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Distinguishing among Mechanisms of Social Contagion in New Product Adoption: Framework and Illustration

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Report Summary

Marketers have become very keen on leveraging social influence among customers. One form of influence that has received much attention is “social contagion”—that people’s decision to adopt a new product or technology is affected by the extent to which their peers are already using it.

Research on how new products gain market acceptance is moving from investigating *whether* to *why* contagion is at work. To answer that question, one must be able to distinguish among multiple contagion mechanisms in real market settings rather than just within consumer laboratories. In this report, authors Iyengar, Van den Bulte, and Choi propose a framework to do so.

To begin, they suggest that different contagion mechanisms operate through different stimuli, through different sources or conduits, or with different strength across products, market conditions, and people. They apply this contingency-based framework to distinguish between learning about the product’s benefits and risks (“social learning”) and adherence to norms of proper behavior (“normative influence”).

Their research setting is the adoption of a new prescription drug used by physicians to treat a chronic and potentially life-threatening medical condition. Leveraging extant theory and research findings, the authors propose that social learning and normative influence operate through different sources or conduits (discussion/referral ties versus immediate colleagues) and through different stimuli (share versus number of prescriptions), and that social learning is moderated by whether a physician feels he or she can learn from others.

There are four key findings in the context of their study:

- Both immediate colleagues and discussion/referral partners (a majority of which were outside the physicians’ workplace) were sources of social contagion.
- Contagion from immediate colleagues was based on the extent to which they were committed to the new drug, as reflected by its share in their prescriptions in the category, and was not affected by their experience with the new drug as reflected by prescription volume.
- In contrast, contagion from discussion and referral partners was based on their experience with the new drug (prescription volume) rather than on their commitment to the new drug (share in category).
- The extent to which a physician feels he or she cannot learn much from others (self-reported opinion leadership) depresses contagion from discussion and referral partners, but does not affect contagion from immediate colleagues.

Those findings suggest that social influence operated through both social learning and normative influence. Understanding the broad mechanisms driving contagion has great relevance to marketing practice. The nature of the mechanism at work not only affects aggregate-level

diffusion curves, but should also inform marketing decisions about which customers to target as seeding points and which ties to activate with a particular message or appeal.

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Introduction

Marketers have become very keen on better understanding how social contagion affects customer behavior. Several studies have focused on documenting the presence of contagion in new product adoption after controlling for potential confounds such as marketing effort, customer heterogeneity, and various contextual factors (e.g., Godes and Mayzlin 2009; Goldenberg et al. 2009; Iyengar et al. 2011; Katona et al. 2011). Other research has documented contagion in behaviors after product adoption or customer acquisition, such as revenue and churn (e.g., Haenlein 2011; Nitzan and Libai 2011). As several scholars have noted, the conditions are now ready for the research frontier to move from investigating *whether* contagion is at work to *why* it occurs (Aral 2011; Chen et al. 2011; Godes 2011; Goldenberg et al. 2010; Narayan et al. 2011; Peres et al. 2010).

Social contagion, the phenomenon that the behavior of one's peers affects how likely or how much one engages in that same behavior, may occur for several reasons. In the context of new product adoption, these can be organized in five broad categories. The process may operate through (i) spreading awareness and interest, (ii) social learning leading one to change one's beliefs about the product's risks and benefits, (iii) social-normative influence increasing the legitimacy of the new product, (iv) concerns that not adopting may result in a competitive or status disadvantage, or (v) direct and indirect "network" or installed base effects (Van den Bulte and Lilien 2001). Each of these categories can be refined further. Social learning, for instance, can be driven by observation of adoption behavior, observation of post-adoption outcomes, or actual communication between adopters and prospects.

Identifying the specific mechanism(s) at work in non-experimental data requires either an astute choice of research setting to rule out all but one mechanism (e.g., Zhang 2010) or rich data

and fine-grained analysis to tease apart multiple processes, as illustrated in work by Burt (1987), Karshenas and Stoneman (1993), Hannan et al. (1995), Goolsbee and Klenow (2002), and Chen et al. (2011). Our work contributes to the latter approach.

We propose a framework for improving one's ability to distinguish among various contagion mechanisms in non-experimental studies, and we apply the framework to distinguish between social learning and normative influence in the adoption of a new drug.

The approach we propose consists of exploiting contingencies to narrow the interpretation of an effect and to distinguish among several possible mechanisms generating it. Theory often implies that different contagion mechanisms operate through different stimuli, different sources or conduits, or with different strength across products, market conditions, and people. The proposed approach is to exploit such theoretically informative contingencies. This is akin to the practice in behavioral research of using interactions to gain a sharper understanding of the meaning(s) of the main effect.

The general framework we present covers the five main contagion mechanisms, and discusses how prior research has operationalized each using specific sources or conduits, stimuli, or adopter characteristics related to susceptibility to a particular mechanism. Our own illustrative application of the framework uses all these sources of systematic variation *jointly*, but focuses on two of the five mechanisms: social-normative influence versus social learning about the new product's risks and benefits.

The distinction between normative and informational social influence introduced by Deutsch and Morgan (1955) is long established in psychology, consumer research, sociology, and economics (e.g., Bikchandani et al. 1992; Burnkrant and Cousineau 1975; DiMaggio and Powell 1983; Van den Bulte and Stremersch 2004). Social-normative influence arises from the desire of

an individual to conform to the expectations of another person or group. Social learning, in contrast, refers to social influence in which information obtained from another serves as evidence about reality and so changes one's beliefs about the true state of the world. The institutional details of our research context, the adoption of a new risky drug, are such that other contagion mechanisms besides social learning and normative influence are quite unlikely to be at work.

Applying our framework to this specific research setting and leveraging extant theory and research findings, we propose that social learning and normative influence operate through different sources or conduits (discussion/referral ties vs. immediate colleagues), through different stimuli (share vs. number of prescriptions), and that only the social learning is moderated by whether a physician feels he or she can learn from others. There are four key findings. (i) Both immediate colleagues and discussion/referral partners (a majority of which are outside one's workplace) are sources of social contagion. Accounting for both sources significantly improves the model fit over and above accounting for only one. (ii) Contagion from immediate colleagues is based on the extent to which they are committed to the new drug as reflected by its share in their prescriptions in the category, and is not affected by their experience with the new drug as reflected by prescription volume. (iii) In contrast, contagion from discussion and referral partners is better understood as being based on their experience with the new drug rather than on their commitment to the new drug. (iv) The extent to which a physician feels he or she can learn from others (self-reported opinion leadership) negatively moderates contagion from discussion and referral partners, but does not moderate contagion from immediate colleagues. These findings are robust across several model variants controlling for unobserved heterogeneity, simultaneity, and endogenous detailing.

Overall, the evidence is consistent with the notion of contagion in adoption behavior being driven both by social-normative influence through immediate colleagues and by risk reduction through discussion and referral partners. While each contingency in our framework (difference in source/conduit; difference in stimulus; difference in moderator effect), when considered in isolation, may not be a sufficiently strong identifying assumption to distinguish between social learning and normative influence, the cross-validation from combining them in a single analysis allows one to confidently interpret the evidence of two separate mechanisms at work.

Identifying Mechanisms: A Framework

Researchers have used several approaches to sharpen the identification of contagion mechanisms at work in their observational data. Besides leveraging institutional details that rule out particular alternatives or help distinguish credible from merely conceivable explanations (e.g., Zhang 2010), these approaches are very often based on the theoretically informed notion that a specific mechanism is likely to operate through specific stimuli, specific sources or conduits, or with different strength across products, market conditions, and people. The same idea can be used to distinguish among multiple mechanisms operating simultaneously. The functional form of the covariate or model can also be used to tease apart different contagion mechanisms.

