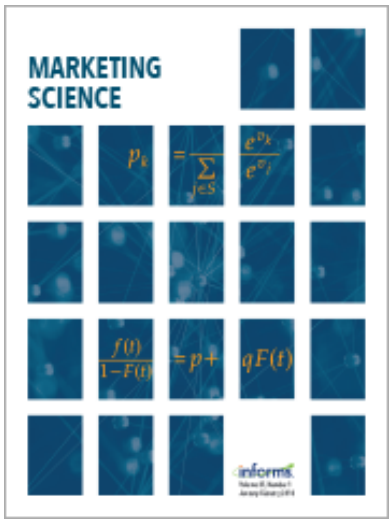

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Social Contagion in New Product Trial and Repeat

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The notion of peer influence in new product adoption or trial is well accepted. We propose that peer influence may affect repeat behavior as well, though the process and source of influence are likely to differ between trial and repeat. Our analysis of the acceptance of a risky prescription drug by physicians provides three novel findings. First, there is evidence of contagion not only in trial but also in repeat. Second, who is most influential varies across stages. Physicians with high centrality in the discussion and referral network and with high prescription volume are influential in trial but not repeat. In contrast, immediate colleagues, few of whom are nominated as a discussion or referral partner, are influential in both trial and repeat. Third, who is most influenceable also varies across stages. For trial, it is physicians who do not consider themselves to be opinion leaders, whereas for repeat, it is those located towards the middle of the status distribution as measured by network centrality. The pattern of results is consistent with informational social influence reducing risk in trial and normative social influence increasing conformity in repeat.

Keywords: new product diffusion; social contagion; social networks; social status; trial-repeat
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1. Introduction

How new products gain market acceptance is of key interest to marketers. The notion that adoption or trial can be affected by peer influence or social contagion is well accepted. Having customers try a new product, however, does not mean that they will keep using it and that the product will gain market acceptance. Marketers seek not only trial but also sustained use or repeat purchases. Research on how social contagion helps new products gain market traction, however, focuses almost exclusively on adoption or trial.

Thus, several important questions remain unanswered. Can social contagion affect not only trial but also repeat behavior? If so, are those who influence others to adopt the same as those who influence others to repeat? That is, are the same customers influential in both trial and repeat, or should marketers seek to leverage different customers to support trial versus repeat? What about differences in susceptibility to social influence? That is, are those who are the most influenceable at trial also the most influenceable at the repeat stage? Finally, if contagion operates differently at each stage, can we gain some insights about why this happens?

The presence of social contagion in repeat may appear a bit puzzling. Why would adopters' subsequent behavior be affected by peers, since adoption provides the opportunity to learn directly about the product's advantages and disadvantages? Theory and empirical evidence suggest four reasons. First, social contagion can result from both informational and normative peer influence (e.g., Deutsch and Gerard 1955). Whereas one expects informational influence to decline as customers proceed from trial to repeat, theory and empirical research provide no basis for normative influence to decline; some work even implies the opposite (e.g., Tolbert and Zucker 1983). Second, informational influence need not be limited to trial but may affect repeat as well. When learning about product quality from personal experience is slow, customers may rely on peers as a source of information for not only trial but also repeat decisions (e.g., Dulleck and Kershbamer 2006). Third, for products and services where interconnectivity or standardization is important, the utility of use increases with the number of other users, such that contagion affects not only adoption but also repeat or churn (e.g., Haenlein 2013, Nitzan and Libai 2011). Fourth, environmental shocks can raise new doubts about an accepted product, making repeat users again susceptible to informational influence from peers, as suggested by Nair et al. (2010).

Investigating social contagion in trial versus repeat can provide new insights that are theoretically and managerially valuable. Three benefits stand out.

First, who the influentials are, who the influenceables are, and how that varies across trial and repeat matters to marketers keen on leveraging social contagion to help their products gain market acceptance. Who should they seek for leverage at trial versus repeat? Who can they afford not to target with costly resources, and does that change from trial to repeat?

Second, research focusing exclusively on trial provides only limited insights into what drives new product acceptance. This is especially true for three types of products: (1) For consumables and services where trial purchases account for only a fraction of customer lifetime value and overall product profitability, managers need to know what drives trial as well as repeat (Gielens and Steenkamp 2007, Shih and Venkatesh 2004); (2) For credence goods and complex innovations that generate uncertainty or ambiguity for their users even after trial, managers need to understand how these post-adoption sentiments operate so they can prevent them from becoming hurdles to repeat (Wood and Moreau 2006); (3) For products and technologies targeted towards professionals and business users, managers need to understand how intraorganizational factors affect the sustained implementation of innovations (Downs and Mohr 1976).

Third, similarities or differences in who is most influential and influenceable at trial versus repeat may provide insights into the nature of the contagion mechanism(s) at work. This is a key research priority (e.g., Aral 2011, Godes 2011, Iyengar et al. 2011b, Lewis et al. 2012, Libai et al. 2010). Recent work has documented systematic variations across customers in influence and susceptibility, but, to our knowledge, only in the realm of new product adoption (e.g., Aral and Walker 2012, Goldenberg et al. 2009, Hu and Van den Bulte 2014, Iyengar et al. 2011a, Katona et al. 2011) or outside the realm of new products altogether (e.g., Godes and Mayzlin 2009, Trusov et al. 2010). Studying social contagion in both trial and repeat facilitates assessment of the effect of peer behavior on two different dependent variables. This in turn enables one to more sharply identify the nature of the contagion mechanism(s) at work (Oster and Thornton 2012).

We investigate the presence and nature of contagion in trial versus repeat by studying the acceptance of a new prescription drug by physicians. Our study combines individual-level trial and repeat data, social network data, survey data, and individual-level sales call data.

There are three novel findings. First, we find evidence of contagion in both trial and repeat. Second, who is most influential varies across stages. Physicians who are central in the network of discussion and referral and who prescribe the new drug heavily drive the contagion at the trial stage (as found in an earlier analysis of the same drug), but they do not drive contagion at the repeat stage. Instead, repeat prescriptions are affected by the behavior of immediate colleagues, only some of whom are also discussion/referral partners. Third, who is most influenceable also varies across stages. For trial, it is physicians who do not see themselves as opinion leaders (consistent with prior analysis). For repeat, in contrast, it is physicians in the middle of the status distribution as measured by network centrality.

Observing contagion operate in very different ways across trial and repeat suggests that different mechanisms are at work at each stage. Specifically, the moderator effects in each stage as well as the contrast across stages are consistent with informational influence reducing risk in trial and normative influence increasing conformity in repeat. Hence, this study answers recent calls to move research from whether contagion is at work to how and why it is at work (Aral 2011, Godes 2011). In addition, our evidence of a nonmonotonic status effect extends recent insights into how status considerations affect customer behavior (Hu and Van den Bulte 2014).

Our findings are also relevant to managers. Marketers should consider leveraging peer influence not only to trigger adoption but also to support subsequent repeat, at least for risky products such as the one studied here. Also, marketing policies to leverage contagion should be designed and targeted differently, since those who are most influential and those who are most influenceable vary across stages. Finally, the results suggest that marketers of products such as the one we study may want to emphasize different motivations, e.g., perceived risk versus conformity to local norms, in their sales calls and other marketing communications targeted towards prospects versus adopters.

We proceed first by further developing the research questions, building on theories and findings from psychology and sociology. We next describe the research setting, data, and modeling approach. We then present the findings and discuss their implications for theory, research, and practice.

2. Research Questions

Though social contagion and trial-repeat behavior have long been the object of active research, and though studying them jointly would provide three important benefits, to our knowledge there is virtually no research of this kind to build on. So, we rely mostly on theoretical arguments to develop our research questions.

We first very briefly describe marketing research on trial versus repeat. We then discuss informational and normative influence as two distinct contagion mechanisms. This provides the basis for refutable hypotheses on how and why contagion operates differently in trial versus repeat.

2.1. Prior Research on Trial versus Repeat

To our knowledge, prior research on social contagion focuses only on adoption or does not discriminate between trial and repeat. Similarly, to the extent that new product research has studied repeat behavior, it has done so without considering contagion.

Modeling trial-repeat behavior has a long history in marketing (e.g., Parfitt and Collins 1968). However, such work is typically conducted in packaged goods categories for which social contagion was until recently believed to be relatively unimportant, even at the trial stage, because of low functional, financial, and social risk (Du and Kamakura 2011). As a result, empirical studies of this kind have not provided insights into contagion dynamics.

Aggregate-level diffusion modeling also has a long history. Several studies of this kind distinguish between trial and repeat sales, but do not investigate contagion in each stage (e.g., Hahn et al. 1994).

Diffusion researchers have also investigated whether initial deployment or trial of new technologies by organizations is driven by different factors than subsequent deployment within those organizations. However, studies contrasting "inter" and "intra" firm diffusion do not investigate social contagion dynamics (e.g., Levin et al. 1992).

2.2. Informational versus Normative Influence

Peer influence leading up to social contagion in customer behavior can be informational and normative (e.g., Bearden et al. 1989; Deutsch and Gerard 1955; Turner 1991, pp. 34–39). Informational influence occurs when information obtained from peers serves as evidence about reality and so changes one's beliefs about the true state of the world. Normative influence arises from the desire to conform to the expectations of others about what is the right and proper thing to do.

The notion of social contagion through informational influence, affecting awareness or beliefs about products' risks and benefits, is quite familiar to marketing scientists. The notion of contagion through normative influence is less so, and two important characteristics should be kept in mind.

First, normative influence is fundamentally a group phenomenon (Deutsch and Gerard 1955; Hogg 2010; Turner 1991, p. 37). Social norms are rules and standards that are understood, endorsed, and expected by members of a group (Cialdini and Trost 1998). Consequently, conformity to norms is fundamentally a group, rather than interpersonal, process.

Second, normative influence can be of two types (Bearden et al. 1989; Kelman 1958, 2006; Scott 1996, p. 96; Turner 1991, pp. 39, 117–118): compliance based on others' power to mediate rewards and costs, and identification based on the desire to live up to others' expectations of one's role. Whereas compliance requires public observability and monitoring so that people can be rewarded or punished depending on whether they act in accordance to the norm, identification does *not*. Instead, it requires that people care about maintaining a positive relationship with other members of their group (Kelman 1958; Turner 1991, p. 117). Whereas compliance operates primarily through reward and coercive power, identification operates primarily through referent power (Warren 1968).[1] Whereas compliance is about adhering to rules, identification is about enacting roles based on others' expectations (Kelman 2006).

2.3. Informational Influence in Trial versus Repeat

Trial of new products, especially those presenting substantial risk, can be subject to social contagion through informational peer influence. Evidence that contagion increases with the sources' credibility, experience, or expertise and that it decreases with the decision makers' self-confidence in their judgment indicates that contagion stems from informational influence (e.g., Deutsch and Gerard 1955, Kelman 1958, Iyengar et al. 2011a).

Informational influence is less likely to affect repeat decisions, as personal consumption experience substitutes for input from peers. Hence, a contagion effect that is larger in trial than repeat would be quite consistent with informational influence. Yet some peer influence may be at work in repeat when learning from experience is slow. For instance, whereas physicians can quickly learn about the effectiveness of drugs used to treat acute conditions with easy to observe symptoms, this is not so for drugs used for chronic conditions that are hard to monitor. Learning from personal experience can be slow even for such simple products and services as laundry detergents and mobile phone service (e.g., Iyengar et al. 2007). Hence, for risky products with slow experiential learning, some customers may rely on the judgment of peers even when making repeat decisions (Dulleck and Kershbamer 2006).

In short, for risky new products, informational influence considerations lead one to expect that social

[1] Consequently, both theory (e.g., Bicchieri 2006, pp. 11, 42–44) and empirical research (e.g., Cialdini et al. 1990) imply that the actions of any *specific* people need not be observed for norms to operate. Actions of specific influencers need not be observed because people can form normative expectations, i.e., beliefs about what others expect them to do, without directly observing the actions of any specific person. For example, people can infer from the presence of litter on the ground that littering is socially acceptable even if they do not see any specific person littering (Cialdini et al. 1990). Actions of influencees need not be observed either. Though normative influence through compliance involving rewards and punishment requires that others can observe one's actions, public observability is not required for normative influence through identification, as the latter involves only one's own assessment of how well one meets others' expectations.

contagion (i) is at work in trial, (ii) originates from trusted peers, (iii) is lower for people confident in their judgments, and (iv) operates with greater strength in trial than repeat. As a corollary, a contagion effect with characteristics (i)–(iv) is more likely to stem from informational influence than an effect without these characteristics.

