors before initiating any treatment, and zero otherwise. A doctor referring patients even before starting any treatment is less likely to adopt the focal drug early. *Primary Care* is a dummy variable capturing whether the doctor is a primary care physician rather than a specialist (internal medicine, gastroenterology, infectious disease) who is more likely to focus on the relevant medical condition. *Patients Managed* is the number of patients with the medical condition that the physician reported clinically managing in the last six months. Physicians with many patients may adopt sooner.

Category-level prescription volume. Prior research suggests that early adopters and opinion leaders tend

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[5] The carryover parameter δ is estimated jointly with the vector of slope parameters β using standard maximum likelihood.

to be heavy users (Coulter et al. 2002, Taylor 1977, Weimann 1994). Thus, to avoid confounds in our hypothesis tests, we control for the physicians' prescription volume. Because no one uses the product before it is launched, usage volume should be measured at the category level prior to the new product's launch to be useful in identifying early adopters and to avoid reverse causality problems. We therefore use the number of prescriptions for each of the other two drugs during the 12 months prior to the launch of the focal drug.[6] As mentioned above, including these variables, *Drug 1* and *Drug 2*, also avoids a potential endogeneity problem in the detailing levels.

Outdegree is the number of nominations given or "network ties sent" by a physician to others and is computed for each physician separately for discussion, referral, and in total. Given the importance of out-of-town contacts in the study by Coleman et al. (1966), *Outdegree* includes nominations to both in-town and out-of-town colleagues. Unlike *Indegree*, *Outdegree* is not a measure of status or prestige. Simply connecting to many people may be related to being an opinion leader but it may as well indicate a lack of expertise and confidence (Van den Bulte and Wuyts 2007). Hence we include *Outdegree* as a control variable allowing a sharper interpretation of the *Indegree* effect. If *Indegree* is associated with early adoption but *Outdegree* is not, one can be more confident in the interpretation of the former as a measure of status.

City dummies. We control for city-specific differences in the propensity to adopt early by including dummy variables for LA and NYC, treating SF as the reference.

Time dummies. We include a dummy for each period. This has two advantages. First, it captures the effect of any systemwide time-varying factor, such as changes in disease prevalence or the appearance of new clinical evidence. The dummies capture all cross-temporal variation in the mean tendency to adopt, leaving only variance across physicians within particular months to be explained by contagion. As a result, including the dummies provides a stringent test for the presence of network contagion. The second advantage of including monthly dummies is that it provides a nonparametric control for duration dependence. This, in turn, absorbs much of the effects of possible unobserved heterogeneity in hazard models (e.g., Dolton

and van der Klaauw 1995, Meyer 1990). Robustness checks indeed do not detect any evidence of unaccounted unobserved heterogeneity (see the electronic companion).

7. Final Data Set

Data on past prescription of the two other oral antivirals are missing for eight doctors, three of whom had adopted the focal drug. We dropped these eight physicians from our data set. Thus, our analyses are based on data from 185 doctors, 65 of whom had adopted the focal drug after 17 months.

7.1. Descriptive Statistics

We organize the data set as a panel from which all postadoption observations are deleted because they do not contribute to the likelihood function of hazard models. Table 2 presents the descriptive statistics for these data. In this unbalanced panel, physicians' weight in computing the means and correlations equals the number of months until they adopt or are right-censored. Table 3 reports the descriptive statistics for the time-invariant covariates using equal weighting.

Figure 1 shows the diffusion curve, i.e., the cumulative proportion of physicians who adopted, and Figure 2 shows the empirical hazard rate, i.e., the number of adopters divided by the number of those who have not adopted before. The diffusion curve does not have a pronounced S-shape, and the hazard rate does not exhibit an upward trend. This, however, does not imply the absence of contagion because heterogeneity across physicians, which generates a downward bias in the duration dependence, is not accounted for and because the detailing efforts targeted toward physicians who have not yet adopted (called physicians "at risk" of adopting) have a clear downward trend (Figure 3). That the marketing effort toward at-risk physicians decreases over time yet the empirical hazard rate does not suggests that, controlling for marketing effort, the hazard rate may actually be increasing, which would be consistent with contagion being at work.

Figure 4 shows how the three contagion variables for the total network evolve over time among the physicians at risk. Both *Adoption Contagion* and *Use Contagion* increase for the first nine months and tend to level off afterward.[7] Put simply, the exposure to both adopters and prescribers stopped growing after

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[6] We also use the number of prescriptions over the six months prior to launch and over two years prior to launch. The fit (in log-likelihood) of the model using the one-year window was marginally better than those using the shorter and longer windows, but there were no substantive differences in the results. The same holds for using a three-month moving window of the number of prescriptions for the other two drugs (see the electronic companion for details).

[7] *Adoption Contagion* and *Use Contagion* level off after nine months (Figure 4), even though the total number of adopters keeps growing roughly linearly (Figure 1) because those adopting after month 9 tended to have very low indegree and hence not to exert any contagion on colleagues.