Contagion in adoption behavior is typically operationalized as a network autoregressive process, which can be denoted as:

$$\text{Contagion}_{ikst} \sim f_k (\beta_{ikst} \sum_j w_{ijkst} z_{jkst}),$$

where f_k is a functional form, β_{ikst} captures person i 's susceptibility to contagion mechanism k for product s at time t , w_{ijkst} captures the presence or the strength of a conduit for influence through

process k from source j to i for product s at time t , and z_{jst} is the behavior or outcome of source j relevant for social contagion mechanism k having an effect on the adoption of product s at time t . In the great majority of studies, f_k is simply linear, and teasing apart different mechanisms is possible through analyzing systematic variation in contagion susceptibility β , conduit w , or stimulus z . We provide illustrations of each approach.

Variations in susceptibility (β)

One route to gain sharper insight into the nature of the mechanism(s) at work is to consider how various personal, group, or contextual characteristics moderate the amount of social influence. Here is an example of exploiting contextual variation. Imposing each adopter to have only one network tie to a single influencer, Nair, Manchanda and Bhatia (2010) find that physicians' volume of prescription of an established drug was influenced by that of a peer they nominated as an influencer, but only after a change in FDA policy about proper usage for the drug. The presence of detectable contagion only after an uncertainty-inducing contextual change suggests that contagion operated because of physicians' concerns to reduce the perceived functional risk of prescribing the drug (social learning) or because of concerns to reduce the social risk of not behaving according to professional standards (normative influence). Other mechanisms cannot account for the results.

In a study exploiting personal rather temporal variation in susceptibility, Iyengar, Van den Bulte and Valente (2011) find that physicians who perceive themselves to be opinion leaders are less sensitive to their discussion and referral partners' prescription behavior whereas true sociometric leaders are not differentially susceptible to contagion. This finding indicates that self-confidence rather than true expertise moderates sensitivity to contagion, which is consistent with risk reduction rather than any other mechanism. In another study exploiting variation in

susceptibility across adopters, Van den Bulte and Stremersch (2004) use Hofstede's dimensions of culture to investigate what mechanisms are likely to generate evidence of contagion in aggregate-level diffusion.

Differences in susceptibility across products can also be exploited, as illustrated in a study of the effect of publicity on sales. Berger, Sorensen, and Rasmussen (2010) find that receiving a *negative* book review in the *New York Times* boosts sales compared to not being reviewed at all, and that this effect operates only for little-known authors. This effect is hard to explain as anything but the effect of publicity on awareness, an interpretation corroborated by subsequent experimental studies. Van den Bulte and Stremersch (2004) also exploit cross-product variation and find that products with competing standards exhibit more contagion even after controlling for uncertainty avoidance. This indicates that installed base effect was one of the contagion mechanisms at work in their data.

Variations in stimulus (z)

The study of book reviews by Berger et al. (2010) also illustrates how taking into account the valence of the behavior or outcome can help identify the nature of the influence mechanism at work. Situations where negative word of mouth increases rather than decreases sales are consistent with contagion through spreading awareness but not with contagion driven by normative or competitive considerations. Social learning can also be ruled out, unless the contagion recipients consider their tastes to be opposite to those of the spreaders.

Peers' post-adoption performance is another contagion stimulus that can help identify the mechanism at work. Evidence that people or organizations react not to whether their peers have adopted a new product or practice but to whether their peers' performance outcomes have

increased following adoption points to contagion driven by social learning or competition rather than by awareness, social norms, or installed base effects. This variation-in-stimulus approach has been used by several studies in organizational sociology, often in combination with the variation-in-susceptibility approach. Haunschild and Miner (1997), for instance, find evidence of outcome-based contagion in the choice of corporate acquisition as a growth strategy by companies, after controlling for contagion from the total number of adopters and the number of adopters among similar organizations. They also find that only outcome-based contagion is moderated by outcome salience.

Variations in source identity or conduits (w_{ij})

When theory suggests that a specific mechanism involves only specific categories of peers j or only specific social ties or conduits w_{ij} , then systematic variation in the contagion effect across sources or conduits can be used to more sharply identify the nature of the contagion at work. For instance, prospective adopters concerned about high financial, functional, or health risks will put more credence in the actions of knowledgeable experts or experienced users than of others. So, the extent to which adopters with high expertise or experience are more contagious indicates that contagion is driven by belief updating through social learning (e.g., Goolsbee and Klenow 2002; Iyengar et al. 2011; Nair et al. 2010).

Systematic variation across different conduits linking potential adopters i to others j provides additional opportunities to narrow the set of possible the social mechanism(s) at work. A classic example is Burt's (1987) analysis of contagion through awareness, social learning and normative processes (which he collectively labeled social cohesion) versus contagion through competition for status. Burt's key insight was that theory implies that social cohesion mechanisms operate

between directly connected network members, whereas competition operates between “structurally equivalent” network members who are connected to the same other people in the network. Another example exploiting variation in conduits consists of using physical distance, and hence overlap in customer base, between companies to operationally distinguish between competition operating at short distance versus awareness operating at longer distance (Hannan et al. 1995).

Variations in functional form (*f*)

A fourth approach to distinguish among different contagion mechanisms in observational designs hinges on functional form assumptions and has been used very rarely so far. One example is research distinguishing between Bayesian and non-Bayesian social learning (e.g., Narayan et al. 2011). Another is the use of two-stage hazard models of adoption allowing one to distinguish contagion effects at the awareness or consideration stage from social learning, normative pressure or competitive considerations at the evaluation stage (Van den Bulte and Lilien 2009). The drawback of the functional form approach is that identification of the distinct mechanism relies on technical or parametric assumptions rather than data. However, when combined with systematic variation in susceptibility, stimuli, sources, or conduits captures through observed covariates, variation in functional form may provide further confidence about the nature of the contagion mechanisms at work.

Application

We apply the framework to distinguish between contagion driven by social learning about the new product's risks and benefits versus contagion driven by social-normative influence. To this end, we enrich the dataset originally constructed and analyzed by Iyengar et al. (2011).

Research setting

To gain insights into the specific mechanisms driving social contagion in new product diffusion, the research setting should ideally satisfy several conditions. First, the product and potential adopters must have characteristics making it theoretically justified to expect multiple contagion mechanisms to be at work. Second, data on relevant differences in susceptibility, sources, conduits, or stimuli must be available to distinguish among the mechanisms of interest. Third, other contagion mechanisms that might confound the analysis must be ruled out by the choice of the research setting or controlled for by other means. Finally, the setting, data, and analysis must be such that standard threats to validity in contagion research—biases due to truncation, simultaneity, endogenous tie formation, and omitted variables—are avoided.

We study the adoption of a prescription drug used to treat a chronic viral infection that can cause severe damage to internal organs and—if left untreated—sometimes even lead 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 regarding 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 short, there is considerable ambiguity and risk in making the decision to adopt. In such situations characterized by high risk, high complexity and low observability of results, both theory and research suggest that peers' adoption and use of the new drug may reduce the perceived risk of adoption (e.g., Hahn et al. 1994; Rogers 2003). Specifically, experts

and other knowledgeable opinion leaders are sought after to reduce such risk (e.g., Iyengar et al. 2011; Nair et al. 2010). However, just like other professionals, physicians look to their peers not only for information but also for normative guidance (e.g., Haas and Park 2010; Prosser and Walley 2006). In short, the product and potential adopters have characteristics making it theoretically justified to expect contagion through both social learning and normative influence.

Because the drug treats a chronic rather than acute condition and our data span only the first 17 months after launch, physicians using the drug cannot learn very much about its efficacy from their patients' treatment. However, prospective adopters can infer from their peers' behavior whether the latter think the drug is sufficiently attractive for use in treating patients. Hence, as in models of informational cascades (e.g., Bikchandani et al. 1992), social learning in our research setting is based on (i) observation of adoption behavior rather than post-adoption outcomes and/or (ii) adopters' inferred or stated opinions rather than actual experiences.

