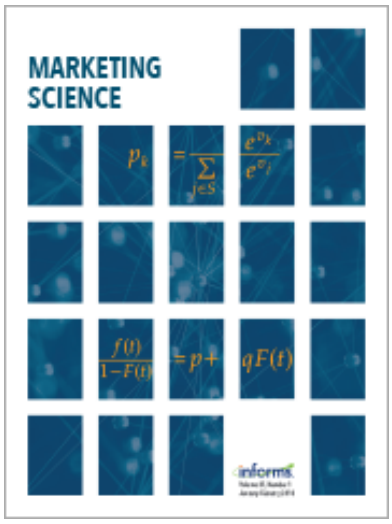

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The Role of Targeted Communication and Contagion in Product Adoption

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The two main influences leading to adoption at the individual consumer level are marketing communication and interpersonal communication. Although evidence of the effect of these two influences is abundant at the market level, there is a paucity of research documenting the simultaneous effect of both influences at the individual consumer level. Thus, the primary objective of this paper is to fill the gap in the literature by documenting the existence and magnitude of both influences at the *individual* customer level while controlling for unobserved temporal effects.

The pharmaceutical industry provides an appropriate context to study this problem. It has been conjectured that adoption and usage patterns of a new drug by physicians—"contagion"—acts as a "consumption externality," as it allows a given physician to learn about the efficacy and use of the drug. In addition, pharmaceutical companies target individual physicians via marketing activities such as detailing, sampling, and direct-to-consumer advertising. Our data contain the launch of a new drug from an important drug category. We chose two unrelated markets (Manhattan and Indianapolis) for our empirical analysis. We model an individual physician's decision to adopt a new drug in a given time period as a binary choice decision. This decision is modeled as a function of temporal trends (linear and quadratic) and individual physician-level contagion and marketing activity (both individual level and market level). Our contagion measure aggregates the adoption behavior of geographically near physicians for each physician in our sample.

Our results from the Manhattan market indicate that both targeted communication and contagion have an effect on the individual physician's adoption decision. A major challenge is to rule out alternative explanations for the detected contagion effect. We therefore carry out a series of tests and show that this effect persists even after we control for the effects of time, individual salespeople, other marketing instruments, local market effects, and the effects of some institutional factors. We believe that our contagion effect arises because the consumption externality is stronger for geographically close physicians. We discuss some underlying processes that are probably giving rise to the contagion effect we detected.

Finally, we compute the social multiplier of marketing and find it to be about 11%. We also use the estimated parameters to compare the relative effect of contagion and targeted marketing. We find that marketing plays a large (relative) role in affecting early adoption. However, the role of contagion dominates from month 4 onward and, by month 17 (or about half the duration of our data), asymptotes to about 90% of the effect.

Key words: new product adoption; social networks; social interactions; contagion; word of mouth; hierarchical Bayesian methods; pharmaceutical industry

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1. Introduction

The adoption of new products by consumers is perhaps one of the most important research problems in marketing (Hauser et al. 2006). The two main influences leading to adoption at the individual consumer level are marketing communication and interpersonal communication (usually referred to as "(social) contagion" or "word of mouth"). The effects of these two influences have typically been documented using market-level sales data over time (also referred to as the aggregate "diffusion" curve) (Bass 1969). However, there is a paucity of research on diffusion at a micro (or disaggregate) level (see Chapter 2 in Rogers 2003). Most of the extant research on diffusion

Table 1 Selective Review of Papers Investigating Social Network Effects

Paper	Product category	Data (self-reported vs. behavioral)	Level of analysis	Targeted marketing variable	Contagion/Word-of-mouth variable	Control for time effects
This paper	Pharmaceuticals	Behavioral	Individual	Yes (Detailing and sampling are set at individual level.)	Geographic proximity	Yes
Bell and Song (2007)	Internet grocery retailer	Behavioral	Region (ZIP code)	No	Geographic proximity	No
Coleman et al. (1966)	Pharmaceuticals	Self-reported + behavioral	Individual	No	Personal social network	No
Burt (1987)	Pharmaceuticals	Self-reported + behavioral	Individual	No	Personal social network	No
Strang and Tuma (1993)	Pharmaceuticals	Self-reported + behavioral	Individual	No	Personal social network	No
Van den Bulte and Lilien (2001a)	Pharmaceuticals	Self-reported + behavioral	Individual	No (Journal advertising is not individually targeted.)	Personal social network	No
Van den Bulte and Lilien (2001b)	(1) Pharmaceuticals (2) ATM adoption by U.S. banks	Self-reported + behavioral	Individual	No (Journal advertising is not individually targeted.)	(1) Personal social network (2) The proportion of banks in the local market that have already adopted	Yes
Conley and Topa (2005)	Urban unemployment	Behavioral	Individual	No	Geographic proximity	No
Bandiera and Rasul (2004)	Agricultural industry	Self-reported	Individual	No	Personal social network; geographic or cultural proximity	No
Sorensen (2006)	Health plan choice	Behavioral	Individual	No	Geographic and demographic proximity	No

(especially in economics and sociology) has focused on demonstrating that contagion exists and has generally ignored the role of marketing communication. More recent research has started to include marketing variables, though the operationalization of these variables is usually as a control (typically as an aggregate series of marketing expenditure). However, in many industries, marketing expenditure is allocated at a disaggregate level (e.g., account, territory or individual consumer) and has a differential impact on the diffusion of a product across the disaggregate units. As a result, there is not much empirical evidence that the effect of each of the two influences mentioned above is significant for individual adoption decisions when both are accounted for simultaneously. Also, many of these studies do not explicitly control for temporal effects (which act as proxies for unobserved environmental effects that affect diffusion over time). A detailed description of relevant past research can be seen in Table 1.

Thus, the primary objective of this paper is to document the existence and magnitude of contagion and marketing communication on new product diffusion.[1] We did this at the *individual* customer level while allowing for marketing communication at both disaggregate and aggregate levels—something that has typically not been done in the literature. This is important because ignoring marketing efforts at the individual level can lead to potential confounds with the individual level contagion effect. In fact, the findings from past research suggest that ignoring marketing efforts even at the aggregate level leads to inconsistent findings vis-à-vis the existence of the contagion effect.[2] In addition, the use of individual-level marketing efforts (instead of aggregate-level marketing efforts) allows us to disentangle marketing effects from temporal effects—with aggregate marketing data, this is often a problem, as an aggregate-level marketing effort is usually highly correlated with temporal effects. Therefore, we can detect the presence of contagion under a much more stringent specification relative to previous literature. Finally, our model allows us to obtain individual-level estimates (thus allowing the firm to implement targeting policies at the individual physician level). We do all this

[1] The concept of contagion has been studied in other research areas outside new product adoption by individual consumer. In an intercompany competitive setting, Debruyne and Reibstein (2005) show that a firm's decision to enter a new market is affected by contagion. Li (2005) analyzes the effect of information sharing on channel coordination under network externalities. Bronnenberg and Mela (2004) study the existence of contagion effect in retailer adoption. They show that the time to adopt a brand by a retail chain is correlated with the time to adopt for competing chains in overlapping retail trade areas.

