Opinion Leadership and Social Contagion in New Product Diffusion

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Abstract

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 system-wide 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.

Keywords: Diffusion of innovations, Opinion leadership, Social contagion, Social networks.

1. Introduction

Marketers are increasingly experimenting with various forms of network marketing. In the area of new product marketing, the rationale of 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) noted disagreement on whether to identify opinion leaders based on their self-reports or their centrality in social networks. More recent research by Coulter, Feick and Price (2002) and Godes and Mayzlin (2009) provides conflicting answers to the question 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 system-wide 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 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. While 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 group membership (e.g., Duflo and Saez 2003; Sacerdote

2001), or 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 have 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 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 with sociometric leadership being 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, common in the pharmaceutical industry but also elsewhere, of targeting heavy users is justifiable based not only on their higher "stand-alone" customer lifetime value, but also on their higher "network value" since they exert more social contagion. Yet, since 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. While this is often taken for granted, it need not always be warranted. For instance, several studies have documented inflated evidence of contagion due to estimation problems and using theoretically over-determined 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 re-analyses 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 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).

So, we assess whether new product adoption is subject to social contagion operating through network ties such that better connected adopters exert more influence than less connected ones,

and whether such contagion operates over and above the effect of targeted marketing efforts and system-wide 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 so 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) discussed 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 whom they turn to 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 some consumer network marketing firms like P&G's Vocalpoint who 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 upwards and that it also reflects self-confidence rather

than actual influence. Conversely, doubts on 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. While 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 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. Since 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 conducted over 30 years ago (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.

While 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 to date 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 to 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, as it would imply that they are not different measures of the same construct, as advanced by Rogers and Cartano (1962) and Jacoby (1974), but distinct theoretical constructs. Investigating the distinction is also of obvious value to marketers seeking to identify who 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 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 they are connected to. While 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, heavy users may tend to be connected

mostly to people already predisposed to be early adopters. This would imply 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, 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 post-adoption 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 or 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 and 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 even lead to patients' 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.

As 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 as 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 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 of patients developing 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 AMA membership records, IMS

prescription data, and its internal records, the company supplied us with a list of such physicians practicing in three large US 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 5-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 local opinion leadership dynamics was the main motivation of the pharmaceutical company to make our study possible.

Since 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 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, and in LA and NYC it took place ten months after 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 Section 7.

Table 1: Response Rates across the Three Cities

	San Francisco (SF)	Los Angeles (LA)	New York City (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

Physician characteristics. Following Coleman, Katz and Menzel (1966), we collected data on the type of primary practice and physician specialty. We also asked about the number of patients seen and referred to other physicians, as 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, Katz and Menzel (1966), we collected network data using a sociometric survey. We asked each physician to name up to eight physicians with whom they feel comfortable discussing the clinical management and treatment of the disease (discussion ties) and up to eight physicians to whom they typically refer 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 and 24 unique nominees, and in NYC the 69 respondents generated 43 and 22 unique nominees. Again following Coleman, Katz and Menzel (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, CA, or a fellow LA physician who had never prescribed in the category prior to launch).[2] Physicians who

[1] In consultation with industry experts, and consistent with findings by Flynn, Goldsmith and Eastman (1994), we excluded one item from the 7-item Childers scale as it was not relevant to our research context. The 6 items included in our survey were: 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 6 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 Technical Appendix provide no evidence that this exclusion affects our results.

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 1 when i cited j and 0 otherwise. We also constructed "total" network matrices by adding the referral and network matrices in each city. The SF matrices were of size 67x150, those for LA were 57x197, and those for NYC were 69x284. 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 extra-dyadic 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 structure. Of the 204 discussion ties among survey respondents, only 3 are symmetric. Of the 138 referral ties among survey respondents, none is symmetric. Of the 234 "total" ties, only 3 are symmetric. Also, our data include only one instance of extra-dyadic cycles: the 3 symmetric ties just mentioned pertain to a triad in the NYC discussion network.[3] So, reflection does not constitute a 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 AC Nielsen and IRI in consumer package goods. For the focal drug, the data start from the month the drug was

[3] Since 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 non-respondents.

introduced. Prescriptions were tracked for the next seventeen months—incidentally the same duration as that in *Medical Innovation* (Coleman et al. 1966). Data on post-adoption 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 than the 87% rate for tetracycline in *Medical Innovation* over the same length of time, consistent with the notion that the present drug poses 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). As 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 0 if physician i has not adopted by period t and is set to 1 if he has. The discrete-time hazard of adoption is then modeled as:

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

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). Since 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. Since we observe all physicians from the time of launch, left-censoring does not exist in our data. Hence, we use the standard loglikelihood function for discrete-time hazard processes which appropriately handles right-censoring and can be expressed as:

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

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. This is the number of other physicians who nominate or "send network ties to" a particular physician, and is computed for each physician separately in the referral, discussion, and total network (in the latter, 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). Since 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 α = 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 1 if *i* nominates *j* and it equals 0 otherwise. In the total network, the weights are simply the sum of the referral and discussion weights. Since $\text{Indegree}_j = \Sigma_i w_{ij}$, 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

[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 Technical Appendix for details).

contagion in the adoption of innovations.

(2) *Use.* In this variant, $z_{jt\text{-}1} = s_{jt\text{-}1}$, where $s_{jt\text{-}1}$ 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, $z_{jt\text{-}1} = q_{jt\text{-}1}$, 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 $z_{jt\text{-}1}$, 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.

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 salescalls) 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 carry-over rate bounded between 0 and 1, 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

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

moderated by Indegree and Self-reported Leadership.

6.4. Control variables

Physician characteristics. We control for several physician characteristics which 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. While 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 1 if the physician reports sometimes referring patients to other doctors before initiating any treatment, and 0 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 who is more likely to focus on the relevant medical condition (internal medicine, gastroenterologists, infectious diseases). *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 to be heavy users (Coulter et al. 2002; Taylor 1977; Weimann 1994). So, to avoid confounds in our hypothesis tests, we control for the physicians' prescription volume. Since 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, Drug 1 and Drug 2, during the twelve months prior to the launch of the focal drug.[6] As mentioned above, including these variables 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, Katz and Menzel (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 system-wide 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

[6]We also used 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 window 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 Technical Appendix for details).

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 (Technical Appendix).