Our research setting allows us to model social learning and social-normative influence as operating through different stimuli, different sources or conduits, and with different strength across potential adopters. As explained in greater detail in the subsequent sections, the former mechanism is modeled as volume-weighted contagion operating over professional discussion ties and patient referral ties, whereas the latter is modeled as share-weighted contagion operating among immediate colleagues practicing at the same location. The extent to which physicians perceive themselves to be opinion leaders who have little to learn from others is the individual-level susceptibility variable that should affect one contagion process but not the other (Deutsch and Gerard 1955).

The institutional details of the research setting are such that other contagion mechanisms are very unlikely to be at work. Unlike communications products or technologies with competing

standards, the effectiveness of the drug is independent of the size of the installed base. Since the product was adequately supported by marketing and the decision to adopt is characterized by high ambiguity and risk, contagion is very unlikely to take place at the awareness stage (Godes 2011). There is no reason for market competition to drive contagion either. Since there was no direct to consumer advertising, new patients were unlikely to be aware of the new drug prior to seeking treatment and to threaten to consult another physician unless they were put on the new drug. Since there was no clear evidence of superior long-term efficacy, well-informed existing patients were also quite unlikely to threaten to seek another physician unless they were put on the new drug. If contagion among immediate colleagues was competitively rather than normatively induced, then the prescription volume of those colleagues would matter more than their share of prescriptions allocated to the new drug, and robustness checks show the reverse holds.

Finally, the data and analysis we use are free of the four standard threats to validity. We avoid truncation bias by estimating the model on all physicians in the sampled risk set, not just those who adopted. Simultaneity bias due to reflection and endogenous autoregression is not an issue because the network data do not exhibit symmetry or higher-order cyclicity (Ord 1975). Moreover, simultaneous adoptions are very rare in the data: Only three physicians adopt in the same month as one or more of their immediate colleagues do, and only four adopt at the same time as one or more of their discussion/referral partners do. Though we model contagion with a temporal lag in the main analysis, robustness checks show that allowing for contemporaneous contagion does not affect the substantive findings. The third bias, endogenous tie formation, is not a credible threat in our setting. Unlike studies of contagion in adolescents' smoking or drug use where friendship ties may be based on use behavior (e.g., Mercken et al. 2010; Moody et al. 2011), physicians do not decide where to practice or who to refer patients to based on their peers'

use of one very specific drug for one very specific ailment. The fourth bias, due to omitted variables, is avoided by including a non-parametric baseline hazard (e.g., Lin and Wei 1989; Struthers and Kalbfleisch 1986) and an extensive set of control variables. Robustness checks show no evidence of unobservable heterogeneity.

Next, we discuss how we operationalize the variations in susceptibility, source identity/ conduits and stimulus in our research setting to distinguish between social learning and normative influence.

Variation in susceptibility to social learning

In their seminal paper, Deutsch and Morgan (1955) distinguished between informational influence where the goal is to reduce uncertainty when trying to make accurate and valid judgments, and normative influence where the concern is to seek social approval from others or social harmony with others. Both types of influence can operate through verbal communication as well as mere observation of peer behavior. Deutsch and Morgan posited, but did not test, that the less an individual is confident in the correctness of the judgment of others, the less susceptible he is to informational social influence in making his judgment. The rationale is obvious: If the reason that a person is influenced by others is that he turns to them for information in order to help him make a difficult decision or judgment under uncertainty, then he will do so only to the extent that he feels the others are actually correct in their assessment. Importantly, Deutsch and Morgan did not make a similar conjecture for normative influence. Subsequent psychological or sociological theory does not provide the basis for any such conjecture either. In the context of new product adoption, this implies that social learning will be positively moderated by the perception that one can learn from others, whereas normative influence will not be moderated. In short, here is an individual-level trait that theory suggests is

related to variation in the susceptibility to one contagion mechanism but not the other, and so can distinguish between the two.

Variation in sources or conduits: Immediate colleagues vs. network contacts

The notion that a person keen to learn about the pros and cons of a complex and risky new product turns to others for information and advice is well accepted. When contagion operates through such social learning, experts and other knowledgeable peers are the most relevant contagion sources, and ties of discussion and advice are the most relevant conduits (e.g., Coleman et al. 1966; Iyengar et al. 2011; Nair et al. 2010; Rogers 2003).

Social-normative influence, in contrast, typically originates from other social sources and through different conduits. Researchers have long recognized that social-normative influence is more pronounced among individuals forming a group than among individuals who do not compose a group (Deutsch and Gerard 1955). Indeed, some even restrict the concept of norms to operate only among group members (e.g., Cialdini and Trost 1998).

Social norms are often local, which may stem from the tendency to be influenced most heavily by those who are closest in physical space because they are observed more often (Cialdini and Trost 1998). The enforcement of norms is often local as well (e.g., Deutsch and Gerard 1955; Fine 2001; Munshi and Myaux 2006). The susceptibility to norms tends to be locally patterned as well. One reason is that the enforcement is local, as just noted. Another is that people are keen to conform to the expectations of those they want to maintain a satisfactory relationship with and such people tend to be nearby rather than far away (Cialdini and Trost 1998). In contrast, apart from the fact that group members may have more source credibility because they are perceived to be more benevolent or more representative of one's own situation,

“there is no reason to expect differential susceptibility to informational social influence among group and nongroup members” (Deutsch and Gerard 1955, p. 629). So, because the way norms are formed, enforced, and cared about has a strong local component, being in tune with local peers is more important than being in tune with people outside one’s own immediate community or work environment.¹

That social learning and normative influence involve different sources or conduits is also implied by social network theory. Social norms are enforced most effectively in tightly clustered groups where everyone is interconnected, whereas non-redundant and hence informative new data critical for social learning tends to come from sources that are not tightly clustered (Burt 2005; Coleman 1990). So, to the extent that local ties are transitive (e.g., colleagues of my colleagues are my colleagues) and hence local ego-network are tightly clustered (e.g., all my colleagues are colleagues of each other), network theory implies that social-normative influence will operate most effectively within specific locales.

In short, theory implies that social learning operates mostly through peers one turns to for discussion and advice whereas social-normative influence operates most strongly through immediate colleagues. Though both social learning and norms operate through conversations, this variation in sources of influence provides a second means to distinguish between these two contagion mechanisms.

Of course, immediate colleagues might also generate social learning and non-colleague peers may also generate normative influence. The former is explicitly accounted for by the data: the behavior of immediate colleagues nominated as discussion or advice partners for the treatment of the focal medical condition does enter into the social learning covariate. The latter notion, that

¹ There is no theoretical reason to expect that local social norms coincide with best practices based on clinical evidence. This may explain why “differences in social norms among local physicians” have been advanced as a likely driver of the great geographical variation in medical treatments within the U.S. (Rosenthal 2012).

non-colleagues also generate normative influence cannot be ruled out a priori, but empirical research indicates that such normative influence is weaker than that from immediate colleagues. More importantly, our identification relies not only on the mapping of mechanisms into sources/conduits but also on the presence or absence of a moderating effect of self-confidence. The mapping is cross-validated even further by incorporating a third source of systematic variation.

Variation in stimulus: Volume vs. share

A person with extensive experience with a product is a more valuable source of information about the benefits of that product than someone with less experience. Similarly, someone who has been using the product is likely to have more positive information to share than someone who tried the product but has not been using it recently. Hence, if contagion operates through social learning, then a contagion source's positive influence should increase with the extent of the source's product usage (Godes and Mayzlin 2009; Iyengar et al. 2011).

The amount of normative influence that prior adopters exert, in contrast, is more likely to vary with their use commitment rather than with their use volume, or with their "share of wallet" rather than their "wallet size." Normative influence is triggered by normative beliefs, i.e. "the person's perception that most people who are important to him think he should or should not perform the behavior in question" (Fishbein and Ajzen 1975, p. 302). But how is one to infer how strongly others believe that using a particular new product is appropriate? How is one to infer others' opinion about the appropriateness of the new product? Whether they use it themselves is obviously diagnostic, but even more so is the extent to which they use that product *exclusively*. Since norms are shared expectations about how all group members ought to behave,

people can use the consistency of peers' behavior to infer the certainty or conviction of their attitude (Petrocelli et al. 2010), which in turn is expected to vary with the strength of their normative beliefs. In other words, in situations of free choice, the consistency and commitment with which someone behaves in a particular way is an indication of the strength of that person's belief that it is proper behavior.