[2] This can be seen in the results of many papers that use the data first introduced in Coleman et al. (1966).

by providing a descriptive model of how new product adoption evolves as a function of targeted marketing communication and interpersonal communication across individual consumers and over time.

Although the study of the adoption of new products at the individual level as a function of contagion can provide unique insights (as described above), it is not without challenges. The overarching challenge is to establish that contagion exists while simultaneously ruling out the alternative explanations arising from other effects that are likely to be confounded with contagion. Other challenges include the designation of other individuals who could influence the adoption behavior of the focal individual, finding the exact definition of an individual's network social (or peer) group, and ruling out the effects caused by the specific context in which the data arise (also known as "contextual effects"), as well as the effects of other variables that are correlated with the defined measure of contagion (also known as "correlational effects"). The use of our data and modeling choices is essentially guided by the objective of meeting these challenges to the extent possible with the data at hand. Specifically, we used data on the launch of a new drug in an important therapeutic category to investigate the existence and size of the contagion effect (while controlling for targeted communication directed at individual consumers as well as other institutional and temporal changes). Our data track the prescription behavior and marketing activity received by each physician as well as aggregate marketing activity for almost three years after the launch. We modeled the physician's decision to adopt the new drug at each temporal unit in the data (a month) via a binary logit model. We specified this decision as a function of current and past marketing activity, other temporal effects (e.g., overall diffusion/adoption pattern, direct-to-consumer advertising (DTCA) and physician meetings and events), and a contagion measure that is based on the adoption behavior of physicians who are geographically close; i.e., our measure is a proxy for true interpersonal communication (see Table 1 and Godes and Mayzlin 2004 for previous work that has used similar measures). Our measure of contagion is specific to each physician and is based on the lagged behavior of the physician's peer group. Finally, our modeling approach combined with the data allows us to estimate the relative effect of contagion and marketing communication over time while controlling for other unobserved temporal factors.

We chose the pharmaceutical industry for our study because it is particularly appropriate for examining individual-level adoption behavior. There are many reasons for this. First, although physicians are (understandably) interested in learning about new drugs, they are also uncertain about the efficacy of these drugs for their specific patient base. To reduce this uncertainty, physicians depend on a variety of sources, including targeted marketing communication (visits by sales force known as "detailing" and sampling) and the behavior of other physicians. Second, prior research has suggested that individual physician behavior may be affected by group behavior. The effect of this knowledge of other physicians' behavior on a specific physician's behavior has been labeled "consumption externality" (Berndt et al. 2003). Consumption externality is largely informational in nature and emerges when the use of a drug by others influences one's perceptions about its efficacy, safety, and acceptability, and thus influences one's evaluation and adoption of the drug. Third, detailed data on targeted marketing activity from the time of new product launch are typically available from industry sources. In fact, the use of this industry is a very strong test for contagion effects, as in most consumer product categories, marketing communication is not targeted at the individual consumer level.

Fourth, given the small number of physicians in the United States (for most drugs, the size of the physician networks is in the tens of thousands), it is relatively easy to construct contagion measures based on distance (either geographic or attribute). Fifth, the results documented by previous studies that have used data from this industry are mixed. Coleman et al. (1966), in a path-breaking study, found that physician-adoption decision is affected by interaction with other physicians. Using a combination of behavioral and survey data in four physician communities, the study also found that a physician's professional interactions have a larger effect on the time to adoption than social interactions. In addition, it found that physicians who are more socially connected adopt more quickly than physicians who are not. However, subsequent studies have reanalyzed these data and called the main finding—the existence of a contagion effect—into question. For example, Burt (1987) found that the contagion effect is rather small in magnitude. Strang and Tuma (1993) concluded that the contagion effect is sensitive to the model specification, and Van den Bulte and Lilien (2001a) found contagion effects disappear once journal advertising, a specific marketing instrument, is controlled for. In a subsequent paper, Van den Bulte and Lilien (2001b) modeled the adoption as a two-stage process—awareness and evaluation/adoption. Using the same data, they found that contagion has a significant effect on the second stage. One factor that contributes to these inconsistent findings is that some studies did not control for the effect of marketing communication (at either aggregate or disaggregate level) while estimating the contagion effect. In our model, we specifically control for

the effect of marketing communication at both the aggregate and disaggregate level to avoid such a bias.

Finally, from an industry point of view, new products that are introduced in this industry are actually different from existing products—e.g., a new drug is based on a new molecule. Thus, physicians and firms are very interested in understanding the efficacy of the new drug when it is launched.

Our results show that a physician's decision to adopt is influenced positively by contagion (i.e., a physician's probability of adoption increases as more physicians near him or her adopt). This finding is consistent with some of the findings in the pharmaceutical domain (cited above) as well as some other domains. For example, Bandiera and Rasul (2004) found that farmers' decisions to plant a new crop are influenced by the adoption choices of farmers in their social network of family and friends. Conley and Udry (2007) also found similar effects for farmers in Ghana deciding to adopt and use a new farming technology. Bell and Song (2007) found similar significant contagion effects (based on geographic proxies) for the adoption of an Internet grocery store at the zip code level. However, most of these studies are based on self-reported (survey) data and/or do not include contemporaneous effects of individual-level marketing, such as targeted emails and coupons in the context of Internet retailing, and targeted detailing visits and sampling in the context of the pharmaceutical industry. Our paper can therefore be seen as complementary to these existing studies.

Using the estimated parameters, we calculated the proportion of the adoption probability arising from the marketing activity relative to that arising from the contagion. Consistent with prior research (e.g., Narayanan et al. 2005), we found that marketing plays a relatively large role in affecting adoption early on. However, the role of contagion dominates from month 4 onward and, by month 17 (or about half the duration of our data), asymptotes to about 90% of the effect. We also used our results to compute the social multiplier of marketing. This multiplier may be seen as the *additional* increase on the probability of adoption arising from contagion over and above the increase based on a targeted marketing effort (detailing). Specifically, we found that this multiplier (on average) is 1.11; i.e., there is an 11% increase in the probability of adoption from contagion, over and above the direct and immediate impact of the marketing effort.

Finally, as mentioned earlier, a major challenge for studies that document social contagion using behavioral data is to rule out alternative explanations for the detected effect. We therefore carried out a series of tests to show that this effect persists even after we control for the effects of time, individual salespeople effects, local market effects, and the effects of some institutional factors.

The paper is organized as follows. Section 2 describes the data and the industry background. Section 3 describes the model formulation. Section 4 discusses the results, and §5 concludes the paper.

2. Data and Industry Background

Our data are from an important pharmaceutical category (that we cannot reveal for confidentiality reasons). The drugs in this category represent a solution to a chronic medical problem that affects a large number of patients in the United States (about 16 million, or 6% of the total population). This medical problem requires constant monitoring and management. The incidence of this problem is not affected by seasonal variables. We focused on the launch of a new drug in this therapeutic category (labeled drug X by us for the rest of this paper). Besides being based on a new molecule, drug X also tackles the medical problem in a new and unique way. Given the breakthrough nature of the drug, the uncertainty around its efficacy, side effects and interaction with other drugs are higher than for a typical new drug. It was launched in the United States in July 1999.