Table 2   Descriptive Statistics and Correlations Among All Covariates for All At-Risk Physician-Month Observations

Variable	Mean	Std. dev.	Min	Max	1	2	3	4	5	6	7	8	9	10	11	12
1. *Adoption* (y_{it})	0.025	0.16	0	1	1.00											
2. *Detailing* (sales calls)	0.29	0.83	0	9	0.23	1.00										
3. *Indegree*—Referral	0.11	0.59	0	17	0.23	0.21	1.00									
4. *Indegree*—Discussion	0.25	0.78	0	19	0.25	0.27	0.82	1.00								
5. *Indegree*—Total	0.36	1.31	0	36	0.25	0.26	0.94	0.97	1.00							
6. *Outdegree*—Referral	1.37	1.39	0	5	0.00	−0.02	0.00	−0.04	−0.02	1.00						
7. *Outdegree*—Discussion	2.36	1.54	0	6	0.03	0.03	0.04	0.02	0.03	0.37	1.00					
8. *Outdegree*—Total	3.73	2.43	0	10	0.02	0.00	0.02	−0.01	0.00	0.81	0.85	1.00				
9. *Self-reported Leadership*	4.25	1.29	1	7	0.11	0.18	0.19	0.25	0.23	−0.17	0.19	0.02	1.00			
10. *LA dummy*	0.31	0.46	0	1	−0.01	−0.02	0.00	−0.04	−0.02	−0.05	0.00	−0.02	0.15	1.00		
11. *NYC dummy*	0.37	0.48	0	1	−0.02	0.04	−0.02	−0.03	−0.02	−0.07	−0.11	−0.11	0.02	−0.52	1.00	
12. *Solo Practice*	0.39	0.49	0	1	0.00	0.13	−0.06	−0.14	−0.03	−0.19	−0.14	−0.12	−0.01	0.09	1.00	
13. *University/Teaching Hospital*	0.21	0.41	0	1	0.00	−0.09	−0.06	−0.04	−0.05	−0.13	0.00	−0.07	0.07	−0.12	0.03	−0.41
14. *Primary Care*	0.13	0.34	0	1	−0.05	−0.10	−0.08	−0.08	−0.08	0.11	−0.05	0.03	−0.24	0.13	−0.14	−0.06
15. *Patients Managed*	36.36	109.42	0	1,200	0.04	0.12	0.08	0.12	0.10	0.03	−0.07	−0.02	0.02	−0.14	0.20	0.14
16. *Early Referral*	0.35	0.48	0	1	−0.07	−0.16	−0.05	−0.13	−0.09	0.21	−0.02	0.11	−0.44	−0.17	0.00	−0.05
17. *Past Drug 1*	10.89	25.76	0	265	0.25	0.50	0.38	0.55	0.50	−0.09	−0.06	−0.09	0.24	−0.07	0.15	0.02
18. *Past Drug 2*	10.45	24.86	0	510	0.25	0.32	0.32	0.42	0.39	−0.02	−0.06	−0.05	0.05	−0.11	0.09	0.09
19. *Contagion*—Referral, adoption	0.57	0.91	0	5	0.02	0.05	0.01	−0.03	−0.01	0.62	0.30	0.55	−0.08	−0.14	−0.11	−0.08
20. *Contagion*—Referral, use	0.54	0.90	0	5	0.03	0.03	0.02	−0.02	−0.00	0.61	0.29	0.54	−0.07	−0.15	−0.09	−0.09
21. *Contagion*—Referral, volume	4.03	10.45	0	104	0.05	0.02	0.02	−0.00	0.00	0.39	0.21	0.35	−0.06	−0.16	−0.09	−0.07
22. *Contagion*—Discussion, adoption	0.65	0.97	0	6	0.04	0.07	0.04	0.00	0.02	0.31	0.36	0.41	−0.06	−0.04	−0..21	−0.11
23. *Contagion*—Discussion, use	0.57	0.89	0	5	0.04	0.03	0.06	0.02	0.04	0.31	0.35	0.40	−0.07	−0.08	−0.19	−0.12
24. *Contagion*—Discussion, volume	3.85	9.64	0	89	0.06	0.00	0.05	0.05	0.05	0.20	0.21	0.25	−0.06	−0.13	−0.15	−0.08
25. *Contagion*—Total, adoption	1.23	1.71	0	9	0.04	0.06	0.03	−0.01	0.01	0.51	0.37	0.53	−0.08	−0.11	−0.18	−0.11
26. *Contagion*—Total, use	1.11	1.63	0	9	0.04	0.03	0.04	0.00	0.02	0.51	0.36	0.52	−0.08	−0.14	−0.16	−0.11
27. *Contagion*—Total, volume	7.88	18.49	0	178	0.06	0.01	0.04	0.02	0.02	0.33	0.23	0.33	−0.07	−0.16	−0.14	−0.08

	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27
13. *University/Teaching Hospital*	1.00														
14. *Primary Care*	−0.01	1.00													
15. *Patients Managed*	−0.13	−0.08	1.00												
16. *Early Referral*	0.13	0.15	0.00	1.00											
17. *Past Drug 1*	−0.07	−0.09	0.22	−0.16	1.00										
18. *Past Drug 2*	−0.06	−0.05	0.46	−0.04	0.53	1.00									
19. *Contagion*—Referral, adoption	−0.11	0.02	0.03	0.13	−0.02	−0.02	1.00								
20. *Contagion*—Referral, use	−0.09	0.00	0.03	0.13	−0.04	−0.02	0.98	1.00							
21. *Contagion*—Referral, volume	−0.11	−0.03	0.02	0.11	−0.04	−0.02	0.72	0.76	1.00						
22. *Contagion*—Discussion, adoption	−0.06	0.02	−0.03	0.09	−0.04	−0.05	0.65	0.62	0.47	1.00					
23. *Contagion*—Discussion, use	−0.06	0.03	−0.03	0.09	−0.05	−0.03	0.63	0.64	0.49	0.96	1.00				
24. *Contagion*—Discussion, volume	−0.08	0.00	−0.01	0.09	−0.05	−0.03	0.47	0.49	0.69	0.67	0.71	1.00			
25. *Contagion*—Total, adoption	−0.09	0.01	0.00	0.12	−0.04	−0.04	0.90	0.87	0.66	0.91	0.88	0.63	1.00		
26. *Contagion*—Total, use	−0.08	0.02	0.00	0.13	−0.05	−0.04	0.89	0.90	0.68	0.87	0.91	0.66	0.97	1.00	
27. *Contagion*—Total, volume	−0.10	−0.02	0.00	0.11	−0.05	−0.03	0.65	0.68	0.93	0.66	0.65	0.91	0.71	0.73	1.00