Hence, the distinction between use volume versus use share as the contagion stimulus provides a third means to distinguish between social learning and normative conformity. Note, the effectiveness of this approach hinges on the auxiliary assumption that people infer others' normative beliefs from the consistency of their actions. For this reason, it may be cautious to use the distinction between volume and share not as the main contrast but as a complementary contrast, i.e., as a triangulation for the variation in susceptibility and contagion sources.

Data

We track the adoption of the new drug by physicians in Los Angeles (LA), New York City (NYC) and San Francisco (SF) over a period of 17 months starting from the launch time onwards. As the drug was the third entry in its category, the relevant population within each city was defined as every physician who had prescribed at least one of the other two drugs in the two years prior to the focal drug's launch. Hence, in each city, the relevant population was bounded based on both a positional criterion, being a physician practicing in one of a specific set of 5-digit ZIP codes, and an event criterion, having prescribed related drugs in the past.

Our data consists of (i) monthly physician prescription data, (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 in the population at risk practiced, (iv) U.S. Census data on local market characteristics, and (v)

company records on sales calls to each physician. Compared to the prior analysis by Iyengar et al. (2011), data items (iii) and (iv) are new and make it possible to construct several new covariates distinguishing between contagion mechanisms and controlling for contextual effects.

Prescription data

For each physician within the network boundary (not only respondents), the time of adoption is measured using monthly individual-level prescription data from IMS Health. Of the 193 doctors who responded to the survey, 68 or 35% adopted within 17 months. Data on post-adoption prescriptions are available as well. We also have prescription data for the two other drugs in the category for two years prior to the launch of the focal drug.

Discussion and referral ties

A mail and Internet survey was administered to all physicians in the relevant population. The survey asked each physician to name up to 8 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 6 and that of referral partners was 5. Both these values are below the maximum number of nominations allowed.

67 of the 150 physicians in the population of interest in SF responded. 57 out of 197 did in LA, and 69 out of 284 in NYC. The response rates, between 24% and 45%, are high enough not to produce any sizable bias in our network-based covariates and there is no evidence of non-response bias (Iyengar et al. 2011). We discuss this in more detail after describing the covariates.

Physicians who were nominated by survey respondents but who did not meet the positional or event criterion to be considered part of the population of interest were excluded from the study. Physicians who were part of the population of interest but did not respond to the survey, in contrast, were included in the set of potential discussion or referral partners. This resulted in rectangular “discussion” and “referral” network matrices for each city, with respondents as rows and all relevant population members as columns and with the (i,j) th cell being 1 when i cited j and 0 otherwise. Both matrices were then added to form a “total” network matrix of both discussion and referral for each city. This implies that a physician who is both a discussion and a referral partner is twice as influential as one who is only one or the other.

The data collected restrict the relevant networks to physicians practicing in specific zip code areas and thus capture local networks. The importance of local as opposed to national opinion leaders is well documented in the modern medical literature (e.g., Doumit et al. 2007; Keating et al. 2007; Kuo et al. 1998). Whereas nationally reputed “expert opinion leaders” may be respected for their research, to most physicians they are much less representative than local “peer opinion leaders” who are members of their own community and face similar patients and working conditions (Locock et al. 2001). The pharmaceutical industry is keenly aware of the importance of such social dynamics at the local level. Better understanding local opinion leadership dynamics was the main motivation of the pharmaceutical company making this study possible.

Since the three cities we study are major metropolitan areas, the local networks also contain several national opinion leaders. That the physicians who the company considered to be national opinion leaders also emerged as opinion leaders within their city made the network data fully credible to the managers.

Ties among immediate colleagues

We use the group practice or hospital where each physician in the relevant population practices to identify his or her immediate colleagues. For each city, we create a rectangular physician-by-physician joint affiliation matrix with respondents as rows and all population members as columns and with the (i,j) th cell being 1 when physician i and physician j are immediate colleagues and 0 otherwise.

The ties to immediate colleagues form a different network of connections than the ties to discussion and referral partners. First, ties to immediate colleagues are by definition symmetric, whereas the discussion and referral ties are highly asymmetric. Only 3 of the 204 discussion ties among survey respondents, and only 3 of the 138 referral ties among survey respondents are symmetric. Of the 234 “total” ties, only 3 are symmetric. Second, the network of immediate colleagues exhibits perfect local clustering: if someone has more than one colleague, then all of them are also each other’s colleagues. Finally, the network of immediate colleagues also exhibits perfect structural isomorphism: Two physician connected through a collegial tie have exactly the same pattern of ties to similarly positioned peers in the collegial network. Network theory and research indicate that structural isomorphism and local clustering are important in social-normative influence processes (Coleman 1990; Van den Bulte and Wuyts 2007). The discussion and referral networks in each city do not exhibit any of those tendencies, though extra-dyadic patterns may partly be masked by imperfect response.

The most compelling evidence that ties to immediate colleagues form a different pattern of connections than the ties to discussion and referral partners comes from direct dyadic comparisons of the presence of absence of each type of tie between the same two physicians. Only a small fraction of collegial ties are matched by discussion or referral ties. As shown in the

top row of Table 1, only about 13% of all collegial ties between two physicians in SF are matched by a discussion or advice tie. (Note that Tables and Figures follow References throughout.) Though this incidence rate of discussion and referral ties between immediate colleagues is quite low, quadratic assignment procedure (QAP) regression (e.g., Dekker et al. 2007; Krackhardt 1988) shows that it is significantly higher than that to other physicians in one's city ($p < .01$). In other words, though physicians referred patients with the focal medical condition or discussed its clinical management with only a small fraction of their immediate colleagues, such referral and discussion ties were still more likely to exist among immediate colleagues than among non-colleagues once one controls for the fact that many more non-colleagues are available.

For the sake of completeness, Table 2 reports what fraction of referral or discussion ties are also collegial ties. Once again, the evidence is clear that ties among immediate colleagues indeed form a different network of connections than the ties among discussion and referral partners.

Contagion variables

We model social contagion as the effect of exposure to prior adopters, and do so using lagged endogenous autoregressive terms. The extent to which physician i is exposed at time t to social influence prior adopters through conduit k is captured through the term $\sum_j w_{ijk} z_{jkt-1}$ where w_{ijk} captures how well connected each physician j is to i for influence through conduit k , and z_{jkt-1} is the relevant behavior (stimulus) of j for the social contagion process operating through conduit k at time $t-1$.

Contagion through discussion and referral ties. 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, and q_{jt-1} is the number of prescriptions written by j at time $t-1$. In both the referral and the discussion network, w_{ij1} equals 1 if i nominates j and it equals 0 otherwise. In the total network, the weights are simply the sum of the referral and discussion weights. Unless noted otherwise, all analyses use this total network only.

Setting $z_{j1t-1} = q_{jt-1}$ defines influence from discussion and referral partners as volume-weighted contagion. It captures exposure to risk-reducing information better than simply being connected to prior adopters: The more a physician's network contacts have prescribed the drug recently, especially in high volumes, the more credible their input is and hence the more confident the physician feels that adopting the drug may help her own patients.

Contagion from immediate colleagues. The extent to which physician i is exposed at time t to influence from immediate colleagues is captured through the term $\sum_j w_{ij2} s_{jt-1}$ 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 which included two older drugs besides the focal drug.

Setting $z_{j2t-1} = s_{jt-1}$ defines influence from immediate colleagues as share-weighted contagion. We do so because it captures exposure to colleagues strongly committed to the new drug better than simply having colleagues who prescribe the drug. A colleague treating 5 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.