7. Final data set

Data on past prescription of the two other oral antivirals (Past Drug 1, Past Drug 2) are missing for 8 doctors, 3 of whom had adopted the focal drug. We dropped these 8 physicians from our dataset. 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 post-adoption observations are deleted since 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, since heterogeneity across physicians which generates a downward bias in the duration dependence is not accounted for and since the detailing efforts targeted towards

Table 2: Descriptive Statistics and Correlations among All Covariates for all At-Risk Physician-Months Observations

Variable	Mean	SD	Min	Max	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1. Adoption (y_{it})	0.025	0.16	0	1	1.00																			
2. Detailing (Sales Calls)	0.29	0.83	0	9	.23	1.00																		
3. Indegree - Referral	0.11	0.59	0	17	.23	.21	1.00																	
4. Indegree - Discussion	0.25	0.78	0	19	.25	.27	.82	1.00																
5. Indegree - Total	0.36	1.31	0	36	.25	.26	.94	.97	1.00															
6. Outdegree - Referral	1.37	1.39	0	5	.00	-.02	.00	-.04	-.02	1.00														
7. Outdegree - Discussion	2.36	1.54	0	6	.03	.03	.04	.02	.03	.37	1.00													
8. Outdegree - Total	3.73	2.43	0	10	.02	.00	.02	-.01	.00	.81	.85	1.00												
9. Self-reported Leadership	4.25	1.29	1	7	.11	.18	.19	.25	.23	-.17	.19	.02	1.00											
10. LA Dummy	0.31	0.46	0	1	-.01	-.02	.00	-.04	-.02	-.05	.00	-.02	.15	1.00										
11. NYC Dummy	0.37	0.48	0	1	-.02	.04	-.02	-.03	-.02	-.07	-.11	-.11	.02	-.52	1.00									
12. Solo Practice	0.39	0.49	0	1	.00	.13	-.06	-.14	-.11	-.03	-.19	-.14	-.12	-.01	.09	1.00								
13. University/Teaching Hospital	0.21	0.41	0	1	.00	-.09	-.06	-.04	-.05	-.13	.00	-.07	.07	-.12	.03	-.41	1.00							
14. Primary Care	0.13	0.34	0	1	-.05	-.10	-.08	-.08	-.08	.11	-.05	.03	-.24	.13	-.14	-.06	-.01	1.00						
15. Patients Managed	36.36	109.42	0	1200	.04	.12	.08	.12	.10	.03	-.07	-.02	.02	-.14	.20	.14	-.13	-.08	1.00					
16. Early Referral	0.35	0.48	0	1	-.07	-.16	-.05	-.13	-.09	.21	-.02	.11	-.44	-.17	.00	-.05	.13	.15	.00	1.00				
17. Past Drug 1	10.89	25.76	0	265	.25	.50	.38	.55	.50	-.09	-.06	-.09	.24	-.07	.15	.02	-.07	-.09	.22	-.16	1.00			
18. Past Drug 2	10.45	24.86	0	510	.25	.32	.32	.42	.39	-.02	-.06	-.05	.05	-.11	.09	.09	-.06	-.05	.46	-.04	.53	1.00		
19. Contagion - Referral, Adoption	0.57	0.91	0	5	.02	.05	.01	-.03	-.01	.62	.30	.55	-.08	-.14	-.11	-.08	-.11	.02	.03	.13	-.02	-.02	1.00	
20. Contagion - Referral, Use	0.54	0.90	0	5	.03	.03	.02	-.02	-.00	.61	.29	.54	-.07	-.15	-.09	-.09	-.09	.00	.03	.13	-.04	-.02	.98	1.00
21. Contagion - Referral, Volume	4.03	10.45	0	104	.05	.02	.02	-.00	.00	.39	.21	.35	-.06	-.16	-.09	-.07	-.11	-.03	.02	.11	-.04	-.02	.72	.76
22. Contagion - Discussion, Adoption	0.65	0.97	0	6	.04	.07	.04	.00	.02	.31	.36	.41	-.06	-.04	-..21	-.11	-.06	.02	-.03	.09	-.04	-.05	.65	.62
23. Contagion - Discussion, Use	0.57	0.89	0	5	.04	.03	.06	.02	.04	.31	.35	.40	-.07	-.08	-.19	-.12	-.06	.03	-.03	.09	-.05	-.03	.63	.64
24. Contagion - Discussion, Volume	3.85	9.64	0	89	.06	.00	.05	.05	.05	.20	.21	.25	-.06	-.13	-.15	-.08	-.08	.00	-.01	.09	-.05	-.03	.47	.49
25. Contagion - Total, Adoption	1.23	1.71	0	9	.04	.06	.03	-.01	.01	.51	.37	.53	-.08	-.11	-.18	-.11	-.09	.01	.00	.12	-.04	-.04	.90	.87
26. Contagion - Total, Use	1.11	1.63	0	9	.04	.03	.04	.00	.02	.51	.36	.52	-.08	-.14	-.16	-.11	-.08	.02	.00	.13	-.05	-.04	.89	.90
27. Contagion - Total, Volume	7.88	18.49	0	178	.06	.01	.04	.02	.02	.33	.23	.33	-.07	-.16	-.14	-.08	-.10	-.02	.00	.11	-.05	-.03	.65	.68

Table 2: Descriptive Statistics and Correlations among All Covariates for all At-Risk Physician-Months Observations --Continued

	21	22	23	24	25	26	27
21. Contagion - Referral, Volume	1.00						
22. Contagion - Discussion, Adoption	.47	1.00					
23. Contagion - Discussion, Use	.49	.96	1.00				
24. Contagion - Discussion, Volume	.69	.67	.71	1.00			
25. Contagion - Total, Adoption	.66	.91	.88	.63	1.00		
26. Contagion - Total, Use	.68	.87	.91	.66	.97	1.00	
27. Contagion - Total, Volume	.93	.66	.65	.91	.71	.73	1.00

Note: Values computed on all physician-month observations for which physician was at risk of adopting, $N = 2575$. All correlations equal or larger than 0.04 are significant at $p \leq .05$.