Notes. Values computed on all physician-month observations for which physician was at risk of adopting, $N = 2{,}575$. All correlations equal to or greater than 0.04 in absolute value are significant at $p \leq 0.05$.

nine months. Under such conditions, the firm's strategy to decrease the sales effort over time might have been inappropriate to drive late adoptions. Instead, increasing detailing once the effect of word of mouth has stalled (i.e., after nine months) might have been more suitable. However, consider how *Volume Contagion* trends upward throughout the entire 17-month observation period. If contagion based on peers' prescription volume is more important than that based on their adoption or user status, then the firm's detailing strategy may have been quite appropriate. This simple analysis shows how the precise nature of the social contagion process may be quite relevant to marketing policy.

Table 3   **Descriptive Statistics and Correlations Among Time-Invariant Covariates**

Variable[a]	Mean	Std. dev.	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
3. *Indegree*—Referral	0.37	1.57	1.00														
4. *Indegree*—Discussion	0.58	1.90	0.95	1.00													
5. *Indegree*—Total	0.95	3.42	0.98	0.99	1.00												
6. *Outdegree*—Referral	1.34	1.43	−0.12	−0.14	−0.13	1.00											
7. *Outdegree*—Discussion	2.40	1.58	−0.09	−0.11	−0.10	0.41	1.00										
8. *Outdegree*—Total	3.74	2.54	−0.13	−0.15	−0.14	0.82	0.86	1.00									
9. *Self-reported Leadership*	4.46	1.34	0.29	0.34	0.32	−0.21	0.13	−0.04	1.00								
10. *LA dummy*	0.31	0.46	−0.09	−0.13	−0.11	−0.02	0.00	0.00	0.09	1.00							
11. *NYC dummy*	0.36	0.48	−0.09	−0.07	−0.08	−0.13	−0.08	−0.12	0.02	−0.49	1.00						
12. *Solo Practice*	0.38	0.49	−0.09	−0.14	−0.12	0.03	−0.12	−0.06	−0.17	0.00	0.04	1.00					
13. *Univ/Teaching Hospital*	0.22	0.41	−0.06	−0.02	−0.04	−0.15	−0.06	−0.12	0.12	−0.09	0.05	−0.41	1.00				
14. *Primary Care*	0.11	0.32	−0.08	−0.09	−0.09	0.09	−0.06	0.01	−0.27	0.13	−0.12	−0.04	−0.02	1.00			
15. *Patients Managed*	44.67	109.82	0.26	0.28	0.28	−0.02	−0.09	−0.07	0.09	−0.16	0.21	0.08	−0.11	−0.09	1.00		
16. *Early Referral*	0.3	0.46	−0.12	−0.17	−0.15	0.21	−0.02	0.11	−0.48	−0.16	0.00	−0.01	0.08	0.17	−0.06	1.00	
17. *Past Drug 1*	21.36	47.11	0.54	0.62	0.59	−0.21	−0.21	−0.25	0.37	−0.11	0.09	−0.02	0.00	−0.13	0.29	−0.22	1.00
18. *Past Drug 2*	21.44	56.55	0.57	0.62	0.60	−0.12	−0.12	−0.14	0.21	−0.12	−0.04	0.03	−0.05	−0.09	0.31	−0.14	0.71

Notes. Values computed on a single observation for each physician, $N = 185$. All correlations equal to or greater than 0.15 in absolute value are significant at $p \leq 0.05$.

[a]The numbers in front of the variables match those in Table 2. Min and Max values are the same as in Table 3.

7.2.   Respondents vs. Nonrespondents

As mentioned earlier, the response rate was markedly higher in San Francisco than in the other two cities. However, the 185 respondents were not significantly different ($p > 0.05$) from the 411 nonrespondents on

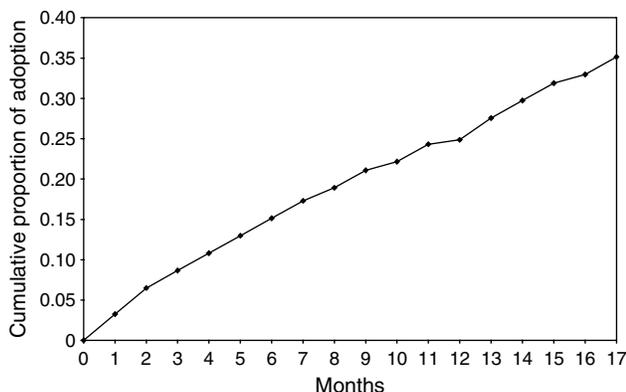**Figure 1**   **Cumulative Proportion of Physicians Having Adopted**