Susceptibility to social learning

Self-reported Leadership (SRL) is used to capture the variation in susceptibility to social learning, i.e., the extent to which a physician feels he or she can learn from others. The items were adapted from the widely used opinion leadership scale of Childers (1986) to our particular research setting:

- In general, do you talk to others doctors about X? (Never/Very often)
- When you talk to your colleagues about X do you (Offer very little information/Offer a great deal of information)?
- During the past 6 months, how many physicians have you instructed about ways to treat X? (Instructed no one/Instructed multiple physicians);
- Compared to your circle of colleagues, how likely are you to be asked about ways to treat X? (Not at all likely to be asked/Very likely to be asked);
- In discussions of X, which of the following happens more often? (Your colleagues tell you about treatments/You tell your colleagues about treatments);
- In general, when you think about your professional interactions with colleagues, are you ... (Not used as a source of advice/Often used as a source of advice).

In these items, each measured on a seven point scale, “X” stands for the medical condition treated by the focal drug. The scale reliability was quite high (Cronbach $\alpha = 0.88$), and factor analysis confirmed the metric validity of the scale. We construct the SRL variable by taking the average of the six items.

Note that the first two 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. As a result, *SRL* should moderate contagion through social learning. Perceiving others to be less

knowledgeable than oneself, however, is distinct from disregarding social norms, so there is no reason to expect SRL to moderate contagion through normative influence (Deutsch and Gerard 1955). Hence, to gain sharper insights into the contagion processes operating through each conduit, we test whether the effect of each contagion variable is moderated by *SRL*. To avoid confounds in the interpretation of these interactions, we also include interactions of the two contagion variables with one's centrality in the network.

Other covariates

Indegree is the number of nominations a physician received from other physicians and can be computed separately for the referral, discussion, and total network. Unless noted otherwise, all analyses use this Indegree in the total network only. Indegree is the most basic measure of status or prestige in a network, and is a sociometric measure of opinion leadership.

We control for several other physician characteristics which might be associated with early adoption. *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, Drug 1 and Drug 2, during the twelve months prior to 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 or not. *Early Referral* is a dummy variable taking the value 1 if the physician reports sometimes referring patients to other doctors before initiating any treatment, and 0 otherwise. A doctor referring patients even before starting any treatment is less likely to adopt the focal drug early. *Primary Care* is a dummy variable capturing whether the doctor is a primary care physician rather than a

specialist more likely to focus on the relevant medical condition (internal medicine, gastroenterologists, and infectious diseases).

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

Percent below Poverty Level is the fraction of households below the poverty level, measured at the zip code level, based on the 2000 US Census. This is a relevant control variable but its effect is not clear *a priori*: One of the main causes of contracting the medical condition is more prevalent among poor people, yet they are less likely to seek and obtain costly treatment.

Percent Asians is the fraction of Asians at a zip code level, based on the 2000 US Census. This is a relevant control variable because the medical condition is more prevalent among Asians. Since we are interested not only in contagion operating over an entire city-specific network but also contagion operating within specific locales, it is important to control for such within-city spatial heterogeneity. There is no clinical research evidence that any other patient characteristics interact with drug efficacy.

Detailing Stock is a depreciation-adjusted stock measure of monthly physician-level detailing (sales calls) for the focal drug. Let D_{it} be the amount of detailing received by physician i in month t . The Detailing Stock of physician i for month t (DS_{it}) is then defined as follows:

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

where δ is the monthly carry-over rate bounded between 0 and 1, and Detailing Stock in month 1 is the amount of detailing in that month. The carry-over parameter δ is estimated jointly with the vector of slope parameters β using standard maximum likelihood. To control for a potential confound in the interaction between contagion and the leadership variables, we allow the effect

of marketing effort to be moderated by Indegree and Self-reported Leadership. There was only very limited medical journal advertising and no direct-to-consumer advertising. There was no sampling either, because of the chronic nature of the therapy and major concerns of patients developing resistance after taking a sample but not continuing on the drug.

Time dummies for each period capture the effect of any system-wide time-varying factor, such as changes in disease prevalence or the appearance of new clinical evidence. The dummies capture all cross-temporal variation in the mean tendency to adopt, leaving only variance across physicians within particular months to be explained by contagion. As a result, including the dummies provides a stringent test for the presence of contagion. It also provides a nonparametric control for duration dependence which absorbs much of the effects of possible unobserved heterogeneity in hazard models (e.g., Lin and Wei 1989; Struthers and Kalbfleisch 1986).

Final data set

Data on past prescription of the two other drugs introduced earlier are missing for 8 doctors, 3 of whom had adopted the focal drug. We delete these 8 physicians from the dataset and base our analyses on data from 185 doctors, 65 of whom had adopted the focal drug after 17 months.

We organize the data set as a panel from which all post-adoption observations are deleted since they do not contribute to the likelihood function of hazard models. Table 3 presents the descriptive statistics for these data.

Data validity under incomplete sampling

Respondents vs. non-respondents

The 185 respondents were not significantly different ($p > .05$) from the 411 non-respondents on time-invariant characteristics of focal interest that we observe for both groups²: The amount of prescription of two other drugs in the category for twelve months prior to launch (21.4 vs. 15.6 for Drug 1; 21.4 vs. 20.4 for Drug 2) and sociometric leadership (Total Indegree: 0.95 vs. 0.49). Nor were any of those variables associated significantly with the probability of responding after controlling for city in a multivariate test ($p > .05$). Hence, there is no evidence of response bias based on usage or sociometric status.

Validity of network measures

Non-response raises some special concerns in network studies since variables of interest are measured not only on the respondents included in the analysis but also on their connections to non-respondents. We discuss to what extent response rates of 24%-45% affect our network variables.

Contagion variables. For all responding physicians i whose adoption we are modeling, we observe both their outgoing ties (w_{ij}) and the prescriptions of all their connections in the network (z_{jt-1}). Hence, the variables of social contagion $\sum_j w_{ij} z_{jt-1}$ are not affected by non-response.

Indegree. The number of nominations received from others is not based on the respondents' own reports, but on reports from others. As a result, the measurement quality of respondents' indegree can be affected by the response rate. The question, then, is whether random node sampling rates of 24%-45% preserve the concordance of the indegree metric between the true (complete) and the measured (sampled) network. The answer is yes. Studies of sampling in both

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

real and simulated networks show that indegree values computed from 20% and 40% random samples of nodes tend to correlate quite highly with the values one would have obtained from the full network (Costenbader and Valente 2003; Kim and Jeong 2007; Leskovec and Faloutsos 2006; McCarty et al. 2007).

Model

We operationalize the time of adoption as the time of first prescription, and model the discrete-time hazard of adoption as:

$$P(y_{it} = 1 \mid y_{it-1} = 0) = F(x_{it}\beta) \quad (2)$$

where y_{it} indicates adoption, x_{it} is a row vector of covariates, β is a column vector of parameters to be estimated, and F is the logistic cumulative distribution function. Since the data set includes only physicians who had prescribed within the category at least once in the two years prior to the focal drug's launch, we consider each and every physician to be at risk of adopting the new drug, and express the log likelihood function as:

$$LL = \sum_{i=1}^N \sum_{t=1}^{T_i} y_{it} \ln[F(x_{it}\beta)] + [1 - y_{it}] \ln[1 - F(x_{it}\beta)] \quad (3)$$

where T_i is the number of monthly observations on physician i and $N = 185$ is the number of physicians. This is the standard log likelihood function for a discrete-time hazard model and does not suffer from truncation bias generating spurious contagion (Allison 1982; Van den Bulte and Iyengar 2011).

Results

Main analysis

Table 4 reports the estimates for the main model including contagion through both conduits, and for two nested models including contagion through only one. The Indegree and SRL variables are mean-centered, so the coefficients of the linear effects of contagion and detailing pertain to the average of the 185 physicians whose time to adoption we analyze.