Figure 1: Cumulative Proportion of Physicians Having Adopted

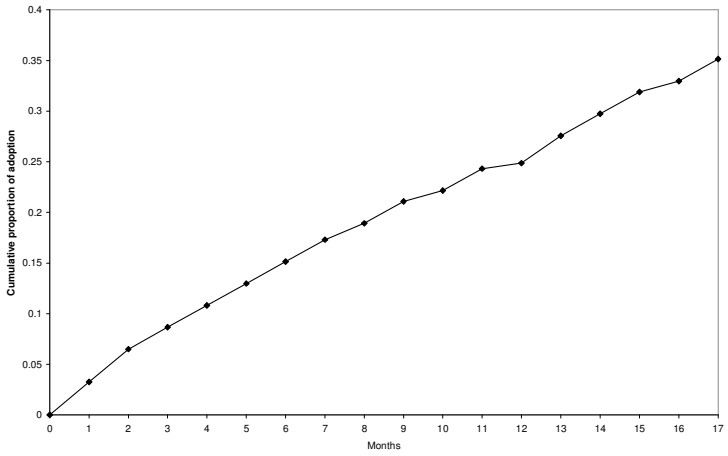


Figure 2: Empirical Hazard Rate of Adoption

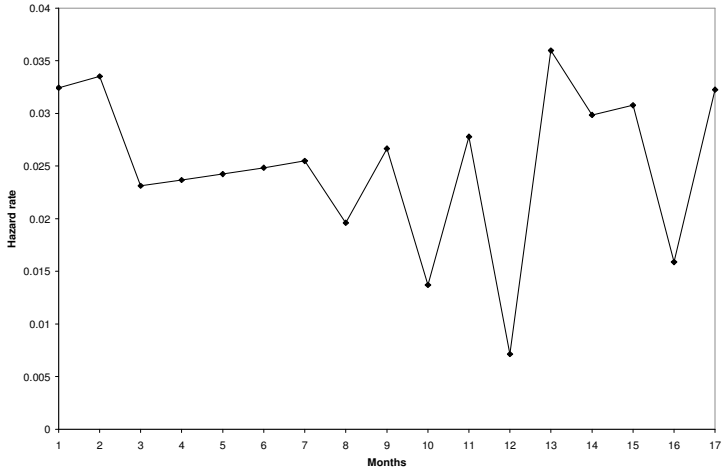


Figure 3: Average Detailing Effort per At-Risk Physician

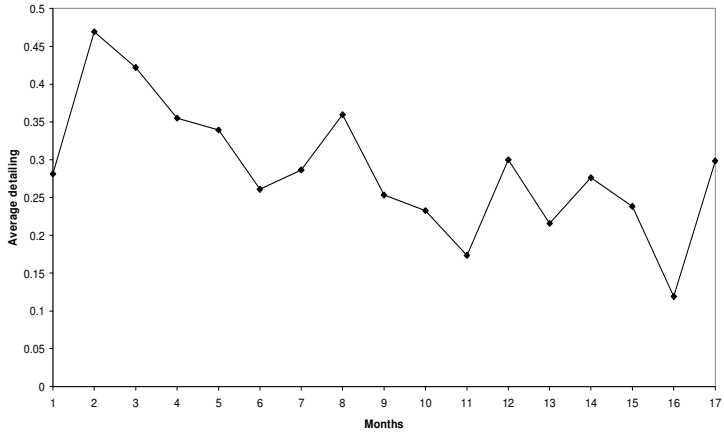


Table 3: Descriptive Statistics and Correlations among Time-invariant Covariates

Variable[a]	Mean	SD	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	.95	1.00													
5. Indegree - Total	0.95	3.42	.98	.99	1.00												
6. Outdegree - Referral	1.34	1.43	-.12	-.14	-.13	1.00											
7. Outdegree - Discussion	2.40	1.58	-.09	-.11	-.10	.41	1.00										
8. Outdegree - Total	3.74	2.54	-.13	-.15	-.14	.82	.86	1.00									
9. Self- reported Leadership	4.46	1.34	.29	.34	.32	-.21	.13	-.04	1.00								
10. LA Dummy	0.31	0.46	-.09	-.13	-.11	-.02	.00	.00	.09	1.00							
11. NYC Dummy	0.36	0.48	-.09	-.07	-.08	-.13	-.08	-.12	.02	-.49	1.00						
12. Solo Practice	0.38	0.49	-.09	-.14	-.12	.03	-.12	-.06	-.17	.00	.04	1.00					
13. Univ/Teaching Hospital	0.22	0.41	-.06	-.02	-.04	-.15	-.06	-.12	.12	-.09	.05	-.41	1.00				
14. Primary Care	0.11	0.32	-.08	-.09	-.09	.09	-.06	.01	-.27	.13	-.12	-.04	-.02	1.00			
15. Patients Managed	44.67	109.82	.26	.28	.28	-.02	-.09	-.07	.09	-.16	.21	.08	-.11	-.09	1.00		
16. Early Referral	0.3	0.46	-.12	-.17	-.15	.21	-.02	.11	-.48	-.16	.00	-.01	.08	.17	-.06	1.00	
17. Past Drug 1	21.36	47.11	.54	.62	.59	-.21	-.21	-.25	.37	-.11	.09	-.02	.00	-.13	.29	-.22	1.00
18. Past Drug 2	21.44	56.55	.57	.62	.60	-.12	-.12	-.14	.21	-.12	-.04	.03	-.05	-.09	.31	-.14	.71

[a] The numbers in front of the variables match those in Table 2. Min and Max values are the same as in Table 3.
Note: Values computed on a single observation for each physician, $N = 185$. All correlations equal or larger than 0.15 are significant at $p \leq .05$.

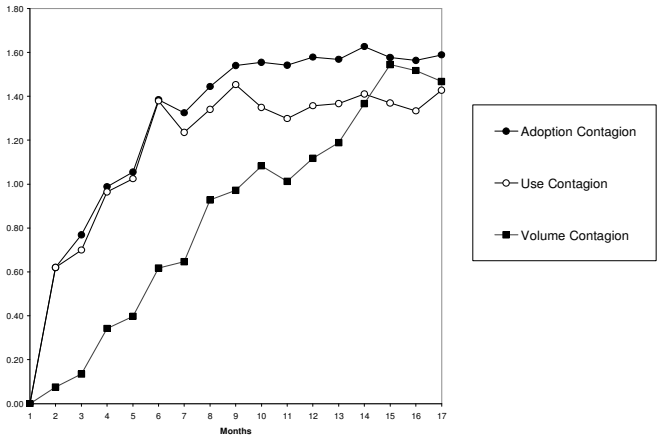
physicians who have not yet adopted (called physicians "at risk" of adopting) has a clear downward trend (Figure 3). That the marketing effort towards at-risk physicians decreases over time yet the empirical hazard rate does not suggests that, controlling for marketing effort, the hazard rate may actually 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 9 months and tend to level off afterwards.[7] Put simply, the exposure to both adopters and prescribers stopped growing after 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. But consider how Volume Contagion trends upwards throughout the entire 17-month observation period. If contagion based on peers' prescription volume is more

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

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.

Figure 4: Average Contagion Pressure per At-Risk Physician



Note: 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.