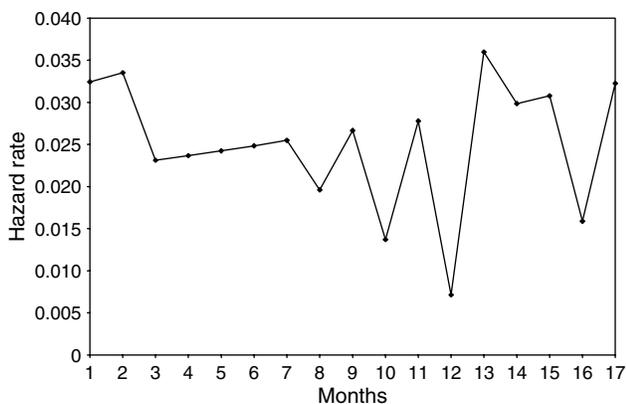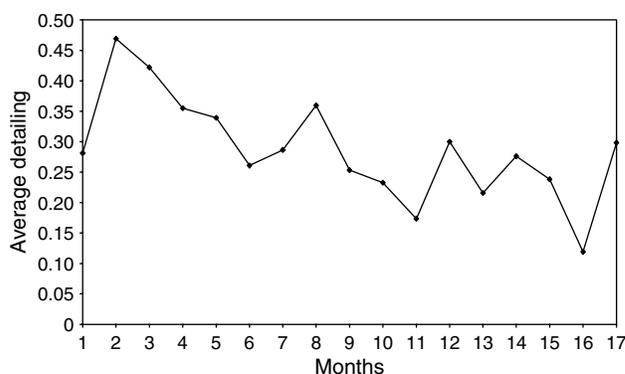**Figure 2**   **Empirical Hazard Rate of Adoption**

**Figure 3**   **Average Detailing Effort per At-Risk Physician**

time-invariant characteristics of focal interest that we observe for both groups:[8] the amount of prescription of two other drugs in the category for 12 months prior to launch (21.4 versus 15.6 for drug 1; 21.4 versus 20.4 for drug 2) and sociometric leadership (total *Indegree*: 0.95 versus 0.49). Moreover, none of those variables was associated significantly with the probability of responding after controlling for city in a multivariate test ($p > 0.05$). Hence, there is no evidence of response bias based on usage or sociometric status.

7.3.   Validity of Network Measures

Nonresponse raises some special concerns in network studies because variables of interest are measured not only on the respondents included in the analysis but also on their connections to nonrespondents. We discuss to what extent response rates of 24%–45% affect our three network variables, (i) *Exposure* to *Prior Adopters*, (ii) *Outdegree*, and (iii) *Indegree*.

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[8] Data on prelaunch prescriptions are missing for 27 of the 438 nonrespondents.

Figure 4 Average Contagion Pressure per At-Risk Physician
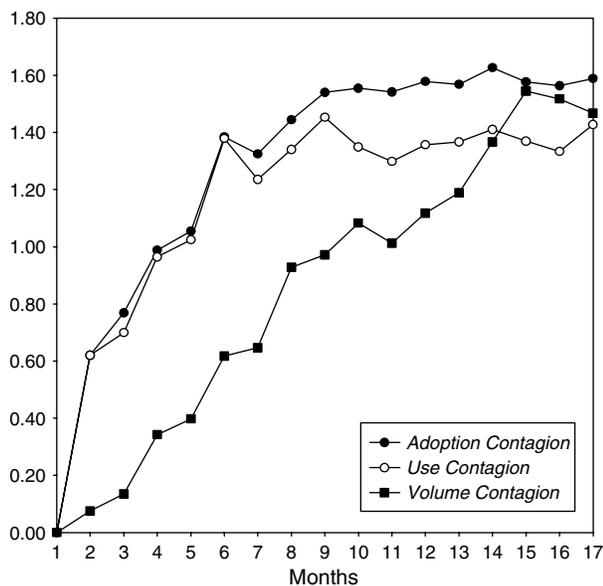
Notes. *Volume Contagion* is divided by 10 in this figure. *Adoption Contagion* decreases at times because the set of physicians at risk varies over time. *Use Contagion* decreases at times for the same reason and because adopters need not use the product every month after adoption.

Contagion Variables. For all responding physicians i whose adoption we are modeling, we observe both their outgoing ties (w_{ij}) and the adoption status, prescription status, or prescription of all their alters in the network (z_{jt-1}). Hence, the variables of social contagion $\Sigma_j w_{ij} z_{jt-1}$ are not affected by nonresponse.

Outdegree. The number of nominations given to others is based on the respondents' own reports and is unaffected by whether those others responded to the survey themselves. Hence, outdegree is not affected by the response rate.

Indegree. The number of nominations received from others or indegree is not based on the respondents' own reports but on reports from others. As a result, the measurement quality of respondents' indegree can be affected by the response rate. The question, then, is whether indegree measures are robust with a 24%–45% response rate from the entire population (we surveyed the entire set of network members, not just a sample). The answer is yes. Obviously, the characteristic scale of the number of nominations received will be biased downward from the true value as the response rate goes down (Leskovec and Faloutsos 2006). In a network of 300 physicians, for instance, the highest possible indegree is 299 but in a 20% sample of 60 physicians it is only 59. The scale parameter, of course, is important for studies that seek to draw inferences about network topology but *not* for studies as ours that seek to relate differences in nodes' indegree to differences in nodes' outcomes or behavior. For the latter, the correlation between the inde-

gree measures in the true and sampled network is what matters. Prior research clearly shows that random node sampling rates of 24%–45% preserve the concordance of the indegree metric between the true (complete) and the measured (sampled) network.