The main model indicates the presence of contagion operating through both conduits, with one showing a significant main effect and the other showing a significant moderator effect. Also, accounting for contagion through both conduits simultaneously fits the adoption data significantly better than accounting only for contagion through immediate colleagues ($\chi^2 = 9.86$, $p = 0.02$) or through discussion and referral ties ($\chi^2 = 18.84$, $p < 0.01$). Thus, contagion based on discussion and referral ties significantly improves model fit over and above contagion from colleagues, and vice versa. This supports the notion that modeling contagion through each conduit provides additional information.

The pattern of interactions provides insight into the specific *mechanism* operating through each conduit. As in the prior analysis by Iyengar et al. (2011), there is evidence of volume contagion through discussion and referral ties that is moderated by SRL but not Indegree. Also, both SRL and Indegree are significantly associated with early adoption. Figure 1 plots how this contagion effect varies by SRL, and overlays this with the histogram of SRL. The effect is positive for all physicians with a Self-reported Leadership score of 5.0 or lower, which amounts to 65% of the physicians analyzed. Additional analysis indicates that the estimated effect of volume contagion on adoption is significantly positive at 95% confidence for physicians with a Self-reported Leadership score of 4.1 or lower, which corresponds to the bottom 41% of the

distribution. The estimated effect is significantly negative only for physicians with a Self-reported Leadership score of 6.4 or higher which amounts to a mere 6% of the distribution. This pattern is consistent with the notions that volume contagion through discussion and referral ties represents social learning that reduces the perceived ambiguity and risk in adopting this new drug, and that SRL captures how little physicians feel they can learn from others.

Contagion from immediate colleagues exhibits a markedly different pattern. It has a significant main effect and is not moderated by either Indegree or SRL. This pattern is consistent with the notion that share contagion from immediate colleagues practicing in the same location captures social-normative influence rather than social learning about the product's risks and benefits.

Additional analysis with “mismatched” models

Further insight into the specific mechanisms operating through each conduit comes from assessing how the results change if we impose collegial ties to convey volume rather than share contagion, and discussion and referral ties to convey share rather than volume contagion. If collegial ties are the relevant conduit (w_{ij}) and colleagues' commitment to the new drug is the relevant stimulus (z_{jt-1}) for social-normative influence, and if a similar mapping holds among discussion and referral ties, use volume, and social learning, then a model with “mismatched” conduits and stimuli should fit worse than our main model.

Table 5 reports the key information for such models with “mismatched” conduits and stimuli. The models differ only in how the contagion variables were constructed, and feature the entire set of control variables reported in Table 4. The first row in Table 5 reports the difference in deviance between the main model with matched conduits and stimuli (Column 1 in Table 4) and

each mismatched model. The main model with share contagion from colleagues and volume contagion from discussion and referral fits better than the alternatives. Since all models have the same number of parameters, the difference in deviance equals the difference in BIC. The three differences are larger than 2.5, which is sufficient to favor the main model with matched conduits and stimuli (Raftery 1995).

Since share and volume contagion have different scales, one cannot compare the effect sizes of variables based on different stimuli. The significance test, however, are robust to scaling. Of particular interest is the pattern in row 2: Contagion from colleagues is significant only for share but not for volume. This clearly suggests that contagion from immediate colleagues is based on the extent to which they are committed to the new drug as reflected in its share in their prescriptions, and is not based on their experience with the new drug as reflected in its volume of prescription.

Conversely, the difference in deviance (row 1) between models (1) and (2) and that between models (3) and (4) show that contagion from discussion and network partners is best understood as being based on their experience with the new drug as reflected by prescription volume, rather than based on their commitment to the new drug as reflected by prescription share. Also noteworthy is that contagion from immediate colleagues is never moderated by SRL (row 3), whereas contagion from discussion and referral partners always is (row 4). This further strengthens the evidence that the mechanism operating through each conduit is different.

Using nested model tests rather than fit comparisons between non-nested models shown in Table 5 leads to the same substantive conclusions. The main model (Column 1 in Tables 4 and 5) contains share-weighted Colleagues Contagion and volume-weighted Discussion & Referral Contagion. Extending this model with the two contagion variables with mismatched conduits and

stimuli (i.e., volume weighted Colleagues Contagion and share weighted Discussion & Referral Contagion) does not significantly improve the model fit ($\Delta -2LL = 0.30$, $df = 2$). Allowing the two mismatched contagion variables to interact with Indegree and Self-reported Leadership does not significantly improve model fit either ($\Delta -2LL = 2.80$; $df = 4$).

Non-contagion effects

A brief discussion of the effects of the non-contagion variables is warranted. Their main purpose is to avoid omitted variable bias in the contagion effects of key interest. So, their own effects are not important for our research objectives. Still, several do have a significant effect (Table 4). Both sociometric leadership (Indegree) and self-reported leadership (SRL) are associated with early adoption, as is being a heavy prescriber of Drug 2 prior to launch. Sales calls also tend to raise the odds of early adoption, even after controlling for being a heavy user.

Robustness checks

The results reported here are quite robust to several changes in model specification.

No flexible baseline hazard. As one might be concerned about the degrees of freedom taken up by the extensive set of control variables for analyzing 185 durations, we re-estimated all models in Table 5 without the flexible baseline hazard. This reduced the number of coefficients in the main model to only 23 without affecting the key insights (Table 6).

Controlling for out-of-town contacts. By restricting the relevant networks to physicians practicing in specific zip code areas, our contagion variables do not encompass each and every colleague that the survey respondents nominated. Specifically, of all the people nominated by the survey respondents, we excluded 40 physicians nominated in SF, 63 in LA and 80 in NYC. The excluded contacts received only one nomination on average, with 4 being the maximum, and

accounted for 36% of all nominations. To the extent that our network definition is overly narrow, our contagion variables do not account for all the social influence experienced by the physicians whose adoptions we model. Since this may but need not affect our results, we checked that our results are robust to distinguishing between in-town and out-of-town contacts.

We first approximated physicians' Discussion & Referral Contagion from out-of-town contacts by multiplying their number of out-of-town contacts by time, which is reasonable as the in-town Discussion & Referral Contagion variable increases linearly over time. Adding this new covariate to the main model did not significantly improve fit ($\Delta -2LL = 3.02$) or affect the results. Allowing the effect of approximated out-of-town Discussion & Referral contagion to vary as a function of Indegree and Self-reported Leadership by adding the two relevant interaction terms did not improve model fit ($\Delta -2LL = 4.04$) or affect the results either.

We next approximated Discussion & Referral Contagion from out-of-town contacts in another way. For each physician, we first divided the in-town Discussion & Referral Contagion variable by the number of in-town nominees. This normalized variable provided the contagion per nominee. We then multiplied that variable by the number of out-of-town contacts to approximate the Discussion & Referral Contagion from such contacts. Adding this new covariate to the main model did not significantly improve fit ($\Delta -2LL = 3.12$) or affect the results. Allowing the effect of approximated out-of-town Discussion & Referral contagion to vary as a function of Indegree and Self-reported Leadership by adding the two relevant interaction terms did not improve model fit ($\Delta -2LL = 5.46$) or affect the results either. In short, our findings are robust to distinguishing between within-town and out-of-town contacts.

Contemporaneous contagion. Since it is conceivable that contagion occurred within monthly periods, we also specified a model allowing for such simultaneous contagion. To this end, we

used an instrumental variable approach which protects one's estimates from endogeneity bias. We constructed the Discussion & Referral Contagion variable as $\sum_j w_{ij} q_{jt}$ and regressed it on an intercept, dummies for LA and NYC, the contagion variable lagged at $t-1$, $t-2$ and $t-3$, and the network-lagged detailing variable, i.e., the detailing to the nominees of the focal physician, at time t , $t-1$ and $t-2$ ($\sum_j w_{ij} D_{jt-k}$, for $k = 0, 1$ and 2). We then took the predicted values of this first-step regression ($R^2 = 96\%$; all coefficients significant at $p < .01$), and used them as the instrumented values for contemporaneous Discussion & Referral Contagion in the hazard model. We constructed similar instrumental variables for Colleagues Contagion variables as well. Imposing simultaneous contagion while avoiding endogeneity bias leads to a slightly worse fit ($\Delta -2LL = 1.30$) and does not change any of the substantive conclusions.