2.4. Normative Influence in Trial versus Repeat

The acceptance of innovations can be subject to social contagion through normative influence (e.g., DiMaggio and Powell 1983, Rossman 2014, Van den Bulte and Stremersch 2004). Because norms are endorsed and expected by members of a group, customers are more likely to experience normative influence from group members than from outsiders, even those with experience or expertise (Deutsch and Gerard 1955; Turner 1991, pp. 117–118). This suggests that informational and normative influence may stem from different sources (e.g., experts versus family members or colleagues).

The extent to which customers conform to social norms is likely to vary by status, i.e., social rank in terms of esteem and respect. Customers with low status have little to lose from not conforming and little to gain from conforming. Whether they conform simply does not affect them very much (Dittes and Kelley 1956, Harvey and Consalvi 1960). The same holds for customers with the highest status. They gain little additional esteem from adhering to group norms and are given greater latitude than others to deviate from group norms (Hollander 1958). Consequently, it is customers in the middle of the status distribution who have the greatest tendency to conform to norms, a pattern referred to as middle-status conformity and documented in adoption studies by Phillips and Zuckerman (2001) and Hu and Van den Bulte (2014). Along similar lines, Bosk (2003, p. 75) describes how physicians of middle status experience the most pressure to adhere to their surgical ward's local norms.

In contrast to informational influence, there is little theory or empirical research suggesting that the susceptibility to normative influence declines as customers proceed from trial to repeat. Rather, the opposite is likely. The first reason is that adopters' desire to appear legitimate by conforming to normative expectation increases over the diffusion process, as several studies suggest. Whereas early adoptions are affected primarily by technical and performance considerations, the evidence suggests, later behavior is increasingly affected by the need to appear legitimate (Kennedy and Fiss 2009, Tolbert and Zucker 1983, Westphal et al. 1997). The mechanism posited to be at work is that, as time progresses, products and practices are increasingly evaluated using a "logic of social appropriateness" rather than a "logic of instrumentality" (Westphal et al. 1997, p. 374). This shift is similar to that in Maslow's hierarchy of needs: As one feels that basic functional requirements are met, social acceptability and integration become more important considerations. To the extent that customers similarly shift some emphasis from functional performance to social acceptability after adoption, repeat use and sustained implementation should be more affected by normative concerns than initial use. The second reason to expect susceptibility to normative influence to increase as customers proceed from trial to repeat is that social disapproval based on deviations from the norm are easier to condone for trial than for repeat. Normative disapproval of a trial decision can easily be deflected by claiming exigent circumstances (when proven right) or by showing contrition and desisting (when proven wrong). These tactics, however, are not available to someone who repeatedly violates norms of proper behavior (Bosk 2003, pp. 35–70).

Note that the two reasons to expect the susceptibility to normative influence to increase as customers proceed from trial to repeat are of a different nature. The first does not pertain to a genuine difference between trial and repeat but to a change over time in how much people care about conforming to social norms. Thus, the difference across stage is merely a corollary of a temporal effect. The second reason pertains to a genuine difference between trial and repeat, regardless of time since launch.

In short, normative influence considerations lead one to expect that social contagion (i) is at work in repeat, (ii) originates from group members, (iii) varies in an inverse-U fashion with the decision maker's status, and (iv) operates with greater strength in repeat than trial. As a corollary, a contagion effect with characteristics (i)–(iv) is more likely to stem from normative influence than an effect without these characteristics.

2.5. Hypotheses

The theoretical arguments lead to four predictions for risky products:

Hypothesis 1 (H1). *New product adoption is affected by social contagion that originates from trusted peers, and people with low confidence in their judgments are more susceptible to it.*

Hypothesis 2 (H2). *Social contagion that originates from trusted peers and that is negatively moderated by the recipients' self-confidence is more pronounced in trial than in repeat.*

Hypothesis 3 (H3). *New product repeat behavior is affected by social contagion that originates from group members, and people with middle status are more susceptible to it.*

Hypothesis 4 (H4). *Social contagion that originates from group members and that is nonmonotonically moderated by the recipients' status is more pronounced in repeat than in trial.*

Two observations are in order. First, the hypotheses are based on the assumption that contagion in adoption is driven primarily by informational considerations whereas contagion in repeat is driven primarily by normative considerations. Support for the hypotheses would provide credence to this underlying assumption, but does not provide direct evidence of the informational or normative nature of contagion. This is not a major limitation, as theoretical mechanisms are typically inferred from their observable consequences rather than observed directly even in experimental research. Second, the hypotheses go far beyond basic main effects. This makes it hard to find credible alternative explanations for the data in case the hypotheses are supported.

3. Strengthening Internal Validity in Contagion Studies

Obtaining good estimates of an effect is rarely straightforward in nonexperimental studies. Whereas observational designs do not offer the same level of internal validity as randomized field experiments (e.g., Aral and Walker 2012, Hinz et al. 2011), researchers have found many ways to strengthen the internal validity of observational contagion studies.

3.1. Temporal Precedence

One way is simply to be mindful that causes precede effects, and to plan one's study accordingly. For instance, one can avoid simultaneity bias by using panel data with sufficiently fine temporal resolution and by modeling contagion in terms of lagged rather than contemporaneous peer behavior. As another example, one can avoid endogenous tie formation and truncation biases by not operationalizing contagion in terms of social ties that can come into existence only after the adoptions that one seeks to explain have occurred.

3.2. Technical Fixes

The second way to boost the internal validity of contagion research is by using one or more of the standard approaches to strengthen causal inference in observational designs. These include studying acyclic networks to avoid simultaneity bias (e.g., Iyengar et al. 2011a), using covariates or fixed effects to control for common contextual effects and attributes (e.g., Nair et al. 2010, Van den Bulte and Lilien 2001), using matching techniques to do the same (e.g., McShane et al. 2012), using instrumental variables to capture exogenous variations in contagion (e.g., Land and Deane 1992), and jointly modeling ties and behavior to account for endogenous tie formation (e.g., Lewis et al. 2012).

3.3. Theoretical Elaboration

The third way to more confidently identify contagion is theoretical elaboration. The idea is conveyed in an anecdote involving two eminent statisticians, R.A. Fisher and W.G. Cochran.

> "About 20 years ago, when asked in a meeting what can be done in observational studies to clarify the step from association to causation, Sir Ronald Fisher replied: 'Make your theories elaborate.' The reply puzzled me at first, since by Occam's razor, the advice usually given is to make theories as simple as is consistent with known data. What Sir Ronald meant, as subsequent discussion showed, was that when constructing a causal hypothesis one should envisage as many *different* consequences of its truth as possible, and plan observational studies to discover whether each of these consequences is found to hold." (Cochran 1965, p. 252, emphasis in original)

The idea, in essence, is that more elaborate predictions cannot be accounted for as easily by threats to internal validity. As Shadish et al. (2002, p. 105) note, "The more complex the pattern that is successfully predicted, the less likely it is that alternative explanations could generate the same pattern, and so the more likely it is that the treatment had a real effect." Shadish et al. call this method of strengthening internal validity "coherent pattern matching" whereas Rosenbaum (2002, pp. 209–214) calls it "increasing the specificity of predictions."

Theoretical elaboration often entails putting forward boundary conditions and moderator effects (e.g., Cochran 1965; Shadish et al. 2002, p. 105). Many psychologists and laboratory scientists have made this notion central to their research strategy. Even when using randomized experiments, they put greater confidence in results supporting moderator predictions than basic main effects. For instance, a moderator effect limits the set of possible confounds to only those that would generate the same pattern, e.g., only omitted variables that are similarly moderated.

Theoretical elaboration may also increase causal confidence by positing nonmonotonic effects, Cochran (1965) notes. For instance, a predicted nonmonotonic effect rules out monotonic confounds as threats to validity.

Theoretical elaboration may also involve positing that a specific cause has an effect on one outcome variable but not on another. Threats to internal

validity in such "nonequivalent dependent variables" designs are less plausible when purported confounds are expected to affect all dependent variables but one observes only responses on those outcomes consistent with one's theory (Rosenbaum 2002, pp. 209–213; Shadish et al. 2002, pp. 110–111). Though specificity of outcome does not guarantee the causal nature of associations in observational designs, it makes potential confounds common across outcomes less likely and so strengthens the evidence of a causal connection (Hill 1965, Holland 1986).

Our hypotheses follow R.A. Fisher's dictum, as they involve different dependent variables, different sources of contagion, different moderators, and a nonmonotonic pattern. This allows us to be more confident that the analysis detects genuine effects. Before we proceed with the empirics, a brief clarification about the role of informational versus normative influence in our application may be in order. We use the experimentally documented theoretical distinction between informational and normative influence to motivate nonobvious hypotheses involving (i) different dependent variables, (ii) different sources of contagion, (iii) different moderators, and (iv) a nonmonotonic pattern. The distinction between informational and normative influence is *a means* and *not the end* in our application of R.A. Fisher's insight. Accordingly, the hypotheses are stated in terms of observables rather than informational versus normative influence, and support for the hypotheses provides indirect credence but not direct evidence of the informational versus normative nature of contagion.

4. Research Setting

We analyze the acceptance of a risky new prescription drug over a 17-month period, studied earlier by Iyengar et al. (2011a), hereafter referred to as IVV. We extend that earlier work by investigating (i) both trial and repeat[2] and (ii) contagion from both trusted expert peers and immediate colleagues.[3]

The drug is used to treat a chronic viral infection that can cause severe damage to internal organs and which, if left untreated, sometimes even leads to patients' death. Physicians cannot observe drug efficacy quickly and adjust a patient's therapy if necessary. Also, there is uncertainty in the medical community about the best treatment because there is no compelling evidence about the new drug's long-term efficacy compared to that of two older drugs. In such situations characterized by high risk, high complexity, and low observability of results, potential adopters are likely to turn to opinion leaders for guidance (Hahn et al. 1994).[4]

Social contagion may also be at work after trial. The first reason is that the physicians cannot quickly assess the drug's efficacy even after having prescribed it. The drug treats a chronic rather than an acute condition that is primarily asymptomatic until the patient is gravely ill. Not only do patients not feel whether the treatment is working, but even physicians have difficulty assessing improvements in patient health. They can only do so using indirect indicators, such as viral loads. Moreover, even if the treatment is effective, progress occurs very slowly. All this makes the product's effectiveness with one's patients difficult to assess. The effectiveness of the focal drug compared to its two established competitors is ambiguous as well. Even large-scale clinical trials with strict test/control conditions provide far from definitive evidence for long-term superiority. Considering how difficult it is for physicians to gain conclusive information from experience, it is possible that they rely on their peers' judgment even after trial.

The second reason that contagion may affect repeat behavior is that physicians want to act in a way that their peers deem proper and legitimate. Physicians look to their peers for information as well as normative guidance (Bosk 2003, pp. 35–70; Prosser and Walley 2006). Normative influence is likely to be stronger in repeat than in trial decisions and to vary as a function of status, something which is quite salient among physicians (Bosk 2003, pp. 36-67, 111–146; Menchik and Meltzer 2010) and can affect their prescription behavior (Burt 1987, Menzel 1957).

Physicians who are influenced by the normative expectations of their colleagues do not necessarily make medically suboptimal choices that jeopardize the lives of their patients to look good. Believing they do would be misguided in our research setting where it was far from clear-cut which treatment option was medically optimal. When faced with such ambiguity, acting in ways that fellow medical professionals deem proper and legitimate is medically reasonable. More generally, social-normative influence is especially important when there is ambiguity about the objectively right course of action (Asch 1956, Deutsch and Gerard 1955).

Perhaps more surprising is that social-normative influence can also be important when there may be

[2] We exclude refill prescriptions from the repeat data. Hence, the repeat events we study involve the physicians writing a new prescription.

[3] Hypothesis H1 was already documented by IVV using the same data but omitting immediate colleagues as a distinct source of contagion. Though our evidence in support of H1 is hence a robustness check of IVV's earlier finding rather than truly new evidence, H1 is part of our broader aim to document differences in social contagion between trial and repeat posited in H2.

[4] The severity of the medical condition and the limited observability of effectiveness also make willful experimentation on patients by forward-looking physicians quite unlikely (Chintagunta et al. 2012, pp. 807–808).

major consequences to one's choice of action. Some might expect normative influence effects to vanish for such important decisions as the treatment of a potentially lethal medical condition. However, decisions of great importance are more stressful, which increases rather than decreases the tendency to conform to the group (Darley 1966, Janis 1972, Perrin and Spencer 1981). Baron et al. (1996) show that conformity is especially high when *both* task importance and task difficulty are high. This corresponds to our research setting where physicians need to decide on using a new drug to treat a potentially lethal medical condition in the absence of unambiguous evidence on the relative clinical superiority of the new drug.