Our data comprise the physician level prescription and marketing effort data over a 34-month period postlaunch. These data are compiled from internal company records and IMS Health Inc.'s pharmacy audits. The total number of physicians in the United States who prescribed in this product category during our data period was 120,000. The data also contain the physical address of the primary practice location for a given physician (i.e., the physician spends the majority of his or her time at this location).[3] Besides primary care physicians (PCP), there are two major specialties that prescribe this drug (we label them as SPE). Thus, for drug X, there are two types of physicians: SPE and PCP/OTH (where OTH comprises all other specialties unrelated to the condition treated by drug X).

With the objective of delineating social networks of physicians in a manner that we can create relevant contagion measures using only behavioral data, we carried out a series of interviews with the brand and sales teams of the company that produces drug X. We found that all the company's executives firmly believed in the existence of a social contagion (word-of-mouth) effect and were interested in developing methods to measure such an effect.[4] They noted

[3] It is possible that the physicians in our sample may practice at more than one location. As we cannot explicitly control for this possibility, it remains a limitation of our approach.

[4] Although the pharmaceutical industry in general is very interested in quantifying this word-of-mouth effect, no formal measures of this exist (to the best of the authors' knowledge).

that using geographic proximity to capture the effect is reasonable, as physicians who are geographically close are likely to have more social interaction, especially of the noncommercial type (e.g., medical conferences). In addition, they noted that detailers (of this and other firms) usually highlight adoption behavior in local markets to influence physician-adoption behavior. These mechanisms are consistent with the information mechanisms that form the basis for the consumption externality. Recall that consumption externality is largely informational in nature and emerges when the use of a drug by other physicians influences a given physician's perceptions about its efficacy, safety, and overall acceptability (Berndt et al. 2003).

Our next decision was therefore to choose a geographic market for our analysis and to define the boundary of the social network based on this geography. Consistent with the previous literature[5] that has used geographic proximity to define a social network, our choice of such a geographic market is guided by the definition of the local region from the company's point of view. During the interviews with the brand and sales manager of the company, we discovered that the firm plans activities around sales areas that loosely correspond to metropolitan sales areas. Broadly speaking, physicians within each of these areas have similar patient bases and are exposed to similar levels of noncommercial/scientific and commercial (marketing) activity. For our focal analysis, we chose the New York metropolitan area as the basic geographic entity and focused our attention on the adoption behavior of physicians in Manhattan (a borough of New York). Using Manhattan has some advantages—it is geographically compact and organized and has a patient base that is less likely to be affected by insurance status (we discuss this in detail later). Note that, as reported later in the paper, we also estimated our model on another market, Indianapolis, which is quite different from Manhattan.

We restricted our analysis to the set of adopter physicians in Manhattan (we discuss the reasons for restricting our analysis to the set of adopters in a later section). As per industry practice, a physician is defined as an adopter when she writes the first prescription for drug X. This is also the definition of adoption that has been used in the previous literature (Coleman et al. 1966; Burt 1987; Strang and Tuma 1993; Van den Bulte and Lilien 2001a, b). To ensure that this definition not only captures trial-data, we checked the behavior of physicians in our sample postadoption (where adoption is defined as above). We found that 90% of the physicians continued to prescribe the drug postadoption.[6] In other words, our measure of adoption reflects the medium-term prescription behavior of physicians and not just short-term experimentation. Relative to a measure of prescription intensity postadoption, our adoption measure is also perhaps "cleaner" in terms of measuring the effects of contagion. This is because, postadoption, a physician may rely more on her experience with her own patients in terms of the decision to continue prescribing the drug.

Using the above criteria, our data comprise a set of 466 physicians. Of these physicians, the distribution across specialties is 90% and 10% for PCP/OTH and SPE, respectively. Relative to national distribution of specialties, SPE are somewhat overrepresented in the data—the national proportions are 97.8% and 2.2%. This is most likely because we include only adopters in our analysis. Table 2 provides descriptive statistics of the adoption behavior and marketing activity directed toward our set of physicians over the 34-month period.

Finally, in terms of industry practice, it is important to understand how marketing activity is allocated for product launches. The pharmaceutical firm informed us that the primary criterion used to allocate marketing resources (both pre- and postlaunch) is the category volume prescribed. Specifically, physicians are binned into deciles using this criterion, and a higher level of detailing is allocated for the heavier prescribing physicians. Although other criteria may also be used, the volume-based criterion typically explains most of the variance in detailing levels across physicians.[7] Other studies have documented the use of this rule for existing drugs (e.g., Manchanda et al.

[5] For example, to define the information neighborhood of each pineapple farmer based on geographic proximity, Conley and Udry (2007) characterize geographic neighbors of a given plot to be those within one kilometer of the geographic center of all their pineapple plots. To define the encroachment area within which customers are likely to view two same-brand hotels as substitutes, Kalnins (2004) uses the distance to the 10th, the 15th, and the 20th closest hotel from the incumbent hotel. Bucklin et al. (2008) also use the distance to the 10th closest car dealer to capture dealer concentration. Bell and Song (2007) use the zip code as their unit of analysis and use adoption patterns in an "arbitrary" geographic area (the neighboring zip codes of the focal zip code) as their contagion measure.

[6] To check that our results were not sensitive to the inclusion of the (noncontinuously prescribing) 10% of physicians, we ran the model using data from the physicians who continue to prescribe. We found there is no appreciable change in the results.

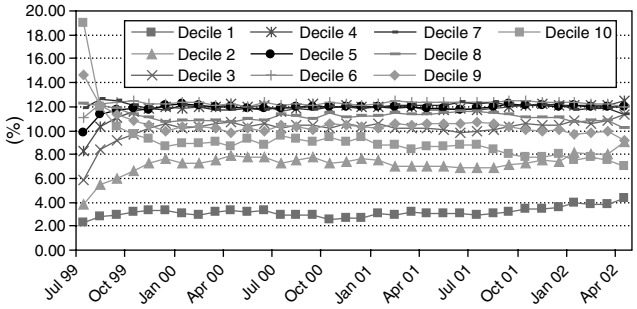
[7] The firm that provided the data confirmed that if did not target physicians on their propensity to adopt. This is mostly because for new drugs, firms do not have much knowledge (as this propensity to adopt is unobserved) on which to base this targeting. The right way for the firm to do this would be to calibrate the physician's propensity to adopt as a function of detailing by looking at her adoption pattern for multiple drugs in the same category (while controlling for other effects). However, the firm typically does not have access to these data. This is partly because launches in the same category by the firm are rare (and the firm typically does not obtain individual-level detailing data for competitive launches).