Controlling for contextual effects. Controlling for the total number of prescriptions of the other two drugs by all immediate colleagues and/or by discussion and referral partners did not improve fit significantly and did not affect any of the estimates significantly. So, there is no evidence that contagion is confounded with time-varying contextual effects affecting category-level demand. Our results are also robust to controlling for the lagged prescription volume of other physicians in same census tract but excluding one's own colleagues. Similarly, extending the main model with volume contagion from physicians beyond one's immediate colleagues while allowing the influence weights to decay with distance according to power function $w_{ij} = (d_{ij})^\alpha$ did not significantly improve model fit or affect other coefficients. So, the contagion effect of temporally lagged colleagues' behavior is not confounded with the temporally lagged effect of demand-generating correlated unobservables that are common across physicians practicing in the same neighborhood and vary over time.

Unobserved heterogeneity and serial correlation. Unobserved heterogeneity induces spurious negative duration dependence in hazard models and so may create a downward bias in the contagion effect. Adding physician-specific fixed effects in a logit or probit hazard model of adoption or of any other non-repeated event leads to biased estimates (Van den Bulte and Iyengar 2011). The same is true for adding practice-level fixed effects, since there are several practices without any physician adopting within the observation period. The fixed-effects hazard model estimators proposed by Allison and Christakis (2006) are not appropriate in hazard models of adoption featuring contagion dynamics either because the estimators do not allow for non-dichotomous covariates that trend over time. We therefore extended our model into a semi-parametric specification, featuring a flexible baseline hazard with monthly dummies and normally distributed random effects on the intercept.

The main model shows no evidence of unobserved heterogeneity. Adding individual-level, practice-level or zip code-level random effects do not provide any evidence of unobserved heterogeneity and so does not affect any coefficient. Nor is there evidence of correlated unobservables among physicians connected by discussion or referral ties, based on a Bonferroni test considering the correlation in the residuals in each of the 17 time periods. There is no evidence of within-person serial correlation either.

Controls for possible time-varying endogeneity in detailing. Allowing for time-varying endogeneity in sales calls using the number of prescriptions for the other two drugs in the previous three months as instruments leads to a slightly worse fit ($\Delta -2LL = 2.42$) and, more importantly, does not affect any of the estimates significantly.

Alternative measure of exposure to normative influence. We measure exposure to normative influence as the sum of immediate colleagues' prescription shares. This assumes that the

normative pressure increases with the number of colleagues but weighs colleagues equally regardless of their prescription volume. An alternative is to take the share of all prescriptions at each physician's location of practice. Replacing our original measure by this alternative leaves all results of substantive interest unaffected but results in a decidedly worse fit ($-2LL = 445.82$ vs. 433.58). Adding the alternative measure to the main model does not lead to a better fit ($-2LL = 433.50$ vs. 433.58). The additional covariate has no significant effect and all results of interest remain unchanged.

Conclusion

Contagion in new product adoption may occur for several reasons, including a lack of awareness through traditional marketing efforts, uncertainty about the product's risks and benefits, social-normative considerations, competitive considerations, or installed base effects. Distinguishing among various contagion mechanisms is of both theoretical and managerial interest. Doing so in observational as opposed to experimental data, however, is not easy.

We propose a framework for leveraging systematic variation in contagion susceptibility, sources, conduits, stimuli, and functional form to disentangle multiple contagion mechanisms. We use this approach to operationally distinguish between two contagion mechanisms, social learning versus normative influence, in the diffusion of a new prescription drug.

The substantive findings from the application can be summarized as follows. (i) Immediate colleagues are sources of social contagion, and so are discussion and referral partners the great majority of which are located outside one's work site. Accounting for both sources of contagion significantly improves model fit over and above accounting for only one. (ii) Contagion from immediate colleagues is based on the extent to which they are committed to the new drug as

reflected by its share in their prescriptions within the category, and is not based on their experience with the new drug as reflected by prescription volume. (iii) In contrast, contagion from discussion and referral partners is better understood as being based on their experience with the new drug rather than on their commitment to the new drug. (iv) Self-reported opinion leadership negatively moderates contagion from discussion and referral partners, but does not moderate contagion from immediate colleagues. These findings are consistent with the notion of contagion in adoption being driven both by social-normative influence through immediate colleagues and by social learning through network ties beyond one's practice.

Understanding the broad mechanisms driving contagion is of great theoretical interest (Burt 1987; Karshenas and Stoneman 1993; Young 2009). It also is of great relevance to marketing practice. The nature of the mechanism at work not only affects how aggregate-level diffusion curves vary across countries (Van den Bulte and Stremersch 2004), but should also inform marketing decisions about which customers to target as seeding points and which ties to activate using what message or appeal.

If contagion works through spreading awareness and interest, then viral campaigns could be made more effective by making the message more "buzz-worthy" by focusing on unusual, hilarious, taboo, or otherwise remarkable content or execution. Also, there would be little benefit of limiting the initial seeding to customers with above-average status, expertise, or credibility. For such campaigns, reaching far into the network is the main objective so seeding people with great indirect coverage should be effective.

If contagion operates through social learning, in contrast, then mobilizing the trust and expertise embedded in the network should be a key campaign objective. This implies focusing on experts and other trusted network members. Since source credibility matters, the influence is

likely to decay very quickly once it goes beyond direct ties. So, one would favor people with many direct ties as opposed to people with great indirect coverage, i.e., people with high degree centrality as opposed to high closeness and betweenness centrality.

Campaigns meant to leverage normative or competitive contagion would be designed differently. Members of one's own immediate group tend to have greater normative influence than experts, whereas competition is stronger between people who are connected to the same set of network neighbors than between people who are connected directly (Burt 1987). So, an astute salesperson would not only play on a different motive (e.g., fear of acting improperly vs. fear of losing business or status) but also mention different adopters (e.g., immediate colleagues vs. competitors), depending on the type of contagion process at work.

Those managerial questions of which customers to target as seeding points and which ties to activate using what message or appeal are currently the topic of intense research and debate (e.g., Berger and Schwartz 2011; Godes and Mayzlin 2009; Goldenberg et al. 2009; Hinz et al. 2011; Iyengar et al. 2011; Kitsak et al. 2010). Giving greater consideration to the type of contagion mechanism at work would help to make sense of the (often only seemingly) conflicting research findings and to use them more effectively when designing marketing campaigns. Meanwhile, we expect that the question how *different social influence mechanisms* may be operating through *different kinds of stimuli* from *different kinds of sources* over *different kinds of ties* and for *different kinds of people* will become increasingly central in new product adoption and word of mouth research.

Glossary

Hazard rate of adoption

Probability that someone who has not adopted earlier does so in the current time period.

Indegree

Number of incoming ties. In this study, the number of nominations one has received from others as someone they discuss medical treatment with or refer patients to.

New product diffusion

Process of a new product's gaining acceptance in a population or market. May but need not be affected by social contagion.

Opinion leader

Person influences others' attitudes or behavior without having formal authority over them and without being formally directed to do so.

Opinion leadership

The degree to which an individual influences others' attitudes or behavior without having formal authority over them and without being formally directed to do so.

Outdegree

Number of outgoing ties. In this study, the number of others one has nominated as people one discusses medical treatment with or refers patients to.

Self-reported opinion leadership

Opinion leadership measured by self-reports. In this study, people rated themselves on a six-item scale.

Sociometric opinion leadership

Opinion leadership measured in network terms. In this study, operationalized as indegree.

Social contagion

Phenomenon of people's behavior being influenced through exposure to others' knowledge, attitudes, or behavior. This study investigates only direct exposure. Exposure can operate through conversations, written documents, or visual observation.

Social-normative influence

Social influence due to the desire of an individual to conform to another person's or group's expectations of proper behavior on their part.

Social learning

Social influence in which information obtained from another serves as evidence about reality and so changes one's beliefs about the true state of the world. In this setting, social influence driven by learning about the new product's benefits and risks.