Finally, some readers may be disturbed by our proposal that status affects medical decision making. Our argument, however, is not that status considerations affect the preference for the new drug directly. Rather, it is that physicians' susceptibility to social-normative influence is contingent on their status, in accordance with the middle-status conformity hypothesis. Thus, we posit that status moderates the effect of contagion from immediate colleagues in a nonmonotonic fashion, without making any claims about the presence or shape of a main effect. To properly test the proposed interaction effect, we include all lower order status terms in the model.

5. Data

The data cover the adoption and repeat prescriptions of the new drug by physicians in Los Angeles (LA), New York City (NYC), and San Francisco (SF) over a period of 17 months from the time of launch. As the drug was the third entry in its category, the relevant population within each city was defined by the firm as every physician who had prescribed at least one of the other two drugs in the two years before the focal drug's launch.

The data consists of (i) monthly physician prescription data (excluding refills), (ii) answers to a survey by physicians providing information on discussion and patient referral ties, self-reported opinion leadership, and several other physician characteristics, (iii) the address where each physician practiced, and (iv) company records on sales calls to each physician.

5.1. Prescription Data

For each physician within the network boundary (not only survey respondents), the time of adoption is measured using monthly individual-level prescription data from IMS Health. Of the 193 doctors who responded to the survey, 68 (35%) adopted within 17 months. The average prescription incidence rate after adoption, i.e., the monthly repeat rate, is around 75%.

5.2. Discussion and Referral Ties

A mail and Internet survey was administered to all physicians in the network boundary. The survey asked the respondents to name up to eight physicians with whom they felt comfortable discussing the clinical management and treatment of the disease for which the drug was developed (discussion ties) and up to eight physicians to whom they typically refer patients with the disease (referral ties). Both lists could but did not need to overlap. The highest number of discussion partners nominated by any physician was six and that of referral partners was five. Both of these values are below the maximum number of nominations allowed. The survey was administered in SF several months before the product launch, and 10 months after the launch in LA and NYC. This exogenous variation helps us address threats to internal validity.

Sixty-seven of the 150 physicians in the population of interest in SF responded. Fifty-seven of 197 in LA responded, and 69 of 284 in NYC responded. As discussed in detail by IVV (see also Christakis and Fowler 2011), there is no evidence of nonresponse bias and the 24%–45% response rates avoid sizable error in the network-based covariates introduced below.

The study restricts the relevant networks to physicians practicing in the same city. The importance of local as opposed to national opinion leaders is well documented in the medical literature. Also, the pharmaceutical industry is keenly aware of the importance of such social dynamics at the local level (e.g., IVV 2011a, Liu and Gupta 2012). Hence, physicians who were nominated by survey respondents but were not part of the population of interest were excluded from the study. In contrast, physicians who were part of the population of interest but did not respond to the survey were included in the set of potential discussion or referral partners. A physician who is mentioned as both a discussion and referral partner is deemed twice as influential as another who is mentioned as only one or the other. Contagion over this total network describes the pattern of adoption better than contagion over only discussion or referral ties (IVV 2011a).

5.3. Immediate Colleagues

Normative influence is more pronounced among individuals forming a group, and norms often operate locally (Bosk 2003, pp. 51–67; Cialdini and Trost 1998, Deutsch and Gerard 1955, Hogg 2010, Turner 1991). Consequently, immediate colleagues that one interacts with daily are likely to exert normative influence through identification. They help define the local norm of what is legitimate practice; the desire to maintain a satisfactory relationship with

Table 1 Fraction of All Colleague-Dyads That Involve a Discussion or Referral Tie

	San Francisco (SF)	Los Angeles (LA)	New York City (NYC)
Discussion	0.086	0.038	0.067
Referral	0.049	0.026	0.017

Table 2 Fraction of All Discussion and Referral Ties That Involve Colleagues

	San Francisco (SF)	Los Angeles (LA)	New York City (NYC)
Discussion	0.170	0.042	0.176
Referral	0.139	0.046	0.058

one's colleagues motivates people to conform to their expectations.[5]

We use the group practice or hospital where each physician works to identify his or her immediate colleagues. Physicians do not consider each of their colleagues to be a trusted expert on the medical condition treated by the new drug. As shown in the top row of Table 1, physicians in SF report on average only 9% of their colleagues for discussion and only 5% for referral about this specific medical ailment. The numbers for NYC and LA are even lower. However, controlling for the fact that there are many more noncolleagues than colleagues available, physicians are significantly more likely to turn to colleagues than to noncolleagues for discussion or referral ($p < 0.01$).[6]

Table 2 reports what fraction of referral and discussion ties involves colleagues. Once again, the evidence is clear that the peers one turns to for discussion or referral about the ailment treated by the drug are rarely one's immediate colleagues.

5.4. Contagion Variables

We model social contagion as the effect of exposure to others' prior use of the drug. The extent to which physician i is exposed at time t to *influence from discussion and referral partners* is captured through the term $\sum_j w_{ij1} q_{jt-1}$, where w_{ij1} captures how relevant each physician j is to i for discussion or referral (0, 1, 2), and q_{jt-1} is the number of prescriptions written by j at time $t-1$. The volume-weighted contagion from discussion and referral partners captures exposure to risk-reducing information. The more a physician's network contacts have prescribed the drug recently, especially in high volumes, the more credible their input and hence the more confident the physician feels that using the drug may help her own patients (IVV 2011a).

The extent to which physician i is exposed at time t to *influence from immediate colleagues* is captured through the term $w_{ij2} s_{jt-1}$, where w_{ij2} equals 1 if i and j are colleagues and zero otherwise, and s_{jt-1} is the share at time $t-1$ of the new drug in j's total number of prescriptions in the category. Though we use volume-weighted contagion from immediate colleagues in our robustness checks, we prefer using the share-weighting based on theoretical grounds. As Turner (1991, p. 87) notes, intrapersonal consistency is a sign of commitment—an insight that underlies the popularity of share-of-wallet or share-of-category-requirements as a measure of affective brand loyalty (Fader and Schmittlein 1993). This implies that share-weighted contagion may capture exposure to colleagues strongly committed to the new drug better than volume-weighted contagion. A colleague treating five patients for the medical condition and prescribing the new drug for all of them is more committed to it than a colleague prescribing it for only half of his 10 patients. Hence, share-weighting may better reflect how strongly each colleague feels that using the new drug is the proper thing to do.

5.5. Confidence: Self-Reported Opinion Leadership

Self-reported opinion leadership (*SRL*) captures the extent to which a physician feels that he or she can learn from others. *SRL* is measured using a six item scale (for details, see IVV 2011a). We construct the *SRL* variable by taking the average of the six items. The first two scale items pertain to frequency of interaction, whereas the last four are an assessment of oneself versus others as a valuable source of information about treatment options. Thus, high *SRL* is likely associated with high self-confidence.[7]

[5] Given our research setting of physicians in the United States making treatment decisions for a potentially lethal medical condition, we expect normative influence to operate through identification and referent power, not through compliance and coercive/reward power. Though the experiments of Deutsch and Gerard (1955) focused on the latter process, the importance of the former is now well documented and accepted (e.g., Kelman 1958, 2006; Turner 1991, p. 37).

[6] Standard test procedures such as a chi-square test on a 2-by-2 matrix (presence or absence of tie versus colleague or not) do not properly handle the lack of independence among the dyadic observations. We resolve that problem by regressing the sociomatrix of discussion/referral ties on the sociomatrix of collegial ties (ordinary least squares (OLS) is unbiased even when errors are not independent) and using the permutation-based quadratic assignment procedure for assessing statistical significance (Krackhardt 1988).

[7] Several studies have shown that *SRL* is rather weakly correlated with sociometric status as an opinion leader (IVV 2011a; Jacoby 1974; Lee et al. 2010; Molitor et al. 2011; Rogers and Svenning 1969, pp. 224–227) or other-reported opinion leadership (Gnambs and Batinic 2013). This suggests that *SRL* need not capture opinion leadership. Based on its low correlation with sociometric status and their finding that *SRL* is negatively correlated with susceptibility to contagion, IVV propose that *SRL* captures self-confidence rather than opinion leadership. Subsequent research by Martin and Lueg (2013) finds that the link between word-of-mouth

Self-confidence is likely to moderate the eagerness to learn from others and, hence, to affect the susceptibility to contagion from peers one turns to for discussion of treatment options and referral of patients. However, there is no reason to expect self-confidence to moderate social-normative influence. How confident a physician is in his medical prowess and judgment will not affect his colleagues' willingness to enforce social norms. It will also not affect the physician's ability to successfully defy social norms (and hence normative influence through compliance) or his eagerness to be considered part of the group (and hence normative influence through identification). The extent to which a physician is subject to and susceptible to social-normative influence from others depends on how others esteem and defer to him, i.e., his status, but not on how confident he is in his own medical prowess and judgment. In short, being self-confident and perceiving others to be less knowledgeable than oneself are distinct from being accorded high status by others (IVV 2011a) and from disregarding social norms. Thus there is no reason to expect SRL to moderate contagion through normative influence (Deutsch and Gerard 1955).[8]

5.6. Status: Indegree Centrality

Status is one's social rank in terms of esteem and respect bestowed by others (e.g., Phillips and Zuckerman 2001). It is measured here as the logarithm of the number of discussion and referral nominations received from other physicians.[9] Such "indegree centrality" is the most basic measure of status in networks, especially those involving deferential ties such as advice-seeking or favor-seeking (Hu and Van den Bulte 2014; Knoke and Burt 1983; Lu et al. 2013; Menchik and Meltzer 2010; Menzel 1957; Sauder et al. 2012; Sgourev 2011; Wasserman and Faust 1994, p. 202). As discussed by IVV, many studies show that indegree is robust to random node sampling as long as the sampling rate is 20% or higher (see Costenbader and Valente 2003). We use the log-transformation (after adding 1 to avoid the log(0) problem) because indegree has a highly right-skewed distribution that creates numerical problems when testing for middle-status conformity by interacting colleagues contagion with indegree and its square. The log transformation stabilizes the estimation.

5.7. Control Variables

We control for several other physician characteristics that might be associated with trial or repeat. *Past Drug* 1 and *Past Drug* 2 are the number of prescriptions written by each physician for each of the other two drugs in the market during the 12 months before the launch of the focal drug. *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. *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. *Primary Care* is a dummy variable capturing whether the doctor is a primary care physician rather than a specialist more likely to focus on the relevant medical condition (i.e., internal medicine, gastroenterologists, and infectious diseases).

Sales Calls are the monthly physician-level amount of detailing for the focal drug. There was very limited medical journal advertising and no direct-to-consumer advertising. There was also no sampling because of major concerns about patients developing resistance after taking a sample but not continuing on the drug.

City dummies for LA and NYC control for city-specific differences. SF is the baseline.

Time dummies for each month capture any system-wide time-varying factor, such as aggregate diffusion, changes in disease prevalence or the emergence of new clinical evidence. The dummies capture all cross-temporal variation in the mean tendency to adopt or repeat, leaving only variance across physicians within particular months to be explained by contagion.

Lagged prescription volume. Including lagged behavior as a covariate often helps controlling for both state dependency and unobserved heterogeneity. It also controls for endogeneity of sales calls when managers or salespeople allocate their effort based on prior prescription volume. In addition, it can capture variation across time and physicians of (i) the number of patients seen by the physician for whom the drug could be part of a treatment plan, and (ii) the physician's "enthusiasm" for the

(WOM) use and attitude is stronger for people with low versus high self-perceived knowledge. Along similar lines, Szymanowski and Gijsbrechts (2013) find that self-reported market mavens (i.e., people reporting acting as an opinion leader and sharing their information and experiences with others) learn less from their experience, which those authors interpret as possibly stemming from overconfidence.

[8] Also, the middle-status conformity hypothesis does not make any prediction about a change in self-perceived status. Instead, our application of the hypothesis implies that physicians expect that their prescription behavior will affect their *true* status, which we measure as degree centrality rather than *SRL*.