Table 2 Descriptive Statistics—Adoption Behavior and Marketing Activity

Variable	SPE $n = 46$	PCP/OTH $n = 420$	All $n = 466$
Time to adopt since the month of launch			
Mean	5.96	13.31	12.58
Standard deviation	5.09	8.39	8.41
Mode	3	4	4
Median	4	11	10
Range	2–27	2–34	2–10
Skew	2.31	0.69	0.77
Avg. number of category prescriptions in a month			
Mean	39.22	15.22	17.59
Standard deviation	26.32	17.95	20.21
Mode	28.09	1.38	1.38
Median	29.47	8.94	10.13
Range	2.65–113.56	0.24–128.85	0.24–128.85
Skew	0.94	2.45	2.13
Avg. number of monthly detailing visits			
Mean	3.58	1.18	1.42
Standard deviation	2.12	1.43	1.67
Mode	3.18	0	0
Median	3.18	0.65	0.77
Range	0–9.79	0–8.03	0–9.79
Skew	1.03	1.53	1.60
Avg. monthly samples dropped in a month			
Mean	4.78	1.44	1.77
Standard deviation	4.65	2.72	3.13
Mode	0	0	0
Median	2.81	0.37	0.59
Range	0–20.50	0–26.85	0–26.85
Skew	1.45	4.29	3.49

2004)[8] as well as new drugs (e.g., Narayanan and Manchanda 2009). We also verified this for our data. We computed the proportion of all details received by each physician decile and found that it is almost constant over time (see Figure 1)—suggesting that the firm follows the decile rule to allocate marketing activity. We then created the (category volume) deciles using data prior to launch of the drug in question, and we regressed the realized detailing on these deciles. We found that the decile classification explains 60% of the variation in the detailing allocated to the physician. However, it is possible that a physician in a higher decile is more likely to adopt. We therefore examined the adoption pattern across deciles for the first three-quarters postlaunch. As can be seen in Figure 2, the proportion of physicians adopting in each

Figure 1 Proportion of Details Received by Decile



decile is not monotonically increasing by decile. Thus, for our data, it does not seem that a higher-category volume of prescriptions written in the category is correlated with the propensity to adopt. Finally, if firms knew that certain physicians were likely to adopt because of the firm's detailing efforts, they would detail these physicians heavily till they adopted and then revert back to the average level of detailing as predicted by that physician's decile. We therefore checked to see if there is any systematic change in the number of details made to a physician postadoption. In fact, we found that although physicians were detailed somewhat more postadoption, the difference is not statistically significant. Therefore, these three factors provide some evidence that the firm did not strategically target physicians based on their propensity to adopt. Thus, this helps rule out selection bias and/or endogeneity concerns with respect to marketing activity.[9] Note that other studies (using data from different pharmaceutical firms in other therapeutic product categories) have also documented evidence for this practice when new drugs are launched (Narayanan and Manchanda 2009).

3. Model and Specification

This section describes the model, the estimation algorithm, and then the model specification.

3.1. Model and Estimation

Each physician in our model decides in each month whether to adopt drug X or not. This decision is modeled as a binary choice. The utility of adoption for physician i in month U_{it} is given by

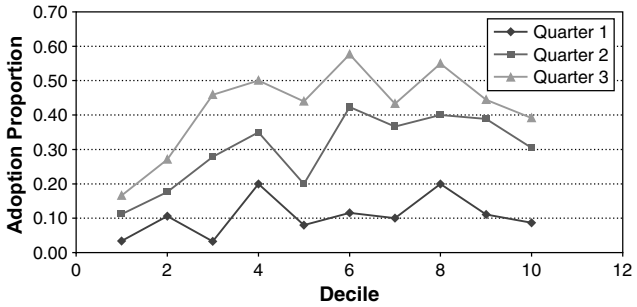
$$U_{it} = x'_{it}\beta_i + z'_t\gamma + \varepsilon_{it}.$$

Here, the xs represent variables that vary over physicians and time (e.g., marketing variables), the zs represent variables that only vary over time (e.g., time

[8] Manchanda et al. (2004) look at a product category that is mature. In that case, firms have multiple data points to calibrate response functions (typically at the segment level) and use that knowledge to allocate marketing resources. Interestingly, it is not clear that the firm gets this right; e.g., Manchanda et al. (2004) find that more (less) responsive physicians are detailed less (more).

[9] Another modeling strategy would be to use instrumental variables. However, any instrument for detailing must vary for each physician and over time. As noted in Manchanda et al. (2004), it is not clear if such an instrument exists. Exploratory analysis using lagged detailing as an instrument for detailing did not change our results in any meaningful manner.

Figure 2 Adoption Proportion by Decile



trend), and the error term ε_{it}s are assumed to follow an extreme value distribution (i.e., we use the logit model). Thus, the conditional probability, $\Pr_{it}$, that physician i adopts in month t is given by the usual expression:

$$\Pr_{it} = \frac{\exp(x'_{it}\beta_i + z'_t\gamma)}{1+\exp(x'_{it}\beta_i + z'_t\gamma)}.$$

Note that the unconditional probability, therefore, of a physician adopting in period T is given by $\Pr_{iT}\prod_{t=1}^{T-1}(1-\Pr_{it})$. Cast in a hierarchical Bayesian framework, the response coefficients, β_is, are expressed as

$$\beta_i = \beta_0 + \nu_i,$$

where $\nu_i \sim N(0, V_\beta)$.

We derived a series of full conditional distributions (detailed in the appendix) for the unknowns in our model, β_i, γ, β_0, and V_β, and sampled iteratively from each distribution until convergence was obtained.

3.2. Model Specification

We focused on four sets of variables that could influence physicians' adoption behavior. The first set consists of the targeted marketing communication variables—detailing and sampling. We allowed for both flow and stock effects of detailing. The flow variable, DET_{it}, represents the contemporaneous effect of detailing and the stock variable, $\text{DETSTK}_{it} = \text{DET}_{i(t-1)} + \lambda_D * \text{DETSTK}_{i(t-2)}$, represents the accumulated stock of detailing (Nerlove and Arrow 1962). For sampling, we used only the accumulated stock of sampling, as we did not find a significant effect of contemporaneous sampling in any of our models. We constructed the sampling stock as, $\text{SAMSTK}_{it} = \text{SAM}_{i(t-1)} + \lambda_S * \text{SAMSTK}_{i(t-2)}$. Based on our past research using pharmaceutical data and our own preliminary analyses,[10] we set both λ_D and λ_S equal to 0.7.

[10] We conducted a preliminary analysis (a grid search) over the range of parameters (between 0 and 1) to fix the carryover parameter. We used two measures, the log-likelihood and the hit-rate (i.e., proportion classified correct), across all observations in the data to make our final choice. While the log-likelihood and the hit rate do not vary much over the range from 0.6 to 0.9, it is the highest for a carryover of 0.7.

The second set of variables consists of aggregate marketing expenditure. We focused on marketing expenditure directed at patients, as they could influence physicians to adopt a drug by requesting it. When a new drug is launched, the main mechanism by which a firm informs its patients about the existence of the new drug is via DTCA (in our case, this was the only form of communication directed at consumers by the firm during the period of our data). We therefore included the monthly DTCA expenditure (measured by millions of dollars) in the model as a covariate.