An important study by Costenbader and Valente (2003) of 59 different social networks of sizes similar to ours indicates that indegree in human networks is a robust metric as long as response rates are higher than 10%–20%. McCarty et al. (2007) corroborate that degree centrality is robust at a random node sampling rate of 20% in a study of 447 45-person networks and do so again in a second study of 554 45-person networks. Very similar results have been reported for much larger networks. Studying five networks each with tens of thousands of nodes, Leskovec and Faloutsos (2006) conclude that, after taking into account scaling, one is able to get very good indegree measures using a 15% random node sampling rate. Another study by Kim and Jeong (2007) documents a Pearson correlation of more than 90% between true and measured degree under 20% sampling in a simulated Barabási-Albert network, increasing to more than 95% under 40% sampling. In short, indegree values computed from 20% and 40% random samples of nodes tend to correlate quite highly with the values one would have obtained from the full network.

8. Results

To assess the association between sociometric and self-reported opinion leadership, we use correlation analysis. To test all other hypotheses, we use discrete-time hazard models with a logit link function estimated using standard maximum likelihood. Because models using the total network tended to fit slightly better than models using only the referral or discussion network, and because using the total network follows the reanalyses of the *Medical Innovation* study (e.g., Burt 1987, Strang and Tuma 1993), the results reported here are from models using the total network (robustness checks are reported in the electronic companion).

8.1. Correlation Results

The correlations reported in Table 2 between *Self-reported Leadership* and the various *Indegree* measures are all significantly positive ($p < 0.001$). However, the correlations are low: 0.19 in the referral network, 0.25 in the discussion network, and 0.23 in the total network. The results are similar if one weighs all physicians equally. For instance, the correlation between total *Indegree* and *Self-reported Leadership* in Table 3 is only 0.32 ($p < 0.01$). Analysis by city shows that the correlations do not vary markedly with the response rate (SF: $r = 0.45$, response rate $\phi = 45\%$; LA: $r = 0.32$,

$\phi = 29\%$; NYC: $r = 0.41$, $\phi = 24\%$). Hence, the low correlation between *Indegree* and *Self-reported Leadership* cannot be attributed to purported measurement error stemming from survey nonresponse.[9]

The positive correlations in Tables 2 and 3 between the *Indegree* variables and past prescription of the other two drugs (*Past Drug 1*, *Past Drug 2*) indicate that high-status physicians were heavy prescribers in the category. The positive correlations in Table 2 between the three *Indegree* variables and *Detailing* suggest that high-status physicians were targeted by the firm. There also is a strong positive correlation between *Detailing* and prescription of past drugs, indicating that the firm targeted heavy prescribers of the incumbent drugs.

Finally, *Adoption* (y_{it}) correlates significantly more highly ($p < 0.01$) with referral, discussion, and total *Indegree* ($r = 0.23$, 0.25, and 0.25, respectively) than with *Self-reported Leadership* ($r = 0.11$) (Meng et al. 1992). This indicates that sociometric leaders tend to adopt earlier than self-reported leaders in this study. However, antecedents of adoption are better identified using a hazard model.

8.2. Hazard Model Results

Table 4 shows models for *Adoption*, *Use*, and *Volume Contagion*. For each, we present models with and without interactions between the opinion leadership variables on the one hand and *Detailing* and *Contagion* on the other. The *Indegree* and *Self-reported Leadership* variables are mean-centered, so the linear effects of *Contagion* and *Detailing* are the effects pertaining to the average physician.

We start by discussing the effects of opinion leadership. Physicians with high *Indegree* adopt earlier, and this result is robust across all model specifications. Indegree, although having a strong main effect, does not affect how sensitive physicians are to *Contagion*. *Self-reported Leadership*, in contrast, has no significant effect on time of adoption in the main-effects models (1–3). But models 5 and 6 including interactions indicate that *Self-reported Leadership* indeed was associated with early adoption and that the latter null result stems from two counteracting effects: physicians with high *Self-reported Leadership* have a higher intrinsic tendency to adopt early, but are less sensitive to contagion from peers. In model 6 with *Volume*

Contagion, which fits markedly better than models 4 and 5, this interaction is significant ($p < 0.01$).[10]

These opinion leadership effects are observed even after controlling for heavy use, outdegree, and targeted marketing effort. Physicians' prior prescription level of Drug 2 has a robust effect on their speed of adoption. None of the other physician characteristics does. Additional analyses indicate the absence of interaction effects of *Outdegree* with either *Contagion* or *Detailing* ($p > 0.05$). Hence, *Outdegree* and *Indegree* effects are quite different. *Detailing* has a very significant effect that is robust across model specifications and exhibits a carryover rate of about 45%. Physicians' responsiveness to sales calls does not vary as a function of their *Indegree* or *Self-reported Leadership*.

We now turn to the contagion effects. These require careful interpretation. Among the models without interactions (columns (1)–(3) in Table 4), the model with *Volume Contagion* fits better than the other two and is the only one showing a significant contagion effect.[11] Among the models with interactions (columns (4)–(6)), the model with *Volume Contagion* again fits markedly better than the other two and is again the only showing a significant contagion effect (its interaction with *Self-reported Leadership*). The difference in deviance ($-2LL$) between the models with *Volume Contagion* and *Use Contagion* equals 7.32, which is strong evidence of superior fit because the models have the same number of parameters (Raftery 1995). Also, the presence of a significant interaction involving *Volume Contagion* suggests that model 3 with only main effects is misspecified. Thus, model 6 is the one best supported by the data.