[9] Self-reported measures of status such as *SRL* are dubious in general because status by definition involves esteem bestowed by others. They are especially useless when testing for middle-status conformity, which requires a common metric across all actors (Hu and Van den Bulte 2014, Phillips and Zuckerman 2001). This requirement is obviously violated when using self-reported status measures subject to the well documented Lake Wobegon or above-average effect.

new drug (Bell and Song 2007). Of course, lagged prescription volume is zero until after adoption, so it can be a covariate only when modeling repeat behavior.

5.8. Final Data Set

Data on past prescription of the two incumbent drugs are missing for eight doctors, three of whom adopted the focal drug. After deleting these eight physicians, there are 185 adoption spells of which 65 end with adoption, and 570 opportunities for repeat of which 424 show repeat behavior. Descriptive statistics for physician-months up to adoption (2,575), physician-months with adoption (65), physician-months after adoption (570), and physician-months with repeat (424) are reported in the Web Appendix (available as supplemental material at http://dx.doi.org/10.1287/mksc.2014.0888).

The plots in Figure 1 show how the average hazard of adoption, sales calls, and the two contagion variables evolved over time among physicians who had not adopted yet. Though the hazard is rather flat with only three of the 17 values outside the narrow 2%–3.5% range, this does not imply the absence of contagion because neither heterogeneity in physician characteristics, which creates spurious negative duration dependence, nor sales calls, which trend downwards, are accounted for (see IVV 2011a for details). The amount of volume-weighted influence from network ties operating before adoption increases steadily, whereas the volume of share-weighted influence from immediate colleagues increases more slowly after month 6. The two tie-specific contagion variables exhibit a different pattern, as do the ties themselves (see §5.3).

The plots in Figure 2 show how the average repeat rate, sales calls, and the two contagion variables evolve over time among physicians who had already adopted. The repeat rate in the second month is 100%, as all six physicians who adopted in the first month also prescribed in the next month. The average repeat rate decreases over time, which is consistent with evidence that heavy users adopted the drug early (IVV 2011a). Average sales calls decrease after month 5, which is consistent with a "hard launch" strategy (Liu and Gupta 2012, Sinha and Zoltners 2000), but may also result from the firm's allocating more sales calls to heavy prescribers; light prescribers, who tend to adopt late, make up an increasing proportion of the repeat-prescriber base. The amount of volume-weighted influence from network ties increases steadily, whereas the amount of share-weighted influence from immediate colleagues increases only after four months. The high value in month 2 is not a fluke and stems from the fact that four of the six adopters in month 1 were colleagues in a prominent research/teaching hospital.

Figure 1 Descriptive Plots for Trial (Using All Physician-Months in Which Physicians Are at Risk of Adopting)

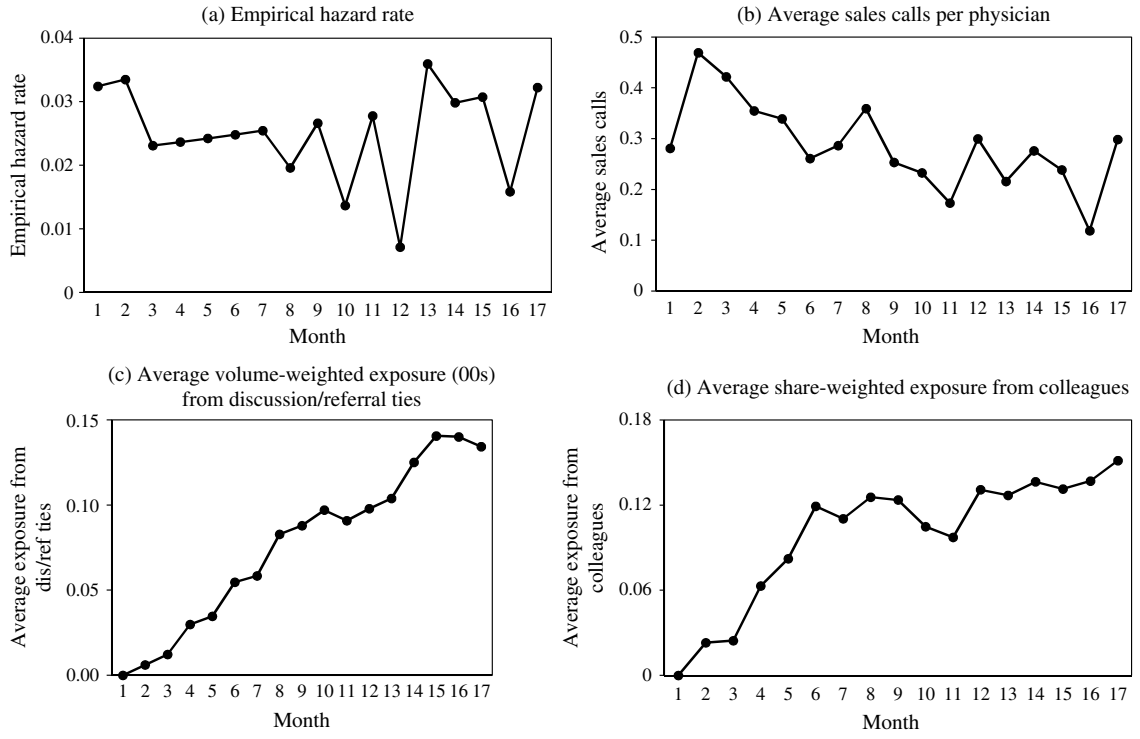
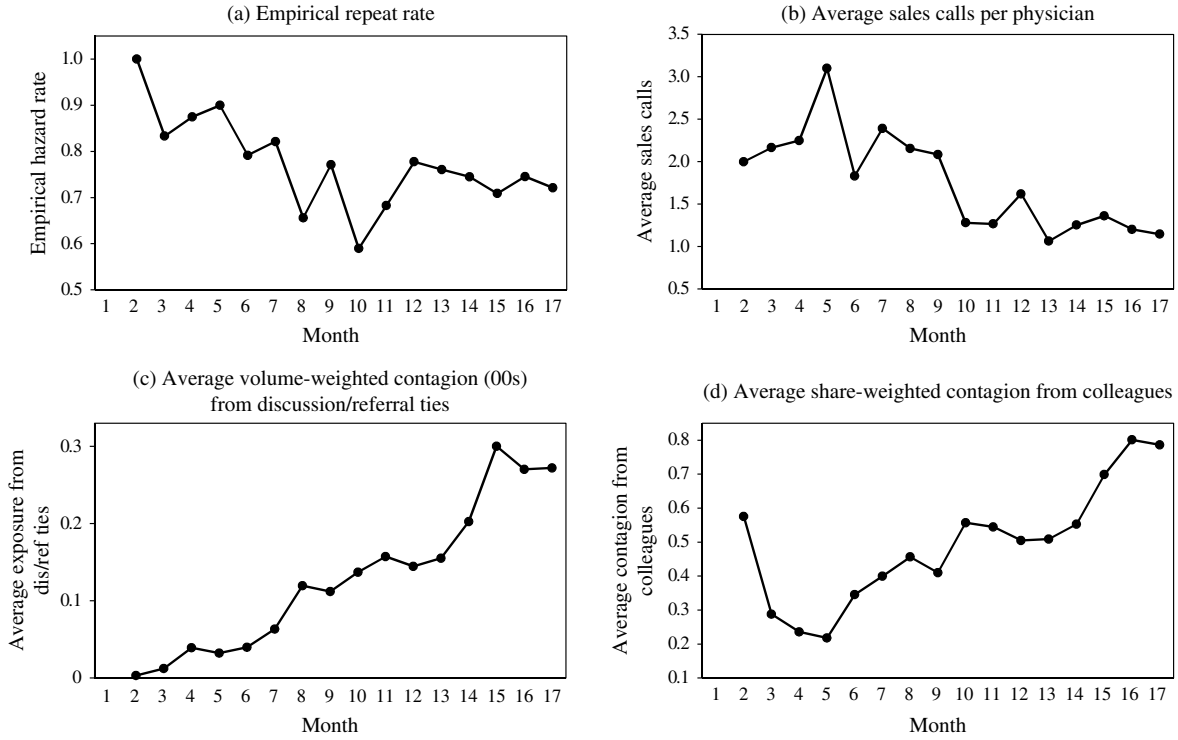


Figure 2 Descriptive Plots for Repeat (Using All Physician-Months in Which Physicians Have Already Adopted)



6. Model

We model adoption and repeat prescription of the focal drug in discrete time. We model repeat conditional on adoption, rendering selectivity moot (Poirier and Ruud 1981). We account for possible endogeneity in sales calls using a control function approach.

6.1. Adoption Model

We specify the appeal or utility that physician i sees in trying the drug at period t (U_{it}^a) as

$$U_{it}^a = \beta_{0i}^a + X_{it}^a\beta_1^a + \varepsilon_{it}^a, \quad \text{where } \varepsilon_{it}^a \sim N(0,1) \text{ and } \beta_{0i}^a \sim N(\bar{\beta}_0^a, \sigma_a^2). \quad (1)$$

The row vector X_{it}^a contains covariates *up to adoption* or month 17, whichever happens first; β_1^a is a column vector of corresponding parameters. The parameter β_{0i}^a is a physician-specific baseline utility and controls for unobserved characteristics related to adoption. We assume that β_{0i}^a follows a normal distribution. We express the discrete-time hazard of adoption or trial as

$$P(Y_{it}^a = 1 \mid Y_{it-1}^a = 0) = P(U_{it}^a > 0) = \Phi(\beta_{0i}^a + X_{it}^a\beta_1^a), \quad (2)$$

where Y_{it}^a is an indicator variable that equals 0 before adoption and 1 at the time of adoption and later, and Φ is the normal cumulative distribution function. Therefore, the likelihood of observing $Y_{it}^a = y_{it}^a$, where $y_{it}^a \in \{0, 1\}$, can be expressed as

$$P(Y_{it}^a = y_{it}^a \mid Y_{it-1}^a = 0) = \Phi(\beta_{0i}^a + X_{it}^a\beta_1^a)^{y_{it}^a} \cdot (1 - \Phi(\beta_{0i}^a + X_{it}^a\beta_1^a))^{1-y_{it}^a}. \quad (3)$$

Two observations are in order. First, since adoption is a nonrecurrent event, the lagged dependent variables are always zero; including them as covariates is pointless. Second, we do not include person-specific fixed effects as they generate truncation biases in the adoption equation (Van den Bulte and Iyengar 2011).

6.2. Repeat Model

Whereas trial can occur only once, repeat can occur several times. We specify the utility that physician i sees in repeat prescribing the drug at time t given adoption at a prior time (U_{it}^r) as

$$U_{it}^r = \beta_{0i}^r + X_{it}^r\beta_1^r + \varepsilon_{it}^r, \quad \text{where } \varepsilon_{it}^r \sim N(0,1) \text{ and } \beta_{0i}^r \sim N(\bar{\beta}_0^r, \sigma_r^2). \quad (4)$$

The row vector X_{it}^r contains covariates *after adoption*; β_1^r is a column vector of corresponding parameters. The parameter β_{0i}^r is a physician-specific baseline of repeat utility, which is normally distributed. The probability of repeat prescription, conditional on having adopted earlier, is then given by

$$P(Y_{it}^r = 1 \mid Y_{it-1}^a = 1) = P(U_{it}^r > 0) = \Phi(\beta_{0i}^r + X_{it}^r\beta_1^r), \quad (5)$$

where Y_{it}^r is an indicator variable that takes a value of 1 if i prescribes at a time t and is 0 otherwise. Therefore, the likelihood of observing $Y_{it}^r = y_{it}^r$, where $y_{it}^r \in \{0, 1\}$, is

$$P(Y_{it}^r = y_{it}^r \mid Y_{it-1}^a = 1) = \Phi(\beta_{0i}^r + X_{it}^r \beta_1^r)^{y_{it}^r} \cdot (1 - \Phi(\beta_{0i}^r + X_{it}^r \beta_1^r))^{1-y_{it}^r}. \quad (6)$$

Several points are worth noting. First, because repeat can be a recurrent event, one can include lagged dependent variables among the covariates as well as random or fixed effects. We use random effects because fixed effects result in inconsistent estimates in probit models (e.g., Wooldridge 2002, p. 484). Second, repeat is by definition conditional on trial. Each physician's adoption and repeat events occur in nonoverlapping time periods. We assume the absence of forward-looking experimentation by physicians in this category, consistently with Chintagunta et al. (2012). Consequently, our repeat model is conditional rather than unconditional on trial, the random shocks between trial and repeat can be treated as uncorrelated, and exclusion restrictions are unnecessary (e.g., Poirier and Ruud 1981). However, the time-invariant physician-specific effects may be correlated across stages. Third, including both random effects and lagged dependent variables is appropriate if the initial value of the lagged dependent variable can be assumed to be independent of the random effect (e.g., Wooldridge 2002, p. 494). In our setting, this requires the random effects in trial and repeat to be uncorrelated.