The third set of variables is the set of contagion measures. Given that we had only behavioral measures and the location of each physician, we decided to construct the contagion measure on the basis of adoption behavior of a reference group of near physicians.[11] A usual problem in the literature has been to define the right reference group (for example, see the discussion in Manski 2000). In our case, this is less of a problem. We knew that, for the most part, this group consists of other physicians. Given this, we needed to come up with a defining characteristic that would allow us to create a reasonable set of near physicians. To do this, we followed the well-established practice of using geographic proximity to determine an individual's network (for a detailed discussion of the benefits and costs of using such measures, see Conley and Udry 2007; also see Conley and Topa 2007 for a comprehensive list of examples of the use of this measure). As we were interested in creating individual physician-specific contagion measures, we defined a physician's network as all other physicians in a geographic band (circle) centered at that physician's location (see Conley and Udry 2007 for another example of such a measure). We based the size of this band on our discussion with the firm, which suggested that physicians in the metropolitan sales area are likely to

[11] There are other equally valid methods such as survey or direct observation. However, each method gives rise to measures with advantages and disadvantages. For example, the geography-based measure is objective, can be easily obtained for all units in the data, and is not subject to any survey or observational bias (the benefits). But its use implies that contagion is inferred (the cost). The survey-based measure is direct (the benefit) but is hard to obtain from all units and is subject to the usual survey biases (social desirability, selective memory, representativeness, etc.) (the costs). Finally, the direct observation measure (such as the one in Godes and Mayzlin 2004) is objective and relatively easy to measure (the benefit) but is limited in its scope (e.g., there must be a point of observation), and it is hard to match up with outcomes (e.g., as Godes and Mayzlin point out, their methodology can only help to document correlations at different levels of aggregation—e.g., between aggregate, national TV ratings and individual message patterns). In conclusion, there is no clear definitive method that has emerged in the literature. Our work must therefore be seen in the spirit of using a measure based on geography to document the effect and being complementary to other work that has used different measures.

interact with each other much more than with physicians from outside the area. In other words, the consumption externality is higher in local geographies.

We therefore set the radius of the band such that this band encompasses the sales planning area of the firm for a given physician in Manhattan. This turns out to be 20 miles (it closely reflects the distance between the center of Manhattan and the city limit).[12] The choice of this specific contagion measure has advantages and disadvantages. The main advantage is that it is unique for each physician (as the band is centered on an individual physician) and is based on objective data. There are two main disadvantages. The first is that using this distance results in a fairly large band (relative to the size of Manhattan). This has the effect of reducing the variance in the contagion measure across physicians. Second, it may be more realistic to expect more physician interaction in a band that is smaller. We deal with the first disadvantage through our specification (below). For the second, we provide in a later section some evidence that our results hold for a much smaller band as well. In fact, that analysis also provides evidence for the contagion effect dissipating with distance. However, we did not use a smaller band, as that confounds physician and sales person effects (details are in §4.2.2).[13]

For each physician i in time period t, contagion is measured as the number of physicians who had adopted the drug by time period $(t-1)$ within a 20-mile radius—this contagion measure is labeled as CTGN.[14] To obtain this measure, we computed the distance between all pairs of physicians in Manhattan and in neighboring towns using latitude and longitude of the physicians' street addresses via geocoding. We geocoded the address of individual physicians using ESRI ArcGIS.[15] Using ArcCatalog we matched the table of addresses of physicians and the street locations in ArcGIS StreetMap. We then used ArcMap that turns the addresses into longitude and latitude coordinates. Finally, based on the longitude and latitude coordinates, we computed the Euclidean distance between each pair of physicians. This allowed us to compute the individual physician-specific contagion measure. The variation in physician street addresses and the adoption patterns of neighboring physicians result in a unique contagion measure for each physician.

Note that the temporal ordering in the relationship between a physician's behavior and the group's behavior allows us to break free of the reflection problem. The reflection problem arises when the effect of the group behavior on a physician's behavior cannot be distinguished from the effect of the physician's behavior on the group behavior (Manski 1993, 2000). In our case, this would arise if a given physician's propensity to adopt is a function of the mean propensity to adopt of the group. However, our analysis is not subject to this problem under the assumption that contagion effects would become apparent over a month's time. Thus, the effect of this group behavior up to time $(t-1)$ affects individual physician adoption at time t. In other words, the individual physician behavior at time t can be affected by group behavior up to $(t-1)$ but not vice versa.[16] Our constructed contagion measure has a mean of 1,092.6, with a standard deviation of 769.0.

Finally, there may be other factors that influence physicians' adoption behavior over time in a systematic manner. These include growth in the base of patients who are using the drug and aggregate diffusion patterns in the geographic area. We controlled for these measures by including a time trend term (TIME), which is the number of months lapsed since the drug launch, as one of the explanatory variables. To allow for potential nonlinear effects in these factors, we also included a quadratic-in-TIME term (TIMESQ). Note that these effects only vary over time and not by physician. The use of these two terms provides a parsimonious way to capture temporal effects. Our binary choice model with duration dependence

[12] We investigated the sensitivity of our results to a $+/-10\%$ variation in this radius. We found no significant changes in our results.

[13] In general, for any distance-based proxy, if the distance is too small, then you run into the problem of not having enough variation in the data. For example, we created a separate contagion measure for zero-distance physicians, i.e., physicians who work at the same physical location. While it is intuitive that we should find an effect of the adoption pattern of these physicians, the model estimates show an effect that is positive but insignificant. This is because there isn't enough variation in the data to be able to estimate this effect in any meaningful manner. Similarly, if the distance is too large, then the measure moves closer and closer to the aggregate diffusion pattern. At the limit, all physicians will have exactly the same contagion measure; i.e., the aggregate adoption pattern over time will predict any given physician's adoption behavior. We also estimated a model with such a measure (using the total number of adopters) and found that this measure is insignificant, although the other results are essentially unchanged. One possible explanation for this is that the total adopter variable is highly correlated with the time variables, which we have already included in our base model.

[14] In a later section, we scale the number of adopters by the total number of physicians and use the resulting proportion as our measure of contagion. This does not change our main result.

[15] ESRI is a leading provider of Geographical Information Software. More information on ESRI and its products (such as ArcMap, ArcCatalog) can be found at http://www.esri.com.

[16] Here, we are also making the implicit assumption that physicians are not forward looking. This assumption may seem strong, but, to the best of our knowledge, modeling individual behavior as a function of group behavior with forward-looking individuals is an unsolved problem in this literature because of identification problems and technical complexity.

can be seen as equivalent to a discrete-time-hazard (survival) model (Wheat and Morrison 1990).

The above discussion results in the following specification:

$$U_{it} = \beta_{0i} + \beta_{1i} * \mathrm{CTGN}_{it} + \beta_{2i} * \mathrm{DET}_{it} + \beta_{3i} * \mathrm{DETSTK}_{it} + \beta_{4i} * \mathrm{SAMSTK}_{it} + \gamma_1 * \mathrm{DTCA}_t + \gamma_2 * \mathrm{TIME}_t + \gamma_3 * \mathrm{TIMSQ}_t + \varepsilon_{it}.$$

4. Results and Discussion

4.1. Results

The results from our model are described in Table 3. As can be seen from the table, the effect of current detailing, detailing stock, and sampling stock is significant and positive. Thus, in terms of adoption behavior, physicians tend to respond to current detailing, past detailing, and the number of samples that have been dropped off in the past.

The monthly DTCA flow does not have a significant effect on physician-adoption behavior (we also found that using DTCA stock instead of flow gives the same result). The DTCA results are not surprising if we take a closer look at the DTCA expenditure time series (Figure 3). As can be seen from the figure, the first month when the firm had nonzero DTCA was the 10th month after the product was launched. This was then followed by 5 months of no DTCA and then a significant campaign in months 15 and 16 (after product launch). Therefore, if DTCA was the major mechanism by which patients become aware of drug X, this would happen in about and after month 16. At this point, more than 70% of the physicians in our sample had already adopted the drug. Thus, it seems that there is no real effect of DTCA on physician-adoption behavior.