The key finding is that *Volume Contagion* exists (models 3 and 6) and is moderated by physicians' *Self-reported Leadership* but not by *Indegree*, i.e., sociometric leadership (model 6). Additional analysis indicates that *Volume Contagion* has a significant positive effect (at 5%) for physicians with a *Self-reported Leadership* score of 4.25 or lower, which corresponds to physicians at the bottom 43% percent of the distribution. Its effect is never significantly negative. *Adoption Contagion* and *Use Contagion* effects are not significantly positive or negative at any level of *Self-reported Leadership*. That model 6 with *Volume Contagion* fits better

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[9] The correlations between *Indegree* and *Self-reported Leadership* are much lower than the 0.59 correlation between degree centrality and self-reported leadership in a study of children by Kratzer and Lettl (2009). This difference is likely to stem at least in part from the facts that (i) those authors symmetrized the network data so that degree is partly based on *self-reported ties*, making it subject to the same biases as self-reported leadership; and (ii) both their measure of degree and leadership measured the *frequency of interaction*, whereas our measure of *Indegree* captures only the *number* of alters.

[10] In the model with *Volume Contagion*, the total effect of *Self-reported Leadership* becomes significantly negative only for levels of *Volume Contagion* above the 99th percentile. In the models with *Adoption Contagion* and *Use Contagion*, the total effect of *Self-reported Leadership* never becomes significantly negative.

[11] Extending models 3 and 6 with *Adoption Contagion* and *Use Contagion* does not change the main insight: only *Volume Contagion* is significant in the main effects model ($p < 0.05$) and only the interaction between *Self-reported Leadership* and *Volume Contagion* is significant in the model with interactions ($p < 0.05$). For details, see the electronic companion, Table A.3.

Table 4   **Main Results Using the Total Network and Flexible Baseline**

	Basis of contagion			Basis of contagion		
	Adoption (1)	Use (2)	Volume (3)	Adoption (4)	Use (5)	Volume (6)
Intercept	−3.35** (0.68)	−3.43** (0.68)	−3.92** (0.69)	−3.27** (0.71)	−3.41** (0.71)	−3.88** (0.74)
Indegree	0.15* (0.07)	0.15* (0.07)	0.15* (0.07)	0.31* (0.14)	0.32* (0.15)	0.30* (0.15)
Outdegree	0.12 (0.07)	0.10 (0.07)	0.07 (0.06)	0.12 (0.07)	0.11 (0.07)	0.08 (0.06)
Self-reported Leadership	0.19 (0.14)	0.19 (0.14)	0.19 (0.14)	0.37 (0.20)	0.38* (0.19)	0.42* (0.18)
Contagion	−0.03 (0.09)	0.01 (0.09)	0.01* (0.006)	−0.02 (0.10)	0.02 (0.10)	0.01 (0.007)
Detailing stock	0.36** (0.14)	0.36** (0.14)	0.37** (0.14)	0.39** (0.13)	0.39** (0.13)	0.41** (0.14)
Detailing carryover	0.48* (0.25)	0.47 (0.25)	0.43 (0.26)	0.44** (0.20)	0.44* (0.20)	0.44* (0.20)
Indegree × Contagion				0.01 (0.04)	0.01 (0.05)	0.001 (0.005)
Indegree × Detailing stock				−0.05 (0.04)	−0.05 (0.04)	−0.05 (0.04)
Self-reported Leadership × Contagion				−0.09 (0.07)	−0.09 (0.07)	−0.01* (0.005)
Self-reported Leadership × Detailing stock				−0.02 (0.07)	−0.02 (0.07)	−0.05 (0.07)
LA dummy	−0.11 (0.38)	−0.09 (0.43)	0.19 (0.40)	−0.18 (0.39)	−0.14 (0.39)	0.09 (0.42)
NYC dummy	−0.54 (0.41)	−0.49 (0.42)	−0.24 (0.42)	−0.57 (0.42)	−0.51 (0.42)	−0.27 (0.43)
Solo Practice	0.04 (0.34)	0.07 (0.34)	0.11 (0.35)	−0.01 (0.35)	0.01 (0.35)	0.01 (0.35)
University/Teaching Hospital	0.58 (0.40)	0.59 (0.40)	0.72 (0.41)	0.55 (0.41)	0.56 (0.41)	0.69 (0.41)
Primary Care	−0.64 (0.76)	−0.65 (0.76)	−0.61 (0.76)	−0.60 (0.76)	−0.59 (0.76)	−0.57 (0.77)
Early Referral	−0.63 (0.43)	−0.62 (0.43)	−0.64 (0.43)	−0.69 (0.43)	−0.68 (0.43)	−0.77 (0.44)
Patients Managed	0.001 (0.001)	0.001 (0.001)	0.001 (0.001)	0.001 (0.001)	0.001 (0.001)	0.001 (0.001)
Past Drug 1	0.003 (0.004)	0.004 (0.004)	0.003 (0.004)	0.004 (0.004)	0.003 (0.004)	0.002 (0.004)
Past Drug 2	0.01** (0.004)	0.01** (0.004)	0.01** (0.004)	0.01** (0.004)	0.01** (0.004)	0.01** (0.004)
LL	−231.22	−231.28	−229.40	−229.08	−229.14	−225.48

Notes. The numbers in parentheses are the standard errors for the parameters. All models include 16 monthly time dummies and thus have a flexible baseline hazard rate. The log-likelihood (LL) of the model with only an intercept and 16 time dummies is −300.20. The LL of the model with only an intercept is −303.32.

  *Indicates $p \leq 0.05$; **indicates $p \leq 0.01$.

than model 5 with *Use Contagion* indicates that the volume effect stems from differences in peers' prescription volume and not simply from whether one's peers are prescribing or not. In short, *Volume Contagion* has a significant effect, whereas the other two types of contagion do not. This is consistent with the notion that connections to heavy users are more influential in driving adoption than connections to light users. In short, we find evidence of social contagion operating over social network ties even after controlling for targeted marketing effort and time shocks, but it is moderated by both the recipients' self-perceived leadership and the sources' usage volume.