6.3. Correlated Random Effects

We allow the physician-specific random effects of trial and repeat to be correlated as

$$\begin{bmatrix} \beta_{0i}^a \\ \beta_{0i}^r \end{bmatrix} \sim N\left(\begin{bmatrix} \bar{\beta}_0^a \\ \bar{\beta}_0^r \end{bmatrix}, \begin{bmatrix} \sigma_a^2 & \sigma_{ar} \\ \sigma_{ar} & \sigma_r^2 \end{bmatrix} \right). \quad (7)$$

Let Y_{it} indicate whether i prescribes at time t, T_i^a denote the period in which physician i adopts the focal drug or is right-censored, and T denote the length of data window (i.e., $T = 17$).[10] The likelihood is then

$$P(Y_{it} = y_{it} \mid \beta_1^a, \beta_1^r) = \int_{\beta_{0i}^a, \beta_{0i}^r} \prod_{t=1}^{T_i^a} P(Y_{it} = y_{it} \mid Y_{it-1}^a = 0, \beta_1^a, \beta_{0i}^a) \cdot \prod_{t=T_i^a+1}^{T} P(Y_{it} = y_{it} \mid Y_{it-1}^a = 1, \beta_1^r, \beta_{0i}^r) \cdot f(\beta_{0i}^a, \beta_{0i}^r) d\beta_{0i}^a d\beta_{0i}^r. \quad (8)$$

We estimate the model using simulated maximum likelihood.

[10] Right-censored physicians who do not adopt within the 17-month data window have $T_i^a = T$.

6.4. Control Function Approach for Endogeneity in Sales Calls

Marketers and salespeople may have set the amount of detailing effort towards a physician in a particular month based on demand shocks that are not accounted for by the covariates in the model. The resulting correlation between sales calls and error terms, if not properly addressed, would bias the model estimates. We handle this possible endogeneity using a control function approach that quantifies its severity by directly estimating the correlation between the random shocks in physician behavior and sales calls, as detailed in the Web Appendix.

6.5. Quasi-Complete Separation

The repeat rate in month 2 was 100% as was the repeat rate of primary-care physicians (PCP). With such "quasi-complete separation," the log-likelihood reaches its true maximum only when the parameter estimate for the month 2 and PCP dummies reach $+\infty$. Hence, there is no finite maximum likelihood estimate (Albert and Anderson 1984). One simple solution follows from recognizing that we already know the true maximum likelihood value of the two coefficients in our data ($+\infty$) and that, at that value, the offending observations provide no information about the other parameters. Hence, we can simply delete the month 2 and PCP observations from the data set, omit their dummy variables from the model, and proceed as usual (Andersen 1987, Lien and Rearden 1990, Oksanen 1986). A variant is to keep the observations in the data while fixing the two dummies' coefficients to such a high value that their predicted values are very close to unity regardless of the other parameter estimates. We do so by forcing the coefficient of the two dummies to 10 ($\Phi(10) > 1 - 10^{-15}$).

7. Results

Our covariates include terms for contagion from expert peers and colleagues, terms for the interactions hypothesized in H1 and H3, and the control variables described in §5.7. We first estimated the model with correlated random effects but without lagged volume. Consistent with prior evidence that a nonparametric baseline absorbs much of the effects of unobserved heterogeneity in hazard models for nonrepeated events (e.g., Lin and Wei 1989, Struthers and Kalbfleisch 1986), the model is overparameterized. Specifically, the variance in random effects in trial is quite small ($\hat{\sigma}_a^2 = 0.014$, $p = 0.533$). A second model without that random effect and its associated covariance performs better in Bayesian

Information Criterion (BIC) terms (Δ BIC = 9.99).[11] Given the absence of random effects in the trial equation, adding lagged volume as a covariate to control for state dependency in the repeat equation does not create an initial condition problem. Because this third model fits markedly better than the first (ΔBIC = 24.10) and second model (ΔBIC = 14.11; $\Delta - 2LL = 20.74$, $p < 0.001$), we use it as the main specification.

Table 3 reports the parameter estimates of substantive interest and of several control variables. SRL and *Indegree* (log-transformed) are mean-centered before estimation. Thus the coefficient of nonmoderated contagion is the effect for the "average" physician. To avoid reporting very small coefficients, volume-weighted contagion is expressed in hundreds of units.

Though our model includes many control variables and several nonlinear effects, collinearity is not a concern since the condition index of the data matrix is only 15.47 in trial and 15.30 in repeat, well below 30, which is commonly considered a necessary condition for harmful collinearity.

Table 3 shows the presence of contagion in not only the trial hazard ($\Delta - 2LL = 25.36$, df = 5, $p < 0.001$) but also in repeat incidence ($\Delta - 2LL = 13.10$, df = 5, $p < 0.05$). Unlike the earlier analysis by IVV, we do not find a significant linear effect of sociometric status on the adoption hazard. That the lower-order degree effects are different is hardly surprising because the higher-order interaction covariates differ between the two analyses designed with different objectives in mind (compare Table 3 with Table 4 in IVV 2011a).

We next turn to the findings of key interest: the contrasts between advice/discussion ties versus colleagues as sources of influence, and the contrast between trial and repeat as stages in new product acceptance behavior.

7.1. Contagion from Discussion/Referral Ties versus Colleagues

Peers one turns to for discussion or referral exert contagion in trial. The strength of that influence varies across potential adopters. In contrast, those same peers exert no influence in repeat. As reported in the first column in Table 3, the main effect of contagion from discussion/referral ties on the "average" physician is not significant, but physicians with a low SRL are significantly more susceptible to such contagion in the trial stage ($p < 0.01$). In contrast, there is no main or moderator effect at the repeat stage. Figures 3(a) and 3(b) visually convey the relationship between contagion and self-reported leadership. Figure 3(a) shows that contagion from discussion/referral ties is positive at trial for physicians with SRL lower than 4.57, which corresponds to 55% of the physicians. It is significantly positive at 95% confidence for physicians with SRL lower than 3.56 (27% of physicians) and never turns significantly negative. Figure 3(b) shows a very different pattern for repeat: There is no significant contagion effect from discussion/referral ties at any level of SRL.

The coefficients for contagion from colleagues in Table 3 and the bottom two panels in Figure 3 show that this type of contagion operates quite differently. In trial, colleagues exert significant contagion on the "average" physician ($p < 0.05$), and the effect is not significantly moderated by the potential adopter's status. In repeat, the effect varies in a pronounced inverse-U fashion with the physician's status ($\Delta - 2LL = 10.64$, df = 2, $p < 0.01$). The latter is conveyed more compellingly by the plot in Figure 3(d). The expected contagion effect from colleagues is the largest for a physician with *Indegree* of about 5, which is well within the observed range. The effect is significantly positive at 95% confidence for physicians with *Indegree* between 1 and 10 (21% of physicians, between the 77th and 98th percentiles of the *Indegree* distribution).[12] The confidence band in Figure 3(c) is extremely wide because of the insignificant moderator effects of status in trial. Though not obvious from the plot, the 76% of physicians with *Indegree* less than 1 exhibit positive contagion from colleagues at trial, significant at 95% confidence.

So, discussion and referral ties have a pronounced effect in trial but not repeat, colleagues have an effect on both trial and repeat, and an inverse-U relation with status is present only for colleagues contagion at the repeat stage. These findings support Hypotheses H1 and H3.

7.2. Trial versus Repeat

We now turn to whether contagion operates differently across trial and repeat, as posited in Hypotheses H2 and H4. Our model structure makes formal testing easy because the discrete-time hazard of trial and the probability of repeat are both modeled using a probit specification. We use an LRT to compare the full model in Table 3 (where all coefficients are allowed to vary freely across stages)

[11] The difference in deviance (−2LL) between the two models is only 3.26. This would not be significant at even 10% under a likelihood ratio test (LRT) with 2 df. However, an LRT is not appropriate here because it involves restricting a variance to zero, which lies on the boundary of the parameter space. Because we observe 185 adoption spells and 570 opportunities for repeat, we use $N = 755$ when computing BIC values.

[12] The critical *Indegree* value at the lower end is 0.38. Because *Indegree* is a count variable we round it up to 1. Re-estimating the model without mean-centering such that the linear contagion effect pertains to a physician with zero *Indegree* confirms that colleagues contagion effect is not significant at 95% confidence at *Indegree* = 0.

Table 3 Model Estimates

Variables	Trial hazard	Repeat probability
Intercept	−2.069***	−0.333
	(0.312)	(0.467)
SRL	0.133	−0.088
	(0.069)	(0.157)
Ln(*Indegree* + 1)	0.106	0.073
	(0.228)	(0.425)
Ln(*Indegree* + 1)^2	0.020	0.126
	(0.132)	(0.299)
Contagion from Dis/Ref Ties (00s)	0.056	−0.067
	(0.344)	(0.423)
Contagion from Dis/Ref Ties (00s) × *SRL*	−0.677**	0.390
	(0.250)	(0.260)
Contagion from Colleagues	0.759*	0.479
	(0.377)	(0.257)
Contagion from Colleagues × Ln(*Indegree* + 1)	0.625	2.533***
	(0.917)	(0.686)
Contagion from Colleagues × Ln(*Indegree* + 1)^2	−0.787	−0.840*
	(1.213)	(0.305)
Solo Practice	−0.044	0.487
	(0.180)	(0.306)
University/Teaching Hospital	0.226	0.975**
	(0.186)	(0.344)
Primary Care	−0.223	10[a]
	(0.307)	
Early Referral	−0.286	0.900
	(0.197)	(0.616)
Past Drug 1	0.000	0.010***
	(0.002)	(0.003)
Past Drug 2	0.006**	−0.003
	(0.002)	(0.003)
Sales Calls	0.556**	−0.201
	(0.195)	(0.385)
Endogeneity Correlation	−0.288	0.269
	(0.201)	(0.342)
Ln(q_{it-1} + 1)	—	0.892***
		(0.183)
Random Effect Stand. Dev.	0[b]	0.473***
		(0.166)
Random Effects Covariance	0[b]	

Notes. Standard errors in parentheses. LL = −406.79, BIC = 1,270.82. The model includes several additional covariates: Monthly time dummies (16 for trial, 14 for repeat) and city dummies for LA and NYC in both equations. These estimates are not reported to avoid clutter.