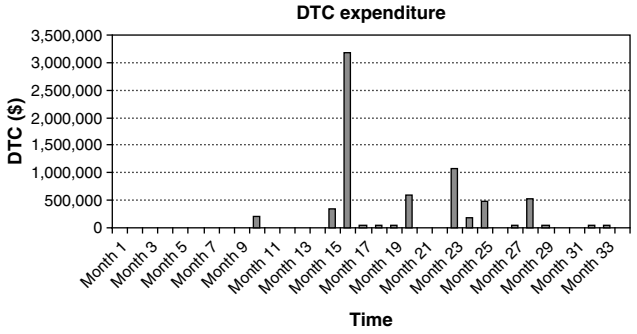
Both the TIME and the TIMESQ variable are significant. Interestingly, the sign of the former is *negative* and the latter *positive*. Note that this arises because we observed no adopters in the first month and then higher subsequent adoption; the temporal diffusion pattern tends to be convex, rather than concave.

Finally, even though we controlled for time trends and marketing variables, we found that the contagion measure is both positive and significant.[17] This also implies that the contagion effect is not just picking up aggregate diffusion but is identified from a sufficient cross-sectional variation.[18] As mentioned earlier, we used geographic distance as a proxy for true social interaction (contagion). Thus, given this operationalization, our model detects the presence of contagion in the adoption of a new drug by physicians. However, to make a convincing case that this is indeed the effect that we want to capture, we need to rule out as many alternative explanations as possible for our finding that the contagion coefficient is significant. Note that, given the lack of any DTCA effects (described previously), we have excluded DTCA from the specification from the analyses discussed in the rest of the paper.

Table 3 Results for the Manhattan Market

Variable	Mean	(post SD)
INTERCEPT	−3.76	(0.18)
TIME	−0.11	(0.04)
TIMESQ	0.0018	(0.0003)
DTCA (flow)	0.07	(0.08)
CTGN	1.02	(0.27)
DET	0.35	(0.08)
DETSTK	0.44	(0.06)
SAMSTK	0.08	(0.04)

Figure 3 Monthly DTCA Expenditure of Drug X



4.2. Alternative Explanations: Contextual and Correlational Effects

A major focus of studies investigating contagion is to rule out alternative explanations for the (detected) contagion effect. These tend to fall into two types of explanations—those arising from contextual and/or those arising from correlational effects (Manski 2000).

4.2.1. Contextual Effects. Contextual effects arise from the possibly unique nature of the social network that is being studied. In our case, this would arise if there was something unique about physicians who treat this condition or about physicians in our chosen geographic area (Manhattan). The first is unlikely to be an issue, as the majority of physicians in our sample are PCPs who prescribe across a wide variety of therapeutic classes. The second is more likely, as it could be argued that Manhattan represents an outlier market. In addition, as Manhattan is geographically compact, we might be more likely to find contagion.

[17] For computational reasons, the contagion measures are scaled down by a factor of 1,000 in the estimation.

[18] The aggregate diffusion pattern for the New York metropolitan area is almost perfectly explained by the TIME and TIMESQ terms—the R square of that regression is 0.99.

Table 4 Manhattan and Indianapolis Demographics

Demographic characteristics	Manhattan	Indianapolis
Total population	1,537,195	781,870
Median age (years)	35.7	33.5
18 years and over (%)	83.2	74.3
College degree (%)	49.4	25.4
Foreign born (%)	29.4	4.6
White (%)	54.4	69.1
Black or African American (%)	17.4	25.5
Asian (%)	9.4	1.4
Hispanic or Latino (of any race) (%)	27.2	3.9
Average household size	2.00	2.39
Total housing units	798,144	352,429
In labor force (16 years and over)	841,633	415,761
Per capita income (dollars)	42,922	21,640
Individuals below poverty level (%)	20.0	11.9

Source. U.S. Census Bureau, 2000 Census Summary File 1 and 3.

We therefore picked another market—Indianapolis—that is quite different from Manhattan. This market is located in the Midwest (as opposed to the Northeast), it is much less dense in the number of physicians, the patient demographics are different (see Table 4 for a comparison of the population demographics), and the firm's marketing effort here is directed in such a manner that the relevant radius for defining the network band is also 20 miles (again, this distance is very close to the distance between the centroid and the city limit).

We created a similar data set by focusing on physicians from the Indianapolis market. We estimated the same model for the Indianapolis data and found significant effects for contagion in this market as well (see Table 5 for detailed results).[19]

4.2.2. Correlational Effects. Correlational effects could act as proxies for contagion, as they represent some unobserved common variables that affect all the members of a network in a similar manner. In our case, if all physicians in the Manhattan area were detailed by the same salesperson over the duration of our sample, the contagion effect could be a proxy for the effectiveness of the salesperson over time. In the rest of this section, we tried to rule out as many such effects as possible.

Let us consider the salesperson effect noted above. Although we know that the same salesperson does not detail all physicians in the Manhattan area, it is possible that contagion effect is confounded with a salesperson effect. In other words, the fact that the adoption behavior of physicians located physically closer to each other is correlated is because they get detailed by the same salesperson. We could not model this explicitly, as we did not have the identity of the individual salesperson in our data. We therefore checked with the firm, which provided us the data. We found that salespersons definitely do not have territories in the Manhattan area that are larger than 10 miles. We therefore split up our contagion measure into two measures (that span sales person territories)—one that is based on a 10-mile radius (CTGN10) and the other that is based on a 10- to 20-mile radius (CTGN20) and reestimated our model (see Conley et al. 2003 for a similar approach).

[19] We found that the coefficient for the TIME and TIMESQ were insignificant for this market. We therefore show the results from a model without these terms.

Table 5 Results for the Indianapolis Market

Variable	Mean	Post SD
INTERCEPT	−3.92	0.20
CTGN	4.86	0.70
DET	0.44	0.09
DETSTK	0.54	0.11
SAMSTK	0.14	0.05

Our results (Table 6) show that the CTGN20 measure is significant in addition to the CTGN10 measure. Thus, we can conclude that our results are not due to a salesperson effect. These results also suggest that our operationalization of geographic distance as a proxy for contagion (interaction) is reasonable. This is because, if our operationalization is correct, physicians closer to the focal physician should have a larger (contagion) effect on her than physicians farther away. From Table 6 it can be seen that the coefficient for the CTGN10 measure is about 21% larger than the coefficient for the CTGN20 measure (the marginal effects also show the same pattern).

The next correlational effect has to do with hospital-specific effects. In other words, it may be possible that the contagion effect is being driven by institutional features relating to the hospital or practice. This could be due to either the fact that physicians in the same hospital (at zero distance) have the potential for a lot of interaction or to some other unobservable, institutional feature. An example of such an unobservable would be that the hospital has a specialty practice in the therapeutic category of a new drug.