7.2. Respondents vs. non-respondents

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 ($p > .05$) from the 411 non-respondents on 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 twelve months prior to launch (21.4 vs. 15.6 for Drug 1; 21.4 vs. 20.4 for Drug 2) and sociometric leadership (total Indegree: 0.95 vs. 0.49). Nor were any of those variables associated significantly with the probability of responding after controlling for city in a multivariate test ($p > .05$). Hence, there is no evidence of response bias based on usage or sociometric status.

[8] Data on pre-launch prescriptions (Drug 1, Drug 2) are missing for 27 of the 438 non-respondents.

7.3. Validity of network measures

Non-response raises some special concerns in network studies since variables of interest are measured not only on the respondents included in the analysis but also on their connections to non-respondents. 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.

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 non-response.

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 downwards 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 indegrees 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, Killworth and Rennell (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. Since models using the total network tended to fit slightly better than models using only the referral or discussion network, and since using the total network follows the re-analyses 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 Technical Appendix).

8.1. Correlation results

The correlations reported in Table 2 between Self-reported Leadership and the various Indegree measures are all significantly positive ($p < .001$). 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 .32 ($p < .01$). Analysis by city shows that the correlations do not vary markedly with the response rate (SF: $r = .45$, response rate $\phi =$ 45%; LA: $r = .32$, $\phi = 29\%$; NYC: $r = .41$, $\phi = 24\%$). Hence, the low correlation between Indegree and Self-reported Leadership cannot be attributed to purported measurement error stemming from survey non-response.[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. Also, there 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, resp.) than with Self-reported Leadership ($r = 0.11$)

[9] The correlations between Indegree and Self-reported Leadership are much lower than the .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 (i) that 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) that both their measure of degree and leadership measured the *frequency of interaction* whereas our measure of indegree captures only the *number* of alters.

(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, while 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 only 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 counter-acting 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 ($p < .01$).[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, including Outdegree. Additional analyses indicate the absence of interaction effects of Outdegree with either Contagion or Detailing ($p > .05$). Hence, Outdegree and Indegree effects are quite

[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 and Use Contagion, the total effect of Self-reported Leadership never becomes significantly negative.

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

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

Note: The numbers in parentheses are the standard errors for the parameters. * indicates $p \leq 0.05$ and ** indicates $p \leq 0.01$. All models include 16 monthly time dummies and so have a flexible baseline hazard rate. The 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.

different. Detailing has a very significant effect that is robust across model specifications, and exhibits a carry-over 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 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 since 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. So, 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. Volume Contagion 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 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

[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 ($p < .05$) and only the interaction between Self-reported Leadership and Volume Contagion is significant in the model with interactions ($p < .05$). For details, see Technical Appendix, Table A-3.

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 Technical Appendix, 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 Technical Appendix indicate that the volume contagion effect is most likely to due to prior adopters' *credibility based on experience* with the focal drug, rather than to (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

Since both social contagion and detailing affect adoption, the question arises 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, though 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 and compare it against that in the base scenario where nothing is changed. Such a network-based intervention is of course not costless, but no cost data are available. So, as a benchmark, we compare the expected effectiveness of the intervention against that of an other intervention where each physician receives one additional detail in the first month. Using the model and taking into account the carry-over 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 (due to the partial carry-over), 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 8 months, the expected cumulative number of physicians who adopt due specifically to the network intervention exceeds that due specifically to the detailing

[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.

intervention. In the model with interactions, the cross-over happens after 12 months. Since 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 system-wide 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 than others to their contacts' behavior, whereas sociometric opinion leaders are not differentially responsive.

Since 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 be contingent on the nature of the product. As noted in sections 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. 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 to found their recommendations on. 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. Since 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 post-adoption outcomes (e.g., Haunschild and Miner 1997). Though our post-hoc analyses of contagion through share of prescription ("share of wallet") provide less support for this process in this specific study (see Technical Appendix), research on the role of post-adoption 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 arisen recently (Thompson 2008; Watts and Dodds 2007). Our evidence, note, 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, but 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, 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 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 towards 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 tend to be more influential within each of those connections. However, since 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.

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 system-wide 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.

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Online Technical Appendix

accompanying

"Opinion Leadership and Social Contagion in New Product Diffusion"

This Appendix reports on various checks documenting that our results are quite robust to variations in model specification.

Construction of network covariates

Table A-1 shows the results for some variants of Model 6 in Table 4, the one best supported by the data. The first column in Table A-1 just repeats the information of Model 6 in Table 4. The second column shows the results for the same model specification, but with Indegree, Outdegree and Contagion based only on referral ties. The third column shows the results for a model using only discussion ties. The fourth column is again based on the total network, but uses normalized social network weights w_{ij} such that $\Sigma_j w_{ij}$ equals unity (or zero for physicians nominating no peers at all), implying that physicians react to the proportion rather than the number of their peers who have adopted or are prescribing. The coefficients of these three alternative models are quite similar to those of the original model, and none fits better than the original. The one slight deviation worth noting is that, in the referral-only model, the coefficient of the interaction between self reported leadership and contagion is very similar to the one in the total network but its standard error is slightly bigger, which pushes the significance level slightly above 0.05.

Contemporaneous contagion

Since it is conceivable that contagion occurred within monthly periods, we also specified a model allowing for such simultaneous contagion. To this end, we used an instrumental variable approach which protects one's estimates from endogeneity bias (e.g., Anselin 1988; Land and Deane 1992; Manski 1993). We constructed the Volume Contagion variable as $\Sigma_j w_{ij} q_{jt}$ and regressed it on an intercept, dummies for LA and NYC, Volume Contagion at t-1, t-2 and t-3, and the network lagged detailing variable, i.e., the detailing to the nominees of the focal

physician at time t, t-1 and t-2 (Σ_j w_{ij} D_{jt-k}, for k = 0, 1 and 2). We then took the predicted values of this first-step regression (R^2 = 96%; all coefficients significant at $p < .01$), and used them as the instrumented values for contemporaneous Volume Contagion in the hazard model. As shown in column 5 of Table A-1, imposing simultaneous contagion while avoiding endogeneity bias leads to a slightly worse fit and does not change any of the coefficients or substantive conclusions.