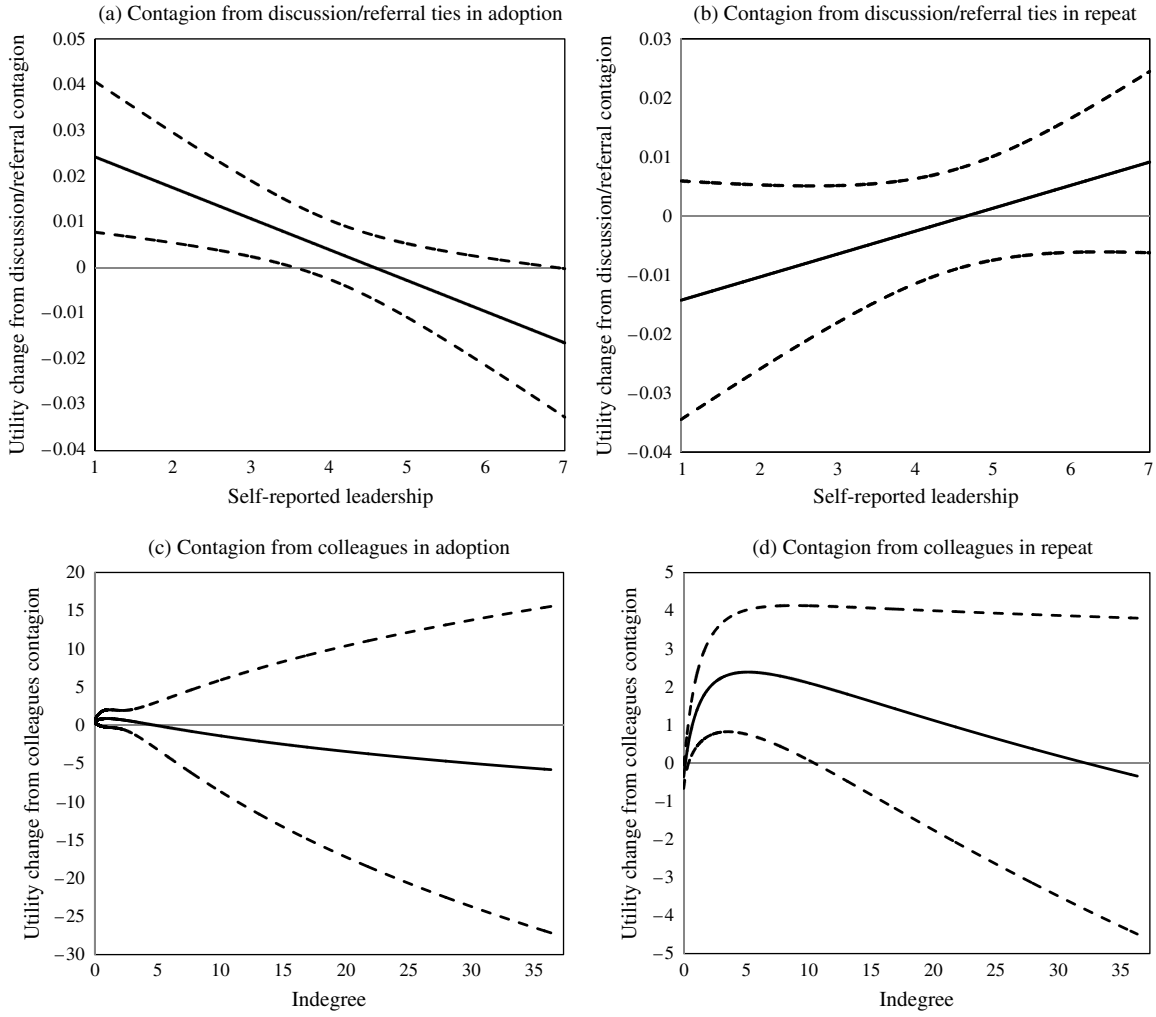
[a]Dummies for Primary Care and Month 2 are perfect predictors for repeat incidence. We set their coefficients to a very large number (10). Thus the predicted repeat probability for these physician-months is essentially 1 and the observations do not affect the likelihood estimation. See §6.5.

[b]Set to zero based on BIC. See first paragraph of §7.

*$p \le 0.05$; **$p \le 0.01$; ***$p \le 0.001$.

against a restricted model where the two discussion/referral contagion coefficients and the three colleagues contagion coefficients are constrained to be equal across trial and repeat. To account for the arbitrary scaling in probit models, we specify a model where the five contagion effects are restricted to be equal across stages up to a common scaling constant, as proposed by Train (2003, p. 26), while all other coefficients vary freely. This model fits significantly worse than the unconstrained model ($\Delta - 2\text{LL} = 14.21$, df = 4, $p < 0.01$), indicating that contagion operates differently across trial and repeat. Additional nested tests indicate that this also holds for contagion from immediate colleagues considered separately (H4, $p < 0.01$) but not for contagion from discussion/referral ties considered separately (H2, $p > 0.10$). The latter is consistent with the wide confidence bounds in Figure 3(b).

Figure 3 Social Contagion in Adoption and Repeat Incidence



Our discussion in §2.4 proposed two reasons to expect the susceptibility to normative influence to increase as customers proceed from trial to repeat. One reason pertained to a genuine difference between trial and repeat, regardless of time since launch, whereas the other pertained to a change over time in how much people conform to social norms, with the difference between trial and repeat only being a corollary of this temporal effect. This raises the following question: To what extent does the cross-stage difference in interactions with SRL and *Indegree* reported in Table 3 represent mere cross-time effects rather than true cross-stage effects?

Extending the model with interactions between time since launch and the two contagion variables in the two stages allows one to answer that question. (There is no need to add linear time trends since the time dummies already capture any main effect of time.) Adding those four interaction terms does not significantly improve model fit ($\Delta - 2\text{LL} = 6.35$, $p = 0.17$). The BIC strongly favors the original model ($\Delta\text{BIC} = 20.15$), though the influence from colleagues in the repeat stage increases over time ($p < 0.05$). More important, the interactions of substantive interest remain significant. Thus, even after controlling for systematic changes over time in the strength of contagion from advice/discussion ties and from colleagues, people who believe themselves to be opinion leaders are less susceptible to contagion from their advice/discussion ties in trial but not repeat, and people of middle-status are more susceptible to contagion from colleagues in repeat but not in trial (Table 4).[13]

[13] Table 4 reports a significant interaction in trial of contagion from colleagues with status squared, $\text{Ln}(\textit{Indegree} + 1)^2$, which is not present in the main model reported in Table 3. However, the extended model reported in Table 4 shows no significant interaction with status itself, and a plot like Figure 3(c) for the extended model shows no inverse-U pattern. Also, deleting the interactions of contagion from colleagues with status and status squared in trial from the extended model does not generate a significantly worse fit to the data ($\Delta - 2\text{LL} = 0.602$, 2 df, $p = 0.740$). Hence, the extended

Table 4 Model Estimates Allowing for Cross-Temporal Changes in Contagion

Variables	Trial hazard	Repeat probability
Intercept	−2.081***	−0.311
	(0.317)	(0.472)
SRL	0.127	−0.103
	(0.068)	(0.157)
Ln(*Indegree* + 1)	0.109	0.023
	(0.223)	(0.394)
Ln(*Indegree* + 1)^2	0.006	0.155
	(0.132)	(0.297)
Contagion from Dis/Ref Ties (00s)	−0.014	0.008
	(0.013)	(0.020)
Contagion from Dis/Ref Ties (00s) × *SRL*	−0.610*	0.404
	(0.259)	(0.260)
Contagion from Dis/Ref Ties (00s) × *Time*	0.112	−0.066
	(0.094)	(0.124)
Contagion from Colleagues	1.057**	−1.297
	(0.402)	(0.688)
Contagion from Colleagues × Ln(*Indegree* + 1)	0.548	2.663***
	(0.373)	(0.400)
Contagion from Colleagues × Ln(*Indegree* + 1)^2	−0.666*	−0.739**
	(0.273)	(0.248)
Contagion from Colleagues × *Time*	−0.026	0.121*
	(0.037)	(0.050)
Solo Practice	−0.038	0.510
	(0.176)	(0.302)
University/Teaching Hospital	0.217	0.997**
	(0.186)	(0.349)
Primary Care	−0.234	10[a]
	(0.308)	
Early Referral	−0.293	0.887
	(0.197)	(0.629)
Past Drug 1	0.000	0.010***
	(0.002)	(0.003)
Past Drug 2	0.006**	−0.003
	(0.002)	(0.003)
Sales Calls	0.567**	−0.239
	(0.193)	(0.377)
Endogeneity Correlation	−0.292	0.291
	(0.198)	(0.334)
Ln(q_{it-1} + 1)	—	−0.837***
		(0.186)
Random Effect Stand. Dev.	0[b]	0.486***
		(0.160)
Random Effects Covariance	0[b]	

Notes. Standard errors in parentheses. LL = −403.61, BIC = 1,290.97. The model includes several additional covariates: Monthly time dummies (16 for trial, 14 for repeat) and city dummies for LA and NYC in both equations. These estimates are not reported to avoid clutter.

[a]Dummies for Primary Care and Month 2 are perfect predictors for repeat incidence. We set their coefficients to a very large number (10); thus, the predicted repeat probability for these physician-months is essentially 1 and the observations do not affect the likelihood estimation. See §6.5.

[b]Set to zero, as in the model in Table 3.

*$p \le 0.05$; **$p \le 0.01$; ***$p \le 0.001$.

In short, the results are consistent with both reasons to expect the susceptibility to normative influence to increase as customers proceed from trial to repeat: (i) Over time, people become increasingly susceptible to normative considerations and hence to colleagues enacting and enforcing norms, and (ii) As they progress from trial to repeat, people find it more difficult to defend their deviations from colleagues' behavior, especially if they are middle-status as one

model in Table 4 does not provide evidence of middle-status conformity to colleagues' behavior in trial.

would expect if those deviations are seen as normative transgressions.

7.3. Other Variables

Physician characteristics included as control variables do not show consistent coefficients across the adoption and repeat columns in Table 3. Sales calls accelerate adoption but not repeat behavior. Assuming that sales calls and expert peer influence are both informative, this contrast is consistent with the presence of expert peer contagion in trial only. The contrast is also consistent with evidence that pharmaceutical detailing is effective primarily as an acquisition tool rather than a retention tool (Montoya et al. 2010), and with the empirical generalization that marketing efforts such as personal selling and advertising are more effective early in the product life cycle (Albers et al. 2010, Lodish et al. 1995, Sethuraman et al. 2011). More generally, the lack of consistency in the estimates across trial and repeat supports the notion that research conclusions can vary across facets of product acceptance (Bell and Song 2007, Chandrashekaran and Sinha 1995).

7.4. Robustness Checks

IVV already reported quite a few robustness checks, but their analysis did not include contagion among co-located colleagues. As reported in the Web Appendix, our results are robust to (i) alternative specifications of contagion among colleagues, (ii) alternative specification of moderators, (iii) controlling for differences in demographics among the zip codes in which the physicians practice, (iv) controlling for lagged sales calls, (v) changing the centering of the status variable to minimize the correlation between status and its squared value, and (vi) excluding the PCPs from the trial, repeat, and control function equations.

8. Threats to Internal Validity

Our findings likely reflect genuine behavioral contagion patterns rather than confounds. Though some alternative explanations are *conceivable*, they are not *credible* given our data and analysis. Of course, this assessment is a matter of judgment and depends on the set of rival explanations one is aware of (Dawid 2013, Stanford 2006).

8.1. Instrumentation Bias

It is conceivable that the sociometric survey may have sensitized the physicians to the new drug or to their peers. Hence it may have increased the baseline prescription behavior or the susceptibility to peer influence. If that were the case, one should see an uptick in the baseline (intercept) or network contagion after the survey was administered. Extending the model with a shift after month 10 in the baselines in LA and NYC indicates that they are not systematically higher after the survey was administered (month 10) than they are before, i.e., they are actually all lower, though not significantly so with $p = 0.12$, or worse. Extending the model with a shift after month 10 in the contagion effects in LA and NYC shows that contagion from discussion/referral ties is insignificantly lower after month 10 in NYC ($p = 0.23$ or worse) and insignificantly higher after month 10 in LA ($p = 0.64$ or worse). The data do not support the presence of instrumentation bias.

8.2. Endogenous Tie Formation: Network Peers

Another concern is that contagion coefficients capture not the effect of ties on behavior but that of behavior on tie formation. For instance, if physicians with low confidence are more likely to build connections with prior adopters of the drug, then the finding that self-reported followers are more sensitive to peer influence might reflect selective tie formation rather than higher susceptibility to social contagion.

Several features of the data indicate that such endogenous tie formation is not a credible threat to internal validity. The first is the wording in the sociometric survey. The questions measuring discussion and referral ties pertained to the medical condition in general rather than the new drug specifically (IVV). The second is the correlation between SRL and the number of connections made to peers for discussion or referral, referred to by IVV as "outdegree centrality." That correlation is -0.04 (IVV, p. 205), indicating that the number of peers one reaches out to is uncorrelated with one's self-reported opinion leadership. The third feature is that the network was measured before launch in SF but after launch in LA and NYC. Whereas endogenous tie formation, in which nonadopters selectively build ties to others they know have adopted, might have affected the measured network in LA and NYC, it cannot have affected it in SF. So endogenous tie formation implies that network contagion effects are smaller in SF than in LA and NYC (ceteris paribus). Extending the model with such contrasts does not support this notion: Network contagion effects are actually larger in SF, though not significantly so in either trial ($p = 0.20$) or repeat ($p = 0.21$). Also, there is no evidence consistent with the notion that the new product's launch prompted physicians to form additional ties. There is no significant difference in the mean or distribution of the number of peer nominations made by physicians in SF versus LA and NYC jointly (t-test: $p = 0.52$; Wilcoxon rank sum test: $p = 0.39$, Two-sample Kolmogorov-Smirnov test: $p = 0.91$), in SF versus LA only (Tukey test: $p = 0.99$; Wilcoxon rank sum test: $p = 0.69$; Two-sample Kolmogorov-Smirnov test: $p = 0.70$), or in SF

versus NYC only (Tukey test: $p = 0.60$; Wilcoxon rank sum test: $p = 0.29$; Two-sample Kolmogorov-Smirnov test: $p = 0.57$). In short, the data are inconsistent with the endogenous formation of discussion or referral ties acting as a confound to our contagion findings.