Table 6 Results—After Splitting the Contagion Measures into Two

Variable	Mean	Post SD
INTERCEPT	−3.58	0.18
TIME	−0.24	0.05
TIMESQ	0.008	0.001
CTGN10	1.49	0.30
CTGN20	1.23	0.44
DET	0.26	0.05
DETSTK	0.58	0.06
SAMSTK	0.08	0.02

Another example of such an unobservable is the role of the "formulary." When firms launch new drugs, they negotiate with insurance companies to get them on their list or formularies. However, this process varies by facility—for large practices (e.g., teaching hospitals), formulary approval is decided on by pharmacy and therapeutics committees that consist of practicing physicians. There are many industry pressures on physicians to not prescribe drugs unless they are on a formulary. If different insurance plans approve a new drug's inclusion on the formulary at different times, then it is likely that the adoption decision of a physician in a hospital will be related to when the drug went on the formulary for the patients of that hospital. Thus, if the drug went onto the formulary for a set of geographically close medical facilities in a short time interval and physicians in these facilities adopted the drug in that time period, the two effects might be confounded.

Given the nature of the process by which a drug goes on a formulary, data on facility-level formulary adoption times is typically unavailable. We took two approaches to investigate the possible role of formularies on physician-adoption behavior. First, we enquired from the brand manager of drug X about the formulary status of drug X at the time of launch. At the time of the launch, the drug had achieved equal or advantaged status (i.e., the drug had been approved by the pharmacy and therapeutics committees and was within the copay structure) in 93% of all the managed care, pharmacy benefits managers (PBMs), and indemnity insurance plans. In terms of lives covered (i.e., insured patients), this translated into 80% coverage. The gap relative to complete formulary coverage (the remaining 7%) and to lives covered (the remaining 20%) was caused by slow approvals in markets other than the ones we considered (California, Arizona, and Florida). In addition, Manhattan also has a high proportion of Medicaid patients, for whom all drugs cost the same (i.e., open formulary).

Second, we verified that formulary effects did not affect adoption. If we assume that all physicians in each facility see the same mix of patients on average with respect to their insurance status, we can assume that the drug is on the formulary once the first physician in that facility prescribes it. If a typical physician is waiting for the drug to be on the formulary before she prescribes, then the adoption process for the second physician should be very quick once this occurs. We therefore grouped all physicians in our Manhattan sample such that each group consists of physicians who work at the *same* facility. We then examined the time to adoption for the second or later adopter in each facility from the time when the first adopter adopted. If this is not instantaneous, then it is unlikely that the formulary status is driving adoption. In our sample, we found 118 such nonfirst adopters (who were already prescribing in the category before drug X). We found that, for these adopters, the mean duration to adopt was 14.6 months (as opposed to 12.5 months across the whole sample). This suggests that, on this dimension, these physicians are not different from the rest of the sample. Moreover, we found that it takes these physicians an average of 8.4 months to adopt after the first adoption took place at the same facility. This again suggests that the adoption of nonfirst adopters does not happen instantaneously when the first adopter adopts. We also reestimated the model with the nonfirst adopter sample to see if we could find the contagion effect with this group. As the results show (Table 7),[20] the contagion measure is positive and significant. This finding also helps to rule out any other hospital specific effects.

Table 7 Results with Nonfirst Adopters

Variable	Mean	Post SD
INTERCEPT	−6.19	0.77
CTGN	2.03	0.41
DET	0.27	0.12
DETSTK	0.41	0.13
SAMSTK	0.007	0.008

Taken together, the two checks above suggest that it is unlikely that our contagion results are a proxy for the inclusion of the drug in formularies or hospital-specific effects over time.

Finally, the contagion effect might be confounded with the role of local physician meetings and events (PME) sponsored by the manufacturer of drug X during the data time period. We were able to obtain aggregate spending on PME over time for a period of 28 months postlaunch. We therefore recreated the data set (by dropping the last six months of data) and adding the monthly expenditure on PME. We estimated our model again using both PME flow and PME stock (with the carryover factor at 0.7, as before).

As can be seen from the results in Table 8, neither PME flow nor PME stock have a significant effect on physician-adoption behavior. As noted above, this may be because we have only aggregate data. The other coefficients also remain basically unchanged.

4.3. Sample and Specification Checks

In terms of our sample of physicians, as noted previously, we decided to focus on adopters only. We did this for a variety of reasons. First, a fundamental reason to adopt the new drug is to prescribe it to patients. Thus, if we do not see any adoption activity

[20] We found that the coefficient for the time trend and time trend squared were insignificant for this sample. We therefore show the results from a model without these terms.

Table 8 Results with PME

Variable	Mean (post SD)		Mean (post SD)	
INTERCEPT	−3.55	(0.23)	−3.52	(0.22)
TIME	−0.14	(0.07)	−0.05	(0.03)
TIMESQ	0.0004	(0.0001)	0.00027	(0.00006)
PME (flow)	−0.03	(0.07)		
PME (stock)			−0.13	(0.08)
CTGN	1.06	(0.048)	1.01	(0.46)
DET	0.32	(0.08)	0.36	(0.08)
DETSTK	0.55	(0.07)	0.48	(0.12)
SAMSTK	0.04	(0.03)	0.07	(0.04)

by a group of physicians over a 34-month period, it is likely that the patient base of that physician does not warrant drug adoption. To test this hypothesis, we look at the *total category* prescription behavior of all the physicians in our Manhattan sample. Over all the months, we found that drug X adopters account for 93% of all category prescriptions. In other words, we found that the nonadopters of drug X do not really prescribe in this *category*. In fact, the median average number of category monthly prescriptions written by the nonadopter physicians is essentially zero (it is 0.03, as opposed to 9.69 by adopters). Given this, we included only physicians who had adopted by the end of the data period in our sample.[21]

In our model, we used the linear and quadratic time terms to control for duration dependence in our data. It could be argued that imposing this functional form on the temporal effects may be too strong an assumption. We therefore estimated the model with time-period fixed effects—this is the most "agnostic" model, as it does not make any functional form assumptions about the temporal effects. In the interest of brevity, the results are described below, but the details are not included (these are available from the authors on request).

We found that 11 of 34 time-period fixed effects are tightly estimated with a t-statistic of 1.65 ($p < 0.10$). The contagion and marketing effects are unchanged. Thus, the inclusion of a nonparametric temporal response using time fixed effects does not change our results. Our decision to use our current specification as our main model instead of this one is based on the fact that our current model is favored overwhelmingly—the Bayes factor in favor of our model is 94.29 (recall that a Bayes factor is ratio—if it is greater than 1, it favors our model).

Our contagion measure consists of the raw count of physicians in the band around each physician. An alternative measure would be to look at the *proportion* of adopters in this band. This measure has the advantage that it conforms to the same scale for each physician. This measure also controls for a situation in which physicians located farther away from the centroid have fewer neighbors. We created this measure and estimated a model with this as the contagion measure (CTGNP). As can be seen from Table 9, we found a positive and significant effect of contagion even with this measure.

[21] We also estimated two models—a model with a single 20-mile-based contagion measure and a model with a 0- to 10-mile and a 10- to 20-mile-based contagion measure—in which we included nonadopters in our sample. In both cases, we found evidence for a positive and significant contagion effect. However, the effect is weaker than in a sample that is restricted to adopters only.