Controlling for out-of-town contacts

By restricting the relevant networks to physicians practicing in specific zip code areas, our contagion variables do not encompass each and every colleague that the survey respondents nominated. Specifically, of all the people nominated by the survey respondents, we excluded 40 nominees in SF, 63 in LA and 80 in NYC. The excluded contacts received only one nomination on average, with 4 being the maximum, and accounted for 36% of all nominations. To the extent that our network definition is overly narrow, our contagion variables do not account for all the social influence experienced by the physicians whose adoptions we model. Since this may but need not affect our results, we checked that our results are robust to distinguishing between in-town and out-of-town contacts. Splitting the Outdegree into these two components did not improve model fit (Δ -2LL = 0.04) or affect the coefficients of substantive interest. Approximating Volume Contagion from out-of-town contacts by multiplying the number of out-of-town contacts by time (which is reasonable since Figure 4 shows that the in-town Volume Contagion variable increases linearly over time), and adding that new covariate to Model 6 did not significantly improve fit (Δ -2LL = 0.38) or affect the results either. Finally, allowing the effect of approximated out-of-town volume contagion to vary as a function of Indegree and Self-reported Leadership by adding the two relevant interaction terms did not improve model fit (Δ -

2LL = 2.54) or affect the results either. In short, our findings are robust to distinguishing between within-town and out-of-town contacts.

Excluding the flexible baseline hazard

Table A-2 shows the results for some additional variants of Model 6 in Table 4, the one best supported by the data. The first column in Table A-2 again shows the estimates of the latter model. The second column in Table A-2 pertains to the original model, but without time dummies. That model has a lower fit, but the loss in fit is small compared to the gain of 16 degrees of freedom (Δ -2LL = 10.40, $p > 0.05$). Unlike the original, the model without flexible baseline hazard exhibits a significant contagion effect for the average physician. Otherwise, the results are again very robust.

Unobserved heterogeneity and serial correlation

As is well known, unobserved heterogeneity induces spurious negative duration dependence in hazard models. This implies that it may create a downward bias in the contagion effect. Obviously, adding physician-specific fixed effects leads to biased estimates in a logit or probit hazard model of adoption or of any other non-repeated event (e.g., Chamberlain 1980). We therefore extended our model into a semi-parametric specification, featuring a flexible baseline hazard with monthly dummies and normally distributed random effects on the intercept.

We specified a hierarchical Bayes hazard model with a probit link function, and found that the convergence of the parameters within the Markov chain Monte Carlo (MCMC) routine was extremely poor, suggesting the model is overparameterized. When estimating the model without time dummies, we found that the estimated variance of the heterogeneity distribution tended to be determined entirely by the prior distribution of that variance, which indicates that there is no information in the data. Comparisons using Bayes Factors confirmed that unobserved

heterogeneity is not a concern. Similarly, empirical Bayes hazard models with a logit link function estimated using adaptive Gaussian quadrature in SAS NLMIXED led to variance estimates of 10^{-8}, the boundary value. All these results document the absence of detectable effects of unobserved heterogeneity. Given our rich set of covariates and since allowing for a flexible baseline hazard is known to make one's results robust to misspecification or even omission of the unobserved heterogeneity distribution, these results are not surprising (e.g., Butler, Baldwin and Johnson 2001; Dolton and van der Klaauw 1995 and 1999; Meyer 1990; Trussell and Richards 1985). In short, there is no evidence to reject the null hypothesis that our results are unaffected by unobserved heterogeneity.

We also checked for the presence of serial correlation within physicians. Extending Model 6 with an AR(1) structure, fixing the detailing carry-over rate to 0.44, and estimating the resulting model using the Generalized Estimating Equations method (GEE), led to an estimated AR(1) coefficient of 0.0015. Obviously, with such a small AR(1) value, the other coefficients and standard errors barely changed and all substantive conclusions were corroborated. Using the same procedure on the model without monthly dummies led to a similarly low serial correlation coefficient (-0.0004). All these results indicate the absence of serial correlation within physicians, which is consistent with the absence of unobserved heterogeneity.

Controls for possible time-varying endogeneity in detailing

It is conceivable that detailing decisions might have been revised after the launch of the product, making the time-invariant variables Past Drug 1 and Past Drug 2 ineffective controls for endogeneity in detailing and for omitted marketing effort variables. We therefore performed a robustness check using the 3-month moving total of the prescription volume for Drugs 1 and 2 (i.e., the sum of the volumes at *t*-1 through *t*-3) instead of their pre-launch total. As reported in

column 3 of Table A-2, this model fits slightly worse while all coefficients and statistical inferences remain unchanged.

Volume contagion after controlling for adoption and use contagion

As Table A-3 shows, extending models 3 and 6 in Table 4 with adoption contagion and use contagion do not change the main result about contagion being moderated by the influencers' prescription volume. Only volume contagion is significant in the main effects model ($p < .05$) and only the interaction between self-reported leadership and volume contagion is significant in the model with interactions ($p < .05$).

Fleshing out the volume contagion effect

That contagion is based on how much of the new drug one's network neighbors prescribe, rather than simply on whether they have adopted it or whether they wrote any prescription for it recently, could be driven by several processes. One possibility is that the volume matters because it is associated with the level of experience with the new drug, and experience in turn makes a colleague a more credible source of information and influence. However, there are alternative explanations as well.

One such alternative is that higher influence is associated with higher volume because the latter conveys enthusiasm about and commitment to the new drug. If this were the case, then one would expect that it is not the *number* of prescriptions written for the new drug but the *share* of the new drug in the source's overall category-level prescriptions that matters. To assess this alternative, we define $SOP_{jt\text{-}1}$ as the focal drug's share of prescriptions by network neighbor j at t-1, and compute it as the number of j's prescriptions for the focal drug at time t-1 divided by the number of j's prescriptions for all three drugs in the category at time t-1. We then define the *SOP*-based contagion variable as $\Sigma_j \, w_{ij} \, SOP_{jt\text{-}1}$. (Note, there is no point in multiplying *SOP* by

either y_{jt-1} or s_{jt-1} since $SOP_{jt-1} > 0$ requires $y_{jt-1} = 1$ and $s_{jt-1} = 1$.) Replacing the volume contagion variable with this SOP contagion variable in Model 6 in Table 4, and re-estimating the model leads to very similar coefficients (see Model 1 in Table A-4), including a significant negative interaction with self-reported opinion leadership, but a worse model fit. The difference in deviance (Δ -2LL = Δ BIC = 2.62) is large enough to favor the original volume contagion model (Raftery 1995).

Another possibility is that high prescribers of the new drug tend to be opinion leaders who not only have more network ties, but—being recognized experts—also exert more influence within each of these ties. If volume matters because it confers not experience with the new drug but expert status in general, then one would expect that it is not the *number* of prescriptions written for the new drug but the sociometric leadership of the prescribing peer that matters. To assess this alternative, we define indegree-weighted versions of the adoption and use contagion variables: $\Sigma_j \, w_{ij} \, K_j \, y_{jt-1}$.and $\Sigma_j \, w_{ij} \, K_j \, s_{jt-1}$, resp., where K is the *Indegree*. (Note, we do not create such variables for self-reported leadership, since the latter is measured only for survey respondents). Replacing the volume contagion variables with these sociometric leadership-weighted contagion variables leads to much worse model fit (Δ -2LL = Δ BIC = 8.54 and 7.58, resp.; see Models 2 and 3 in Table A-4) with the difference in deviance being large enough to markedly favor the volume contagion model (Raftery 1995).