8.3. Endogenous Tie Formation: Colleagues

Endogenous tie formation is not a credible threat for contagion from colleagues. First, the argument does not apply to our research setting. The threat requires that the decisions not to practice solo and to join a specific hospital or group practice rather than another are affected by the extent to which prospective colleagues (are expected to) prescribe the focal drug. The threat also requires that hospitals and group practices are more likely to invite or accept physicians whom they (fore-)see adopting the specific new drug. Both notions are too risibly farfetched to be credible. Second, the specific pattern in colleagues contagion further detracts from endogenous tie formation's credibility as a threat to internal validity (e.g., Rosenbaum 2002, pp. 209–214). Endogenous formation of collegial ties, if it were present, would operate equally across trial and repeat, but we observe different collegial contagion effects across stages ($p < 0.01$). Furthermore, endogenous tie formation cannot account for the nonmonotonic interaction we observe.

8.4. Reflection

Reflection arises when the peer behavior used to explain the behavior of a focal physician is actually caused by that same physician. This is not a credible threat, since we operationalize contagion in terms of lagged rather than current peer behavior, all physicians at risk of adoption have by definition not adopted before, and we control for lagged behavior of the focal physician in the repeat equations. Moreover, the network data are almost perfectly acyclic: Of the 204 discussion ties and 138 referral ties, only three are symmetric and these three ties form the only triad (IVV 2011a, p. 200).

8.5. Correlated Unobservables

Unobserved shocks that vary over time but are common across all physicians are controlled through time fixed effects. This leaves variance across physicians within particular months to be explained by contagion. Time-invariant unobserved differences across cities are also captured through city fixed effects. This leaves only factors that are specific to physicians and their network peers or colleagues as possible sources of bias from correlated unobservables. The latter often cause (justifiable) concern about the validity of main effects in contagion studies. However, they cannot explain our findings involving multiple dependent variables, multiple contagion variables, multiple moderators, and a nonmonotonic effect. What omitted variable(s) could account for peer contagion affecting trial but not repeat, peer contagion being significant only for those who do not consider themselves opinion leaders, and middle-status conformity in colleague contagion? Our contagion interpretation provides a coherent account for this complex pattern of findings, whereas correlated unobservables do not. Consequently, the latter are not a credible threat to validity (Cochran 1965; Hill 1965; Rosenbaum 2002, pp. 209–211; Shadish et al. 2002, p. 105).

For instance, it is likely that unobserved preferences for particular treatment options are correlated among network peers (Landon et al. 2012), but this cannot explain why network contagion is detected in trial but not repeat or why network contagion varies systematically with self-reported opinion leadership. Similarly, unobserved preferences for treatments, unobserved similarities in patient mix or unobserved constraints (e.g., the absence of the drug from a list of approved drugs) may have been correlated among colleagues. Yet that cannot account for the presence of a moderator effect by status.

8.6. Truncation Bias

Our hazard analysis of adoption timing includes all of the physicians at risk rather than only those who adopted. Thus, our contagion estimates do not suffer from upward truncation bias (Van den Bulte and Iyengar 2011).

8.7. Mere Duration Dependence in Use

Yet another concern might be that repeat incidence increases not just over time (a "period effect" already controlled for by monthly dummies) but also with the time since the physician adopted (an "age effect" not yet controlled for). If positive, such duration dependence might inflate the estimates of contagion at the repeat stage. However, controlling for how long it has been since a physician adopted does not improve model fit ($\Delta - 2\text{LL} = 0.16$) and does not affect the estimated contagion patterns in the repeat stage.

9. Discussion

We investigated the presence and nature of contagion in the acceptance of a risky prescription drug by physicians. There are three novel findings. First, there is evidence of contagion not only in trial but also in repeat. Second, who is most influential varies across stages. Physicians with high network centrality and high prescription volume are influential in trial but not repeat. In contrast, immediate colleagues, few of whom are nominated as discussion or referral partner, are influential in both trial and repeat. Third, who is most influenceable also varies across stages. For trial, it is physicians who do not consider themselves to be opinion leaders, whereas for repeat, it is those in

the middle of the status distribution as measured by network centrality.

These findings help move the research frontier from documenting whether contagion is at work to understanding how and why it is at work (Aral 2011, Godes 2011). The pattern of findings is consistent with informational social influence reducing risk in trial and normative social influence increasing conformity in repeat. Marketing scientists have emphasized the former and ignored the latter, yet our findings indicate that contagion in new product acceptance can operate in richer ways than hitherto documented.

Our work provides fresh evidence about the role of status in social contagion and new product acceptance (Van den Bulte and Joshi 2007). Specifically, our findings add to recent evidence that social status affects new product acceptance separately from self-confidence or social class (Hu and Van den Bulte 2014).

Our findings about the presence and nature of social contagion in new product repeat behavior complement and enhance recent work on the role of social contagion and social enrichment in customer retention and churn (Haenlein 2013, Nitzan and Libai 2011, Schmitt et al. 2011). Specifically, new insights into customer management may come from investigating under what conditions social status and normative considerations affect use intensity and customer churn.

Our study will also be of interest to researchers concerned about the identification of contagion effects in nonexperimental studies. We apply R.A. Fisher's advice on how to move from association to causation in observational studies: "Make your theories elaborate." The theoretically informed associations we observe involve multiple dependent variables, multiple contagion variables, multiple moderators, and a nonmonotonic effect. Those specific patterns cannot be accounted for by the standard threats to validity in contagion studies. Going beyond mere linear associations in a single facet of contagion provides empirical insights that are not only substantively richer but also methodologically stronger (e.g., Hodas and Lerman 2014).

A brief discussion of the scope conditions of our theoretical claims and empirical application seems warranted. Contagion in repeat, we contend, may occur when the product poses some significant functional, financial or normative risk even after adoption. This is likely for (i) "credence goods" for which people seek informational guidance even after personal use experience, and (ii) products, services or practices the use of which are subject to normative influence. Contagion can also exist in repeat for products and services with installed-base effects where the utility of use increases with the number of relevant other users, as shown by recent findings on contagious churn among customers of telephone providers (Haenlein 2013, Nitzan and Libai 2011). Contagion can also occur in repeat when environmental shocks raise new doubts about an accepted product (Nair et al. 2010). In short, even though our study focused on only a single drug and even though our evidence of post-adoption contagion is consistent only with normative influence, post-adoption contagion is likely to affect many more product categories than risky drugs.

Because our study was limited to a single product, corroboration in other settings would be useful. Studies covering multiple products with different risk and status characteristics and studies with a longer window extending beyond *early* repeat would be especially valuable as they could further sharpen insight into the nature of the mechanisms at work. Also, research on social learning or contagion in new product acceptance that uses a more direct measure of self-confidence than self-reported opinion leadership or self-reported market mavenship would be useful additions to this study and that by Szymanowski and Gijsbrechts (2013). Further research on the nature of colleagues contagion would also be welcome. Intraorganizational diffusion is a topic of great importance to both users and marketers, and a topic that we know too little about.

Our findings are also of interest to practitioners. Marketers should consider leveraging peer influence not only to trigger adoption but also to support subsequent repeat, at least for risky products like the one studied here. As Christakis and Fowler (2011) note, aptly targeting WOM marketing campaigns requires knowing not only who is especially influential but also who is especially influenceable. Our findings suggest that the answer to both questions may vary between trial and repeat. In-depth assessments of such differentiated targeting at trial versus repeat, using experimental (e.g., Hinz et al. 2011) or simulation designs (e.g., Aral et al. 2013, Haenlein and Libai 2013) would be of clear managerial value.

Practitioners willing to go beyond the mere operational definition of our variables and seeing value in the theoretical lens we used should also consider adapting their messaging so that considerations of perceived risk, status, and normative conformity receive different weights when trying to get prospects to adopt versus trying to get adopters to repeat.

Over the last several years, managers have come to embrace the notion that not only attracting new customers but also retaining them has a large impact on the corporation's profits and long-term value. Managers also have become increasingly keen on leveraging contagion among customers. Our results suggest that these two major endeavors in current marketing

practice are related: Not only trial but also repeat can be subject to social contagion.

Supplemental Material

Supplemental material to this paper is available at http://dx.doi.org/10.1287/mksc.2014.0888.

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References

Albers S, Mantrala MK, Sridhar S (2010) Personal selling elasticities: A meta-analysis. *J. Marketing Res.* 47:840–853.

Albert A, Anderson JA (1984) On the existence of maximum likelihood estimates in logistic regression models. *Biometrika* 71: 1–10.

Anderson GJ (1987) Prediction tests in limited dependent variable models. *J. Econometrics* 34(1–2):253–261.

Aral S (2011) Identifying social influence: A comment on opinion leadership and social contagion in new product diffusion. *Marketing Sci.* 30(2):217–223.

Aral S, Walker D (2012) Identifying influential and susceptible members of social networks. *Science* 337(6092):337–341.

Aral S, Muchnik L, Sundararajan A (2013) Engineering social contagions: Optimal network seeding in the presence of homophily. *Network Sci.* 1:125–153.

Asch SE (1956) Studies of independence and conformity: I. A minority of one against a majority. *Psych. Monographs: General Appl.* 70(9):1–70.

Baron RS, Vandello JA, Brunsman B (1996) The forgotten variable in conformity research: Impact of task importance on social influence. *J. Pers. Soc. Psychol*. 71(5):915–927.

Bearden WO, Netemeyer RG, Teel JE (1989) Measurement of consumer susceptibility to interpersonal influence. *J. Consumer Res.* 15(4):473–481.

Bell DR, Song S (2007) Neighborhood effects and trial on the Internet: Evidence from online grocery retailing. *Quant. Market. Econom.* 5:361–400.

Bicchieri C (2006) *The Grammar of Society: The Nature and Dynamics of Social Norms* (Cambridge University Press, Cambridge, UK).

Bosk CL (2003) *Forgive and Remember: Managing Medical Failure*, 2nd ed. (University of Chicago Press, Chicago).

Burt RS (1987) Social contagion and innovation: Cohesion versus structural equivalence. *Amer. J. Sociol.* 92(6):1287–1335.

Chandrashekaran M, Sinha RK (1995) Isolating the determinants of innovativeness: A split-population tobit (SPOT) duration model of timing and volume of first and repeat purchase. *J. Marketing Res.* 32:444–456.

Chintagunta PK, Goettler RL, Kim M (2012) New drug diffusion when forward-looking physicians learn from patient feedback and detailing. *J. Marketing Res.* 49:807–821.

Christakis NA, Fowler JH (2011) Contagion in prescribing behavior among networks of doctors. *Marketing Sci.* 30(2):213–216.

Cialdini RB, Trost MR (1998) Social influence: Social norms, conformity, and compliance. Gilbert DT, Fiske ST, Lindzey G, eds. *Handbook of Social Psychology*, 4th ed. Vol. 2 (McGraw-Hill, New York), 151–192.

Cialdini RB, Reno RR, Kallgren CA (1990) A focus theory of normative conduct: Recycling the concept of norms to reduce littering in public places. *J. Pers. Soc. Psychol.* 38:1015–1026.

Cochran WG (1965) The planning of observational studies of human populations. *J. Roy. Stat. Soc. (A)* 128:234–255.

Costenbader E, Valente TW (2003) The stability of centrality measures when networks are sampled. *Soc. Networks* 25:282–307.

Darley JM (1966) Fear and social comparison as determinants of conformity behavior. *J. Pers. Soc. Psychol.* 4(1):73–78.

Dawid R (2013) *String Theory and the Scientific Method* (Cambridge University Press, Cambridge, UK).

Deutsch M, Gerard HB (1955) A study of normative and informational social influences upon individual judgment. *J. Abnorm. Soc. Psychol.* 51:629–636.

DiMaggio PJ, Powell WW (1983) The iron cage revisited: Institutional isomorphism and collective rationality in organizational fields. *Amer. Sociol. Rev.* 48:147–160.

Dittes JE, Kelley HH (1956) Effects of different conditions of acceptance upon conformity to group norms. *J. Abnorm. Soc. Psychol.* 53:100–107.

Downs GW, Mohr LB (1976) Conceptual issues in the study of innovations. *Admin. Sci. Quart.* 21:704–711.

Du RY, Kamakura WA (2011) Measuring contagion in the diffusion of consumer packaged goods. *J. Marketing Res.* 48(1):28–47.

Dulleck U, Kershbamer R (2006) On doctors, mechanics, and computer specialists: The economics of credence goods. *J. Econom. Lit.* 44(1):5–42.