Tie

Link or connection between two nodes in a network.

Viral marketing

Marketing strategies or programs aimed at leveraging social contagion.

Word-of-mouth

Communication among existing or prospective customers about products, brands or companies.

In this study, social contagion by talking or writing to other customers about the new product.

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**Table 1. Density of Discussion and Referral Ties among Immediate Colleagues
(Fraction of all Colleague-Dyads that Involve a Discussion or Referral Tie)**

	San Francisco (SF)	Los Angeles (LA)	New York (NYC)
Total Network	0.135	0.064	0.084
Referral	0.049	0.026	0.017
Discussion	0.086	0.038	0.067

Note: The entries for the Total Network are the sum of the corresponding entries for discussion and referral. This is because, in each city, the total network matrix is the sum of the discussion and the referral matrix.

Table 2. Fraction of all Discussion and Referral Ties that Involve Immediate Colleagues

	San Francisco (SF)	Los Angeles (LA)	New York (NYC)
Total Network	0.157	0.044	0.126
Referral	0.139	0.046	0.058
Discussion	0.170	0.042	0.176

Note: The entries for the Total Network are a not the simple average of the corresponding entries for discussion and referral. This is because the total network matrix is the sum of the discussion and the referral matrix in each city, but the relative prevalence of each type of tie is not equal across cities.

Table 3: Descriptive Statistics and Correlations among Covariates for all At-Risk Physician-Months Observations

Variable	Mean	SD	Min	Max	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Adoption (y_{it})	0.025	0.16	0	1	1.00															
2. Detailing (Sales Calls)	0.29	0.83	0	9	.23	1.00														
3. Indegree	0.36	1.31	0	36	.25	.26	1.00													
4. Self-reported Leadership	4.25	1.29	1	7	.11	.18	.23	1.00												
5. LA Dummy	0.31	0.46	0	1	-.01	-.02	-.02	.14	1.00											
6. NYC Dummy	0.37	0.48	0	1	-.02	.04	-.02	.02	-.52	1.00										
7. Percent Asian	0.22	0.16	0.01	0.58	.01	.06	.08	-.18	-.15	-.07	1.00									
8. Percent below Poverty Level	0.13	0.08	0	0.46	-.01	.01	-.05	-.05	.03	.27	.00	1.00								
9. Solo Practice	0.39	0.49	0	1	.00	.13	-.11	-.12	-.01	.09	.16	.15	1.00							
10. University/ Teaching Hospital	0.21	0.41	0	1	.00	-.10	-.05	.07	-.12	.03	-.16	-.05	-.41	1.00						
11. Primary Care	0.13	0.34	0	1	-.05	-.10	-.08	-.24	.13	-.14	.13	-.01	-.06	-.01	1.00					
12. Early Referral	0.35	0.48	0	1	-.07	-.17	-.09	-.44	-.17	.00	.11	.15	-.05	.13	.15	1.00				
13. Past Drug 1	10.89	25.76	0	265	.25	.50	.50	.24	-.07	.15	.01	-.03	.02	-.07	-.09	-.16	1.00			
14. Past Drug 2	10.45	24.86	0	510	.25	.32	.39	.05	-.11	.09	.21	.08	.09	-.06	-.05	-.04	.53	1.00		
15. Referral and Discussion Contagion	7.88	18.49	0	178	.06	.01	.03	-.07	-.16	-.14	.06	-.11	-.08	-.10	-.02	.11	-.05	-.03	1.00	
16. Colleagues Contagion	0.09	0.35	0	4.39	.07	-.05	-.04	.03	-.07	-.08	-.07	-.05	-.08	-.05	.02	.12	-.05	-.04	.29	1.00

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

Table 4. Main Results

	Both conduits (1)	Collegial Ties (2)	Discussion & Referral Ties (3)
Variables of focal interest			
SRL	0.48* (0.20)	0.27 (0.18)	0.43* (0.18)
Indegree	0.34* (0.15)	0.36* (0.15)	0.32* (0.15)
Colleagues Contagion	1.62** (0.49)	1.43** (0.44)	
Colleagues Contagion × SRL	-0.32 (0.30)	-0.31 (0.29)	
Colleagues Contagion × Indegree	0.45 (0.51)	0.31 (0.47)	
Discussion & Referral Contagion	0.01 (0.01)		0.01 (0.007)
Discussion & Referral Contagion × SRL	-0.02** (0.006)		-0.01* (0.005)
Discussion & Referral Contagion × Indegree	0.004 (0.006)		0.001 (0.005)
Control variables			
Intercept	-4.45** (0.81)	-3.98** (0.74)	-4.03** (0.79)
LA Dummy	0.18 (0.45)	0.02 (0.42)	0.09 (0.44)
NYC Dummy	-0.26 (0.45)	-0.42 (0.41)	-0.29 (0.44)
Percent Asian	0.91 (1.12)	0.57 (1.05)	0.27 (1.09)
Percent below Poverty Level	0.78 (1.98)	0.92 (1.91)	0.74 (1.89)
Solo Practice	0.03 (0.36)	0.06 (0.35)	-0.03 (0.29)
University / Teaching Hospital	0.82 (0.43)	0.75 (0.41)	0.59 (0.41)
Primary Care	-0.59 (0.79)	-0.57 (0.77)	-0.57 (0.77)
Early Referral	-1.16* (0.49)	-1.01* (0.47)	-0.75 (0.43)
Past Drug1	0.0001 (0.004)	0.002 (0.004)	0.001 (0.004)
Past Drug2	0.01** (0.004)	0.01** (0.004)	0.01** (0.004)
Detailing Stock	0.42** (0.13)	0.40** (0.13)	0.41** (0.13)
Detailing Carry Over	0.51** (0.17)	0.47** (0.17)	0.45* (0.19)
Detailing Stock × SRL	-0.05 (0.07)	-0.04 (0.07)	-0.04 (0.07)
Detailing Stock × Indegree	-0.06 (0.04)	-0.06 (0.04)	-0.05 (0.04)
-2LL	433.58	443.44	452.42
Number of parameters	39	36	36

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

Table 5. Comparison of Main Model with Mismatched Conduits/Stimuli Combinations

Conduits	Stimuli			
	Share Volume (1)	Share Share (2)	Volume Share (3)	Volume Volume (4)
Collegial Ties Discussion and Referral Ties	-	4.88	6.46	2.76
Colleagues Contagion	1.62**	1.59**	0.03	0.04
Colleagues Contagion \times SRL	-0.32	-0.29	-0.04	-0.03
Discussion & Referral Contagion	0.01	0.34	0.54	0.01
Discussion & Referral Contagion \times SRL	-0.02**	-0.63*	-0.58*	-0.02**

Note: The model in column (1) is the main model reported in column (1) of Table 3. * indicates $p \leq 0.05$ and ** indicates $p \leq 0.01$.

Table 6. Comparison of Main Model with Mismatched Conduits/Stimuli Combinations, Excluding Time Dummies

Conduits	Stimuli			
	Share Volume (1)	Share Share (2)	Volume Share (3)	Volume Volume (4)
Collegial Ties				
Discussion and Referral Ties				
Δ -2LL	-	5.09	6.73	2.97
Colleagues Contagion	1.67**	1.61**	0.04	0.05
Colleagues Contagion \times SRL	-0.27	-0.25	-0.03	-0.03
Discussion & Referral Contagion	0.01	0.45	0.66*	0.01
Discussion & Referral Contagion \times SRL	-0.02**	-0.72*	-0.58*	-0.02**

Note: * indicates $p \leq 0.05$ and ** indicates $p \leq 0.01$.

Figure 1: Contagion through Discussion and Referral Ties is Higher for Physicians with Low Self-reported Leadership



Note: The downward sloping line shows how the effect of contagion through discussion and referral ties (Column 1, Table 4) varies with Self-Reported Leadership (SRL). The vertical bars show the histogram of SRL scores among the 185 physicians analyzed.