Yet another possibility is that what matters is not experience with the new drug specifically, but more *general category-level experience*. To assess this alternative, we define two variants of general experience -weighted adoption and use contagion variables. In the first, we use the number of prescriptions written for the other two drugs in the category during the twelve months prior to the launch of the focal drug, and call the resulting variables "prelaunch weighted"

contagion (Models 4 and 5 in Table A-4). In the second variant, we use the number of prescriptions written in the previous month for all three drugs in the category as the weight, and call the resulting variables "category weighted" contagion (Models 6 and 7 in Table A-4). Replacing the volume contagion variables with these general experience-weighted contagion variables leads to much worse model fit (Δ -2LL = Δ BIC equals 6.80 or more) with the difference in deviance being large enough to markedly favor the volume contagion model in all four cases (Raftery 1995).

It is also conceivable that the volume contagion effect actually stems from "*detailing leakage*," where the amplification of the influence within ties stems from more detailing efforts being targeted towards heavy prescribers. We therefore also estimated models with detailing-weighted adoption and use contagion variables as Σ_j w_{ij} D_{jt-1} y_{jt-1} and Σ_j w_{ij} D_{jt-1} s_{jt-1}, respectively, where D_{jt-1} is the amount of detailing physician j received in the prior month. As columns 8 and 9 in Table A-4 report, these alternative models fit worse than the volume contagion model (Δ -2LL = Δ BIC = 5.28 and 3.82, respectively) and the detailing-weighted variables have no significant main effect or interaction effect with opinion leadership ($p > .10$). Hence, the original interpretation of genuine volume contagion seems best supported by the data.[13]

Finally, we also entertain the possibility that part of the volume contagion process operates through a *back-and-forth flow of patients* between physicians, where (i) i refers patients to j, (ii) j treats these patients and prescribes the new drug to some of those patients, (iii) some of those patients flow back to i, and (iv) i adopts so as not to change the regimen of those patients. While this contagion process through patient referral is conceivable, step (iii) from this sequence of

[13] We also assessed the possibility that not only the volume effect but the entire contagion effect is an artifact due to detailing leakage. Using the variable Σ_j w_{ij} D_{jt-1} instead of the volume contagion variable Σ_j w_{ij} q_{jt-1} leads to a model that fits markedly worse (Δ -2LL = Δ BIC = 6.22) and where detailing leakage has no significant main effect or interaction effect with self-reported leadership ($p > .10$).

events is quite unlikely in our data where both the discussion and referral ties in our data are extremely asymmetric. One implication, however, is testable, since the process implies that the practice size of *j* would influence his or her influence through referral ties. Because of the nature of the treatment, the usage rate does not vary within patients being treated, and a physician's category-level prescription volume is likely to be a good proxy for the number of patients treated. We therefore control for practice size in terms of category-level prescription. With $CQ_{jt\text{-}1}$ being the total category-level amount of prescriptions (for all three drugs) written by physician *j* in period *t*-1, and the network weight w_{ij} now being limited to referral ties only, we constructed yet other variants of the adoption and use contagion variables as (a) $\Sigma_j w_{ij} CQ_{jt\text{-}1} y_{jt\text{-}1}$ and (b) $\Sigma_j w_{ij} CQ_{jt\text{-}1} s_{jt\text{-}1}$. As columns 10 and 11 in Table A-4 report, these alternative models fit markedly worse than the volume contagion model (Δ -2LL = Δ BIC = 8.11 and 7.96, respectively) and the detailing-weighted variables have no significant main effect or interaction effect with opinion leadership ($p > .10$). Hence, the original interpretation of genuine volume contagion seems best supported by the data.

In short, the volume effect is most likely to due to *credibility based on experience* with the focal drug, rather than enthusiasm about the focal drug, expert status, category-level experience, amplification through detailing leakage, or back-and-forth flow from patients.

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Table A-1: Robustness Checks for Network Weights and Simultaneous Contagion

	Total Netw. (1)	Referral Netw. (2)	Discuss. Netw. (3)	Total Netw. Normalized (4)	Total Netw. Simultaneity (5)
Intercept	-3.88**	-3.70**	-3.88**	-3.72**	-3.94**
	(0.74)	(0.70)	(0.74)	(0.73)	(0.75)
Indegree	0.30*	0.51	0.52*	0.32*	0.29*
	(0.15)	(0.34)	(0.25)	(0.15)	(0.15)
Outdegree	0.08	0.12	0.10	0.10	0.08
	(0.06)	(0.12)	(0.08)	(0.06)	(0.06)
Self-rep. Leadership	0.42*	0.36*	0.44**	0.39*	0.43*
	(0.18)	(0.18)	(0.19)	(0.18)	(0.18)
Contagion - Volume	0.01	0.01	0.01	0.04	0.01
	(0.007)	(0.02)	(0.01)	(0.03)	(0.006)
Detailing Stock	0.41**	0.38**	0.41**	0.41**	0.40**
	(0.14)	(0.14)	(0.13)	(0.14)	(0.14)
Carry Over Effect	0.44*	0.47*	0.42*	0.42*	0.44*
	(0.20)	(0.24)	(0.19)	(0.21)	(0.21)
Indegree × Contagion	0.001	-0.02	0.002	-0.003	0.002
	(0.005)	(0.03)	(0.01)	(0.03)	(0.004)
Indegree × Detailing Stock	-0.05	-0.02	-0.11	-0.05	-0.05
	(0.04)	(0.09)	(0.07)	(0.04)	(0.04)
Self-rep. Leadership × Contagion	-0.01*	-0.01	-0.03**	-0.05*	-0.01*
	(0.005)	(0.008)	(0.01)	(0.02)	(0.005)
Self-rep. Leadership × Detailing Stock	-0.05	-0.06	-0.03	-0.05	-0.05
	(0.07)	(0.07)	(0.08)	(0.07)	(0.07)
LA Dummy	0.09	-0.01	0.09	0.02	0.11
	(0.42)	(0.41)	(0.42)	(0.42)	(0.45)
NYC Dummy	-0.27	-0.33	-0.31	-0.23	-0.25
	(0.43)	(0.42)	(0.43)	(0.44)	(0.44)
Solo Practice	0.01	-0.01	-0.01	-0.11	0.04
	(0.35)	(0.35)	(0.35)	(0.36)	(0.35)
University / Teaching Hospital	0.69	0.64	0.54	0.63	0.72
	(0.41)	(0.42)	(0.40)	(0.41)	(0.41)
Primary Care	-0.57	-0.48	-0.65	-0.48	-0.54
	(0.77)	(0.77)	(0.78)	(0.76)	(0.77)
Early Referral	-0.77	-0.86	-0.52	-0.73	-0.78
	(0.44)	(0.47)	(0.42)	(0.44)	(0.44)
Patients Managed	0.001	0.001	0.001	-0.001	0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Past Drug 1	0.002	0.004	0.002	0.003	0.002
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Past Drug 2	0.01**	0.01**	0.01**	0.01**	0.01**
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
LL	-225.48	-227.59	-225.99	-226.02	-225.76