Fader PS, Schmittlein DC (1993) Excess behavioral loyalty for high-share brands: Deviations from the Dirichlet model for repeat purchasing. *J. Marketing Res.* 30:478–493.

Gielens K, Steenkamp JBEM (2007) Drivers of consumer acceptance of new packaged goods: An investigation across products and countries. *Int. J. Res. Marketing* 24:97–111.

Gnambs T, Batanic B (2012) The roots of interpersonal influence: A mediated moderation model for knowledge and traits as predictors of opinion leadership. *Appl. Psychol.* 62:597–618.

Godes D (2011) Invited comment on "Opinion leadership and social contagion in new product diffusion." *Marketing Sci.* 30(2): 224–229.

Godes D, Mayzlin D (2009) Firm-created word-of-mouth communication: Evidence from a field test. *Marketing Sci.* 28(4):721–739.

Goldenberg J, Han S, Lehmann DR, Hong JW (2009) The role of hubs in the adoption process. *J. Marketing* 73(2):1–13.

Haenlein M (2013) Social interactions in customer churn decisions: The impact of relationship directionality. *Int. J. Res. Marketing* 30(3):236–248.

Haenlein M, Libai B (2013) Targeting revenue leaders for a new product. *J. Marketing* 77(3):65–80.

Hahn M, Park S, Krishnamurthi L, Zoltners A (1994) Analysis of new product diffusion using a four-segment trial-repeat model. *Marketing Sci.* 13(3):224–247.

Harvey OJ, Consalvi C (1960) Status and conformity to pressures in informal groups. *J. Abnorm. Soc. Psychol.* 60:182–187.

Hill AB (1965) The environment and disease: Association or causation. *Proc. Roy. Soc. Med.* 58:295–300.

Hinz O, Skiera B, Barrot C, Becker JU (2011) Seeding strategies for viral marketing: An empirical comparison. *J. Marketing* 75(6):55–71.

Hodas NO, Lerman K (2014) The simple rules of social contagion. *Scientific Reports* 4:4343.

Hogg MA (2010) Influence and leadership. Fiske ST, Gilbert DT, Lindzey G, eds. *Handbook of Social Psychology*, 5th ed. (John Wiley, Hoboken, NJ), 1166–1207.

Holland PW (1986) Statistics and causal inference. *J. Amer. Stat. Assoc.* 81:945–960.

Hollander EP (1958) Conformity, status, and idiosyncrasy credit. *Psychol. Rev.* 65:117–127.

Hu Y, Van den Bulte C (2014) Non-monotonic status effects in new product adoption. *Marketing Sci.* 33(4):509–533.

Iyengar R, Ansari A, Gupta S (2007) A model of consumer learning for service quality and usage. *J. Marketing Res.* 44:529–544.

Iyengar R, Van den Bulte C, Valente TW (2011a) Opinion leadership and social contagion in new product diffusion. *Marketing Sci.* 30(2):195–212.

Iyengar R, Van den Bulte C, Valente TW (2011b) Further reflections on studying social influence in new product diffusion. *Marketing Sci.* 30(2):230–232.

Jacoby J (1974) The construct validity of opinion leadership. *Public Opinion Quart.* 38(1):81–89.

Janis JL (1972) *Victims of Groupthink* (Houghton-Mifflin, Boston).

Katona Z, Zubcsek PP, Sarvary M (2011) Network effects and personal influences: The diffusion of an online social network. *J. Marketing Res.* 48:425–443.

Kelman HC (1958) Compliance, identification, and internalization: Three processes of attitude change. *J. Conflict Resolution* 2(1): 51–60.

Kelman HC (2006) Interests, relationships, identities: Three central issues for individuals and groups in negotiating their environment. *Annu. Rev. Psychol.* 57:1–16.

Kennedy MY, Fiss PC (2009) Institutionalization, framing, and diffusion: The logic of TQM adoption and implementation decisions among U.S. hospitals. *Acad. Management J.* 52:897–918.

Knoke D, Burt RS (1983) Prominence. Burt RS, Minor MJ, eds. *Applied Network Analysis: A Methodological Introduction* (Sage, Beverly Hills, CA), 195–222.

Krackhardt D (1988) Predicting with networks: Nonparametric multiple-regression analysis of dyadic data. *Soc. Networks* 10:359–381.

Land KC, Deane G (1992) On the large-sample estimation of regression models with spatial or network effects terms: A two-stage least squares approach. *Sociol. Methodol.* 22:221–248.

Landon BE, Keating NL, Barnett ML, Onnela JP, Paul S, O'Malley AJ, Keegan T, Christakis NA (2012) Variation in patient-sharing networks of physicians across the United States. *J. Amer. Med. Assoc.* 308:265–273.

Lee SH, Cotte J, Noseworthy TJ (2010) The role of network centrality in the flow of consumer influence. *J. Consumer Psychol.* 20:66–77.

Levin SG, Levin SL, Meisel JB (1992) Market structure, uncertainty, and intrafirm diffusion: The case of optical scanners in grocery stores. *Rev. Econ. Stat.* 74(2):345–350.

Lewis K, Gonzalez M, Kaufman J (2012) Social selection and peer influence in an online social network. *Proc. Nat. Acad. Sci.* 109:68–72.

Libai B, Bolton R, Bügel MS, de Ruyter K, Götz O, Risselada H, Stephen AT (2010) Customer-to-customer interactions: Broadening the scope of word of mouth research. *J. Service Res.* 13:267–282.

Lien D, Rearden D (1990) A remark on "An advantage of the linear probability model over probit or logit." *Oxford B. Econ. Stat.* 52(2):223–225.

Lin DY, Wei LJ (1989) The robust inference for the cox proportional hazards model. *J. Amer. Stat. Assoc.* 84:1074–1078.

Liu Q, Gupta S (2012) A micro-level diffusion model for new drug adoption. *J. Prod. Innov. Manag.* 29(3):372–384.

Lodish LM, Abraham M, Kalmenson S, Livelsberger J, Lubetkin B, Richardson B, Stevens ME (1995) How TV-advertising works: A meta-analysis of 389 real-world split cable TV-advertising experiments. *J. Marketing Res.* 32:125–139.

Lu Y, Jerath K, Singh PV (2013) The emergence of opinion leaders in a networked online community: A dyadic model with time dynamics and a heuristic for fast estimation. *Management Sci.* 59(8):1783–1799.

Martin WC, Lueg JE (2013) Modeling word-of-mouth usage. *J. Bus. Res.* 66:801–808.

McShane BB, Bradlow ET, Berger J (2012) Visual influence and social groups. *J. Marketing Res.* 49:854–871.

Menchik DA, Meltzer CO (2010) The cultivation of esteem and retrieval of scientific knowledge in physician networks. *J. Health Soc. Behavior* 51:137–152.

Menzel H (1957) Public and private conformity under different conditions of acceptance in the group. *J. Abnorm. Psychol.* 55(3):398–402.

Molitor D, Hinz O, Wegmann S (2011) The interplay between psychometric and sociometric data and the willingness to adopt innovations. *Z. Betriebwirtsch.* 81:29–59.

Montoya R, Netzer O, Jedidi K (2010) Dynamic allocation of pharmaceutical detailing and sampling for long-term profitability. *Marketing Sci.* 29(5):909–924.

Nair HS, Manchanda P, Bhatia T (2010) Asymmetric social interactions in physician prescription behavior: The role of opinion leaders. *J. Marketing Res.* 47(5):883–895.

Nitzan I, Libai B (2011) Social effects on customer retention. *J. Marketing* 75(6):24–28.

Oksanen EH (1986) A note on observation-specific dummies and logit analysis. *J. Roy. Stat. Soc. D-Sta.* 35(4):413–416.

Oster E, Thornton R (2012) Determinants of technology adoption: Peer effects in menstrual cup take-up. *J. Eur. Econ. Assoc.* 10:1263–1293.

Parfitt JH, Collins BJK (1968) Use of consumer panels for brand-share prediction. *J. Marketing Res.* 5:131–145.

Perrin S, Spencer C (1981) Independence or conformity in the Asch experiment as a reflection of cultural and situational factors. *Brit. J. Soc. Psychol.* 20:205–209.

Phillips DJ, Zuckerman EW (2001) Middle-status conformity: Theoretical restatement and empirical demonstration in two markets. *Amer. J. Sociol.* 107:379–429.

Poirier DJ, Ruud PA (1981) On the appropriateness of endogenous switching. *J. Econometrics* 16:249–256.

Prosser H, Walley T (2006) New drug prescribing by hospital doctors: The nature and meaning of knowledge. *Soc. Sci. Med.* 62:1565–1578.

Rogers EM, Svenning L (1969) *Modernization Among Peasants: The Impact of Communication* (Holt, Rinehart and Winston, New York).

Rosenbaum PR (2002) *Observational Studies* (Springer-Verlag, New York).

Rossman G (2014) The diffusion of the legitimate and the diffusion of legitimacy. *Sociol. Sci.* 1(March):49–69.

Sauder M, Lynn F, Podolny JM (2012) Status: Insights from organizational sociology. *Annu. Rev. Sociol.* 38:267–283.

Schmitt P, Skiera B, Van den Bulte C (2011) Referral programs and customer value. *J. Marketing* 75(1):46–59.

Scott J (1996) *Stratification and Power: Structures of Class, Status and Command* (Polity Press, Cambridge, UK).

Sethuraman R, Tellis GJ, Briesch RA (2011) How well does advertising work? Generalizations from meta-analysis of brand advertising elasticities. *J. Marketing Res.* 48:457–471.

Sgourev S (2011) Leaving in droves: Exit chains in network attrition. *Sociol. Quart.* 52:421–441.

Shadish WR, Cook TD, Campbell DT (2002) *Experimental and Quasi-Experimental Designs for Generalized Causal Inference* (Houghton Mifflin, Boston).

Shih C, Venkatesh A (2004) Beyond adoption: Development and application of a use-diffusion model. *J. Marketing* 68(1):59–72.

Sinha P, Zoltners AA (2001) Sales-force decision models: Insights from 25 years of implementation. *Interfaces* 31(3 suppl):S8–S44.

Stanford PK (2006) *Exceeding Our Grasp: Science, History, and the Problem of Unconceived Alternatives* (Oxford University Press, Oxford, UK).

Struthers CA, Kalbfleisch JD (1986) Misspecified proportional hazard models. *Biometrika* 73:363–369.

Szymanowski M, Gijsbrechts E (2013) Patterns in consumption-based learning about brand quality for consumer packaged goods. *Int. J. Res. Marketing* 30:219–235.

Tolbert PS, Zucker LG (1983) Institutional sources of change in the formal structure of organizations: The diffusion of civil-service reform, 1880–1935. *Admin. Sci. Quart.* 28(1):22–39.

Train K (2003) *Discrete Choice Methods with Simulation* (Cambridge University Press, Cambridge, UK).

Trusov M, Boadapati AV, Bucklin RE (2010) Determining influential users in Internet social networks. *J. Marketing Res.* 47:643–658.
Turner JC (1991) *Social Influence* (Brooks/Cole, Pacific Grove, CA).
Van den Bulte C, Iyengar R (2011) Tricked by truncation: Spurious duration dependence and social contagion in hazard models. *Marketing Sci.* 30(2):233–248.
Van den Bulte C, Joshi YV (2007) New product diffusion with influentials and imitators. *Marketing Sci.* 26(3):400–421.
Van den Bulte C, Lilien GL (2001) Medical innovation revisited: Social contagion versus marketing effort. *Amer. J. Sociol.* 106:1409–1435.
Van den Bulte C, Stremersch S (2004) Social contagion and income heterogeneity in new product diffusion: A meta-analytic test. *Marketing Sci.* 23(4):530–544.
Warren DI (1968) Power, visibility and conformity in formal organizations. *Amer. Sociol. Rev.* 33:951–970.
Wasserman S, Faust K (1994) *Social Network Analysis: Methods and Applications* (Cambridge University Press, Cambridge, UK).
Westphal JD, Gulati R, Shortell SM (1997) Customization or conformity? An institutional and network perspective on the content and consequences of TQM adoption. *Admin. Sci. Quart.* 42: 366–394.
Wood SL, Moreau CP (2006) From fear to loathing? How emotion influences the evaluation and early use of innovations. *J. Marketing* 70(3):44–57.
Wooldridge JM (2002) *Econometric Analysis of Cross Section and Panel Data* (MIT Press, Cambridge, MA).