As reported in the electronic companion, our results are quite robust to whether we (i) consider only the discussion network, only the referral network, or

both; (ii) consider contagion in the number versus the proportion of direct contacts who have adopted; (iii) allow for contemporaneous contagion; (iv) add a proxy for adoptions by out-of-city contacts; (v) omit the flexible baseline hazard; (vi) extend the model with random effects or serial correlation; or (vii) allow for time-varying endogeneity in detailing.

Additional analyses reported in the electronic companion indicate that the volume contagion effect most likely stems from prior adopters' *credibility based on experience* with the focal drug rather than from (i) their enthusiasm about the focal drug operationalized as "share of wallet," (ii) their expert status, (iii) their category-level experience, (iv) amplification through detailing leakage, or (v) back-and-forth flow from patients.

9. Managerial Calculus

Because both social contagion and detailing affect adoption, the question arises as to whether focusing one's marketing efforts on opinion leaders is an effective marketing strategy. Our analysis can provide some guidance on this issue.

Assume a network marketing approach enabling the company, in each city, to have the physician with the greatest following (*Indegree*) not only adopting in the first month after launch but also endorsing the product more strongly in his interactions with colleagues who turn to him for discussion or referrals. In terms of our model, we operationalize this increased word-of-mouth activity as a persistent increase of prescription volume by 10 units, although in practice it may (also) take the form of engaging the opinion leader in medical education efforts (e.g., Dorfman and Maynor 2006, Valente et al. 2003).[12] Using our model, we can then compare the expected number of adopters following the intervention against the number in the base scenario where nothing is changed. Such a network-based intervention is of course not costless, but no cost data are available. As a benchmark, we compare the expected effectiveness of the intervention against that of another intervention where each physician receives one additional detail in the first month. Using the model and taking into account the carryover effects of detailing, one can again compare the expected number of adopters with and without the intervention. Assuming both interventions are equally costly, their relative effectiveness reflects their relative efficiency.

The procedure is easily adapted if managers believe that the network intervention requires less or more effort than the equivalent of 185 details.

We apply this logic using the volume-based contagion models both with and without interactions. In both models, the effect of a general detailing impulse declines smoothly over time (because of the partial carryover), whereas that of the network intervention is very small at first but increases steadily over time. The effect is more muted at first in the model with interactions because of the negative interaction between *Self-reported Leadership* and *Volume Contagion*, but the dampening disappears as more and more self-reported leaders adopt over time and drop out of the at-risk set. Comparing the effects of the two interventions using the model without interactions, we find that after eight months, the expected cumulative number of physicians who adopt due specifically to the network intervention exceeds that due specifically to the detailing intervention. In the model with interactions, the crossover happens after 12 months. Because more than two thirds of all physicians still have not adopted by that time, the network intervention is the more attractive of the two.

The procedure just outlined provides a model-based assessment of the likely effectiveness of different interventions. The illustration assumes that having the top three leaders double their effective network influence costs the same as one additional detail to 185 physicians. Depending on managers' beliefs, one might use different inputs and come to different conclusions.

10. Discussion

We conducted a detailed study about the impact of social networks on the adoption of a new drug by physicians. In contrast to earlier studies, we find evidence of contagion operating over network ties, even after controlling for marketing effort and arbitrary systemwide changes. Another novel finding is that adoption is affected by peers' usage volume, rather than by whether peers have adopted or are prescribing. As to opinion leadership, we find that self-reported leadership and sociometric leadership are distinct characteristics: (i) they are weakly correlated, (ii) the tendency to adopt early is more pronounced for sociometric than for self-reported leaders, and (iii) self-reported opinion leaders are less responsive to their contacts' behavior than are sociometric opinion leaders, who are not differentially responsive.

Because our very detailed field study was limited to a single product and three cities, corroboration of these novel findings by subsequent research would be quite useful. This is especially so as both the amount of contagion and the extent to which it increases with the source's usage volume are likely to

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[12] The scenario of this intervention is quite realistic. In SF and LA, the physician with the highest indegree adopted in month 1, and in NYC, he adopted in month 2. Upon adoption, the average prescription volume per month of the three leaders was 10 units. Thus, we simply assume doubling the average prescription volume of leaders.

be contingent on the nature of the product. As noted in §§2.3 and 3.1, theory suggests that these two findings need not generalize to situations where potential adopters face little risk or ambiguity. This caveat is particularly important when considering how our findings have several implications for diffusion theory and research as well as for marketing practice.

10.1. Implications for Diffusion Theory and Research

Several recent studies documenting confounds between contagion and other causal mechanisms have challenged the fundamental notion of social contagion being an important driver of new product diffusion. Our study is important as it presents the strongest evidence to date of contagion over network ties affecting adoption in a naturalistic setting, after controlling for marketing effort and arbitrary common shocks.

The evidence that self-reported and sociometric leadership are weakly correlated and behave differently within the nomological network of constructs is also quite novel. Our evidence indicates that the two measures most probably tap into different constructs. This issue warrants further investigation. Recent research on a distinction between expertise and social connectivity (Goldenberg et al. 2006, Locock et al. 2001) is a step in the right direction, and more is needed.