Note: The numbers in parentheses are the standard errors for the parameters. * indicates $p \leq 0.05$ and ** indicates $p \leq 0.01$. All Models 1-5 include 16 monthly time dummies and so have a flexible baseline hazard rate.

Table A-2: Robustness Checks for Baseline Hazard and Time-varying Endogeneity

Variables	Total Netw. (1)	Total Netw. No Time Dummies (2)	Total Netw. Past Drugs as Moving Total (3)
Intercept	-3.88**	-4.62**	-3.96**
	(0.78)	(0.45)	(0.75)
Indegree	0.30*	0.22	0.31*
	(0.15)	(0.12)	(0.14)
Outdegree	0.08	0.06	0.07
	(0.06)	(0.05)	(0.05)
Self-rep. Leadership	0.42*	0.39*	0.44*
	(0.18)	(0.18)	(0.19)
Contagion	0.01	0.01*	0.01
	(0.007)	(0.007)	(0.007)
Detailing Stock	0.41**	0.37**	0.42**
	(0.14)	(0.12)	(0.13)
Carry Over Effect	0.44*	0.53**	0.48*
	(0.20)	(0.18)	(0.18)
Indegree × Contagion	0.001	0.003	0.001
	(0.005)	(0.005)	(0.005)
Indegree × Detailing Stock	-0.05	-0.03	-0.05
	(0.04)	(0.03)	(0.04)
Self-rep. Leadership × Contagion	-0.01*	-0.01**	-0.01*
	(0.005)	(0.005)	(0.005)
Self-rep. Leadership × Detailing Stock	-0.05	-0.04	-0.03
	(0.07)	(0.06)	(0.07)
LA Dummy	0.09	0.18	0.07
	(0.42)	(0.43)	(0.44)
NYC Dummy	-0.27	-0.15	-0.19
	(0.43)	(0.43)	(0.42)
Solo Practice	0.01	0.01	-0.07
	(0.35)	(0.34)	(0.35)
University / Teaching Hospital	0.69	0.74	0.67
	(0.41)	(0.41)	(0.41)
Primary Care	-0.57	-0.52	-0.56
	(0.77)	(0.77)	(0.77)
Early Referral	-0.77	-0.75	-0.72
	(0.44)	(0.44)	(0.45)
Patients Managed	0.001	0.001	0.001
	(0.001)	(0.001)	(0.001)
Past Drug 1	0.002	0.001	-0.001
	(0.004)	(0.004)	(0.013)
Past Drug 2	0.01**	0.01*	0.05**
	(0.004)	(0.004)	(0.01)
LL	-225.48	-230.68	-226.75

Note: The numbers in parentheses are the standard errors for the parameters. * indicates $p \leq 0.05$ and ** indicates $p \leq 0.01$. Models 1 and 3 include 16 monthly time dummies and so have a flexible baseline hazard rate. Model 2 does not.

Table A-3: Contagion through Adoption, Use and Volume

Variables	Main Effects	Interaction Effects
Intercept	-3.65**	-3.55**
	(0.71)	(0.76)
Indegree	0.14*	0.31*
	(0.07)	(0.15)
Outdegree	0.13	0.13
	(0.07)	(0.07)
Self-rep. Leadership	0.17	0.31
	(0.15)	(0.20)
Contagion, Adoption	-0.19	-0.12
	(0.28)	(0.40)
Contagion, Use	-0.02	-0.08
	(0.31)	(0.43)
Contagion, Volume	0.02*	0.02
	(0.009)	(0.009)
Detailing Stock	0.39**	0.43**
	(0.13)	(0.14)
Carry Over Effect	0.46*	0.45*
	(0.22)	(0.19)
Indegree × Contagion, Adoption		0.19
		(0.35)
Indegree × Contagion, Use		-0.21
		(0.36)
Indegree × Contagion, Volume		0.003
		(0.007)
Indegree × Detailing Stock		-0.05
		(0.04)
Self-rep. Leadership × Contagion, Adoption		-0.04
		(0.29)
Self-rep. Leadership × Contagion, Use		0.14
		(0.33)
Self-rep. Leadership × Contagion, Volume		-0.02*
		(0.008)
Self-rep. Leadership × Detailing Stock		-0.03
		(0.07)
LA Dummy	0.18	-0.09
	(0.41)	(0.36)
NYC Dummy	-0.36	-0.37
	(0.43)	(0.44)
Solo Practice	0.04	-0.09
	(0.35)	(0.36)
University / Teaching Hospital	0.72	0.73
	(0.41)	(0.42)
Primary Care	-0.61	-0.61
	(0.76)	(0.78)
Early Referral	-0.76	-0.89
	(0.43)	(0.46)
Patients Managed	0.0004	0.0002
	(0.001)	(0.001)
Past Drug 1	0.004	0.003
	(0.004)	(0.004)
Past Drug 2	0.01*	0.01*
	(0.004)	(0.005)
LL	-227.93	-223.79

Note: The numbers in parentheses are the standard errors for the parameters. * indicates $p \leq 0.05$ and ** indicates $p \leq 0.01$. Both models include 16 monthly time dummies and so have a flexible baseline hazard rate.

Table A-4: Alternatives to the Volume Contagion Model

Variable	Share of Prescriptions (1)	Indegree Weighted Adoption (2)	Indegree Weighted Use (3)	Prelaunch Categ. Vol. Weighted Adoption (4)	Prelaunch Categ. Vol. Weighted Use (5)
Intercept	-3.89**	-3.25**	-3.32**	-3.31**	-3.36**
	(0.73)	(0.66)	(0.66)	(0.68)	(0.69)
Indegree	0.28	0.35*	0.37*	0.31*	0.31*
	(0.15)	(0.16)	(0.16)	(0.14)	(0.14)
Outdegree	0.07	0.10	0.09	0.11	0.09
	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)
Self-rep. Leadership	0.44*	0.18	0.16	0.37*	0.36
	(0.19)	(0.19)	(0.19)	(0.19)	(0.19)
Contagion	0.52	-0.00	-0.00	0.08	0.56
	(0.29)	(0.001)	(0.001)	(2.19)	(2.29)
Detailing Stock	0.39**	0.38**	0.37**	0.37**	0.37**
	(0.13)	(0.13)	(0.13)	(0.13)	(0.13)
Carry Over Effect	0.45*	0.41	0.42*	0.45*	0.45*
	(0.21)	(0.21)	(0.21)	(0.20)	(0.20)
Indegree × Contagion	0.11	-0.0001	-0.0002	0.59	0.53
	(0.18)	(0.0005)	(0.0005)	(1.08)	(1.09)
Indegree × Detailing Stock	-0.04	-0.06	-0.06	-0.06	-0.02
	(0.04)	(0.05)	(0.05)	(0.04)	(0.07)
Self-rep. Leadership × Contagion	-0.52*	0.0003	0.0004	-2.79	-2.67
	(0.24)	(0.0007)	(0.0007)	(1.84)	(1.87)
Self-rep. Leadership × Detailing Stock	-0.05	-0.01	-0.01	-0.02	-0.02
	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
LA Dummy	0.03	-0.15	-0.12	-0.22	-0.19
	(0.40)	(0.38)	(0.38)	(0.41)	(0.42)
NYC Dummy	-0.38	-0.49	-0.47	-0.58	-0.55
	(0.41)	(0.39)	(0.39)	(0.42)	(0.42)
Solo Practice	0.11	0.07	0.06	-0.02	-0.01
	(0.35)	(0.35)	(0.35)	(0.35)	(0.35)
University / Teaching Hospital	0.65	0.64	0.63	0.55	0.57
	(0.41)	(0.40)	(0.40)	(0.41)	(0.41)
Primary Care	-0.41	-0.55	-0.57	-0.64	-0.63
	(0.77)	(0.77)	(0.76)	(0.77)	(0.77)
Early Referral	-0.80	-0.69	-0.67	-0.71	-0.71
	(0.45)	(0.44)	(0.43)	(0.43)	(0.43)
Patients Managed	0.0008	0.0003	0.0002	0.0001	0.0001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Past Drug 1	0.003	0.004	0.004	0.003	0.003
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Past Drug 2	0.01*	0.01*	0.01*	0.01**	0.01*
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
LL	-226.79	-229.75	-229.27	-228.88	-228.91

Note: The numbers in parentheses are the standard errors for the parameters. * indicates $p \leq 0.05$ and ** indicates $p \leq 0.01$. All models include 16 monthly time dummies and so have a flexible baseline hazard rate.

Table A-4 (continued): Alternatives to the Volume Contagion Model

Variables	Current Categ. Vol. Weighted Adoption (6)	Current Categ. Vol Weighted Use (7)	Detailing Weighted Adoption (8)	Detailing Weighted Use (9)	Referral Volume Weighted Adoption (10)	Referral Volume Weighted Use (11)
Intercept	-3.32**	-3.37**	-3.43**	-3.54**	-3.36**	-3.39**
	(0.66)	(0.69)	(0.67)	(0.67)	(0.67)	(0.68)
Indegree	0.31*	0.31*	0.25	0.24	0.32*	0.32*
	(0.15)	(0.14)	(0.14)	(0.14)	(0.14)	(0.14)
Outdegree	0.10	0.09	0.09	0.07	0.09	0.09
	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)
Self-rep. Leadership	0.33	0.33	0.33	0.37*	0.28	0.28
	(0.18)	(0.18)	(0.18)	(0.18)	(0.17)	(0.17)
Contagion	0.00	0.00	0.03	0.06	0.00	0.00
	(0.002)	(0.002)	(0.03)	(0.04)	(0.004)	(0.004)
Detailing Stock	0.37**	0.37**	0.37**	0.36**	0.36**	0.36**
	(0.13)	(0.13)	(0.13)	(0.14)	(0.13)	(0.13)
Carry Over Effect	0.45*	0.45*	0.48*	0.47*	0.44*	0.44*
	(0.20)	(0.21)	(0.21)	(0.21)	(0.21)	(0.21)
Indegree × Contagion	0.0006	0.0005	0.04	0.05	0.001	0.001
	(0.001)	(0.001)	(0.02)	(0.03)	(0.002)	(0.002)
Indegree × Detailing Stock	-0.06	-0.06	-0.05	-0.04	-0.06	-0.06
	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)	(0.04)
Self-rep. Leadership × Contagion	-0.002	-0.002	-0.03	-0.04	-0.002	-0.002
	(0.002)	(0.002)	(0.02)	(0.03)	(0.003)	(0.003)
Self-rep. Leadership × Det. Stock	-0.02	-0.02	-0.02	-0.03	-0.02	-0.02
	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
LA Dummy	-0.21	-0.18	-0.16	-0.09	-0.15	-0.13
	(0.41)	(0.42)	(0.39)	(0.39)	(0.39)	(0.40)
NYC Dummy	-0.58	-0.56	-0.56	-0.51	-0.52	-0.51
	(0.42)	(0.42)	(0.42)	(0.42)	(0.41)	(0.41)
Solo Practice	0.003	0.006	0.06	0.07	0.02	0.01
	(0.35)	(0.36)	(0.35)	(0.35)	(0.35)	(0.35)
University / Teaching Hospital	0.58	0.59	0.61	0.61	0.62	0.62
	(0.41)	(0.41)	(0.41)	(0.41)	(0.41)	(0.41)
Primary Care	-0.65	-0.64	-0.68	-0.71	-0.63	-0.61
	(0.77)	(0.77)	(0.77)	(0.77)	(0.77)	(0.77)
Early Referral	-0.71	-0.70	-0.67	-0.66	-0.74	-0.75
	(0.43)	(0.43)	(0.43)	(0.43)	(0.44)	(0.44)
Patients Managed	0.0001	0.0001	0.0004	0.0003	0.0001	-0.0001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Past Drug 1	0.004	0.004	0.004	0.004	0.004	0.004
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Past Drug 2	0.01*	0.01*	0.01*	0.01*	0.01*	0.01*
	(0.004)	(0.005)	(0.004)	(0.004)	(0.004)	(0.005)
LL	-229.26	-229.26	-228.12	-227.39	-229.53	-229.46

Note: The numbers in parentheses are the standard errors for the parameters. * indicates $p \leq 0.05$ and ** indicates $p \leq 0.01$. All models include 16 monthly time dummies and so have a flexible baseline hazard rate.