We also present important new results on how contagion and opinion leadership interact. People who perceived themselves to be opinion leaders responded less to peer behavior. This finding is consistent with standard perceived risk arguments as well as status maintenance mechanisms (e.g., Van den Bulte and Joshi 2007). However, it may also be consistent with social identity considerations, where people react positively to adoptions by people like them and negatively to those by people unlike them (e.g., Berger and Heath 2007). Studies that differentiate between mechanisms involving risk mitigation, "vertical" status, and "horizontal" social identity have the potential to provide a deeper understanding of contagion and new product diffusion processes than we currently have.

Some recent work has argued that opinion leaders central in the network may adopt early not because they are innovative but because they are exposed to more social influence early on through their many social ties (Goldenberg et al. 2009). Our study, in contrast, documents that both mechanisms can be at work simultaneously: sociometric leadership was associated with early adoption even after controlling for contagion, and sociometric leaders were equally sensitive to contagion as non-leaders. More importantly, given differences between studies by Watts and Dodds (2007) and Goldenberg et al. (2009) coming to opposite conclusions in this matter, our evidence of

both mechanisms operating is robust to whether we consider contagion in terms of the number or of the proportion of one's peers who are prescribing.

We found that contagion was affected less by peers' adoption or user status than by their usage volume. Several post hoc analyses suggest that this is likely to stem from a source credibility mechanism. Physicians who prescribe the new drug a lot are a more credible source of information: not only do they act in accordance to their own recommendation, but they also have a larger experiential base on which to found their recommendations. Research documenting in greater detail the sources of relevance and credibility in word-of-mouth communication would be valuable (Goldenberg et al. 2006).

It is also possible that usage volume is important in contagion because it correlates not with the persuasiveness of the source but with the valence of its outcomes and recommendations. Because people who use a product extensively are more likely to be satisfied with its performance, it is possible that volume contagion acts as a proxy for vicarious learning about postadoption outcomes (e.g., Haunschild and Miner 1997). Although our post hoc analyses of contagion through share of prescription (share of wallet) provide less support for this process in this specific study (see the electronic companion), research on the role of postadoption outcomes and satisfaction in contagion dynamics could further our understanding of contagion processes and of how marketers can use them to their benefit.

Our results on volume-based contagion corroborate the argument by Godes and Mayzlin (2009) that heavy users are likely to be more influential than light users when contagion fosters adoption by operating at the evaluation stage rather than at the awareness stage. Our results complement their finding of a larger effect of light users for a product that did not benefit from standard marketing communication and that presented little perceived risk. Further research on the role of usage behavior in contagion dynamics could enhance our understanding of contagion processes and provide useful guidance to managers on whether heavy users or light users are the more attractive seeding points for marketing campaigns.

10.2. Implications for Marketing Practice

Our results support the use of network-leveraging campaigns hinging on central influentials exerting above-average social influence on other customers, a practice about which doubts have recently arisen (Thompson 2008, Watts and Dodds 2007). Note our evidence pertains to a risky product for which one would expect contagion to matter and does not invalidate the warning that contagion cannot simply be taken for granted in every situation. Another caveat is that our study documents that well-connected people

are more influential than others are, but it does not take into account the marketing cost of identifying and converting them. Still, combining model results and their own judgments, managers can assess the attractiveness of a network marketing approach and compare the expected results with those of more traditional marketing.

Our study suggests the existence of hitherto neglected benefits of targeting sociometric opinion leaders. The standard argument is that they influence more peers than less centrally located people do. Our results are consistent with this argument but suggest two additional benefits. First, the stand-alone customer lifetime value (CLV) of opinion leaders may be higher than that of other people because they tend to be early adopters and heavy users. Second, their "network" value may be higher not only because they reach more people but also because by being early adopters and heavy users, they start influencing others sooner and more effectively than do less connected people. Here again, some caveats are due. First, if opinion leaders tend not only to adopt but also to disadopt sooner than others, and if the firm's discount rate is low, then the stand-alone CLV of an opinion leader need not be systematically above average. Second, a customer's heavy use may boost his network value only if the product is perceived to be risky and heavy users are more credible or otherwise more influential than light users (Godes and Mayzlin 2009). Third, when the new product challenges the power base or norms of the opinion leaders, the product is likely to be resisted by them and to be adopted first by members at the fringe of the network (Becker 1970, Leonard-Barton 1985, Valente 1995).

Managers should also take note that heavy prescribers of the last drug launched in the category tended to adopt the new drug early and also tended to be opinion leaders. It suggests that the industry practice to overweight one's marketing efforts at launch toward heavy prescribers is sound to generate not only quick trial sales but also a larger contagion effect. Specifically, heavy users have a higher stand-alone value both because they adopt early and because they use more after they adopt. They also have a higher network value both because they tend to have more connections and because they tend to be more influential within each of those connections. However, because the correlation between prescription volume and sociometric leadership is only moderate, focusing only on heavy users will fail to leverage all potential influential seeding points.

10.3. Conclusion

Just as marketers are rediscovering the idea of leveraging customer networks to accelerate new products' market acceptance, network researchers have started to challenge the basic premises of this practice (Van den Bulte and Lilien 2001, Watts and Dodds 2007). Network and diffusion researchers as well as practitioners will find it encouraging that we were able to document contagion effects operating over social networks, even after controlling for targeted marketing effort and arbitrary systemwide changes. Similarly, our findings about sociometric versus self-reported opinion leadership and about contagion being moderated by usage volume suggest not only venues to gain richer theoretical understanding of social contagion but also ways through which one might ultimately increase the effectiveness of network marketing.

11. Electronic Companion

An electronic companion to this paper is available as part of the online version that can be found at http://mktsci.pubs.informs.org/.

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