Table 9 Results with the Proportion-Based Contagion Measure

Variable	Mean	Post SD
INTERCEPT	−3.23	0.14
TIME	−0.09	0.03
TIMESQ	0.001	0.001
CTGNP	7.36	1.19
DET	0.45	0.04
DETSTK	−0.05	0.04
SAMSTK	0.11	0.02

Finally, we compared our model with the standard diffusion model. As is typical for such models of diffusion, the model is calibrated from aggregate data. We ran the usual regression of adopters on lag cumulative adopters and lag cumulative adopters squared (for the Manhattan data). Note that the coefficients of both these terms are not statistically significant (Table 10). This suggests that the use of the Bass model will lead to the conclusion that there is no contagion in this market. Even if we were to *assume that these parameters are different from zero*, the estimated Bass model parameters are $p = 0.059$, $q = 0.031$, and $N = 492$. Note that even this q is much smaller than the mean of around 0.4 reported in meta-analysis studies.

4.4. Managerial Implications

Our results so far have documented that our operationalization of the contagion measure is predictive of physician-adoption behavior. We have also ruled out, to the extent possible with the data at our disposal, a variety of contextual and correlational effects. Our explanation for the detected contagion effect is that it is due to the consumption externality referred to earlier. Our conjecture is that the mechanism driving this consumption externality is noncommercial interaction among physicians in local geographies, as well as information transmission by industry participants such as detailers.

Table 10 Bass Model Regression Results

	Estimate	SD	t-stat
Intercept	29.64	2.73	10.86
Lag cumulative adopters	−0.028	0.024	−1.1842
Lag cumulative adopters sq	−6.3E-05	4.63E-05	−1.35

Given that we have found a statistically significant contagion effect, it is important to investigate the size of the effect to check if it is economically significant as well. To do this, we conducted a series of computations and counterfactual exercises. First, we focused purely on the demand side and computed the social multiplier of marketing implied by our results. This multiplier may be seen as the *additional* increase on the probability of adoption arising from contagion over and above the increase based on the targeted marketing effort (detailing). Specifically, we found that this multiplier (on average) is 1.11; i.e., there is an 11% increase in the probability of adoption over and above the direct and immediate impact of the marketing effort. This has clear implications for the firm, as, on average, it can reduce marketing expenditure by this amount for qualitatively similar outcomes. We provide more details on the extent of expenditure reduction for a couple of temporal scenarios below.

We also illustrated the aggregate demand (adoption) pattern over time as predicted by our model and contrasted it with the prediction by another version of our model where the contagion measure was assumed to play no part. In other words, we ran the counterfactual experiment where we set the contagion measures to zero and then predicted adoption using our estimates.

These patterns, along with the actual adoption pattern, are graphically depicted in Figure 4. As can be seen from the figure, the adoption pattern based on our model mirrors the actual adoption pattern quite well (though it does smooth out many of the peaks). However, a model with no contagion predicts a significantly different adoption pattern, especially after the first few months. In terms of predicting the total number of adopters, although both models underpredict the total number of adopters, the model without contagion underpredicts by more (only 24% adopt) than the model with adoption (80% adopt).

Figure 4 **Adoption Pattern With and Without Contagion Measure**

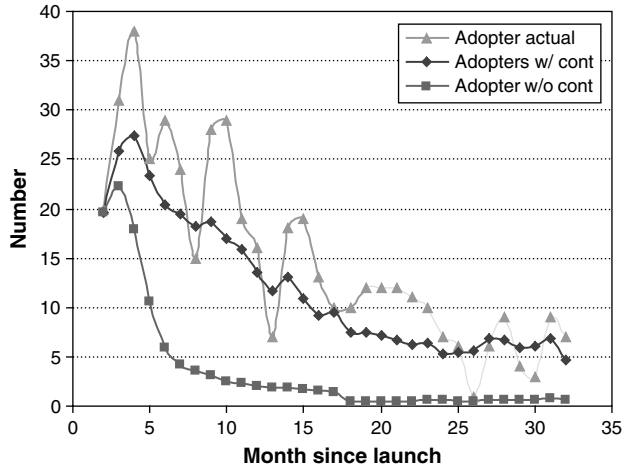


Table 11 **Percentage of Adoption Probability Arising from Contagion Relative to Marketing Communication**

Month after launch	Percent of contagion
2	45
3	46
4	54
5	64
6	71
7	74
8	76
9	77
10	81
11	83
12	85
13	87
14	86
15	88
16	89
>17	∼90

From the firm's point of view, it may be useful to examine the relative effect of contagion versus the marketing instruments. Note that by definition, marketing activity will play a larger role in the early stages. However, our model can help to show the relative effect sizes over time. We therefore decomposed the probability of adoption in a given time period into a part arising from contagion and a part arising from the marketing activity (detailing and sampling).[22] Table 11 shows the proportion of the adoption probability that can be attributed to the contagion effect relative to the total effect (contagion plus marketing activity).

As can be seen from the table, marketing plays a large role in affecting adoption early on (accounting for a majority of the effect up to month 3). However, the role of contagion dominates from month 4 onward and, by month 17 (or about half the duration of our data), asymptotes to about 90% of the effect.

Given the above, it may be useful for the firm to understand why certain physicians are more susceptible to contagion. A median split of the size of the individual-level contagion effect shows that the proportion of specialists is much lower in the top half (7%) than the bottom half (13%). This suggests that specialists need to rely less on contagion, presumably because of the quality of their knowledge

[22] Using the estimated parameters, we predicted the probability of adoption for each observation using (a) marketing variables only, i.e., no contagion, and (b) contagion measure only, i.e., no marketing variables. This, along with the total probability of adoption, allowed us to assess the relative effect of each set of variables. We report the mean effect across all observations for a time period in the table.

about the therapeutic category and the new drug. This finding is consistent with earlier research that has documented that physicians with more knowledge and authority tend to rely less on cheap sources of information such as advertising and sales force effort in terms of their prescription decision (Stern and Trajtenberg 1998).[23]

Finally, from a supply aspect, we try to quantify the amount of marketing resources that can be freed up once contagion is accounted for. To do this, we carried out an experiment where we quantified the economic impact of additional adopters at month 2 (very early) and at month 10 (at the median time to adoption month). In both cases, we predicted the new adoption pattern given the additional level of adoption in each of the two months. We then contrasted the time to adoption for our entire set of physicians under this situation with the situation in the data. We then computed the total number of additional prescriptions generated in the time saved (the time difference between the time to adopt for the entire pool without and with additional adopters). We translated the number of additional prescriptions to the equivalent number of free details. Our results show that for a 5% increase in adopters in month 1, the firm can obtain a mean saving of 3% on detailing. In month 10, a 5% increase in adopters results in a mean saving of 0.2% on detailing. These numbers are more indicative of how much the firm can allocate to increase the front loading of detailing.

5. Conclusion, Limitations, and Directions for Future Research

Our paper adds to the small but growing body of research that is using behavioral data to document contagion. It differs from past research in at least four distinct ways. First, we used behavioral data to measure adoption in our model instead of self-reported survey data. Our work can thus be seen as complementary to the literature that uses self-reported survey data. Second, we estimated the effect of contagion after accounting for the effect of *individually* targeted marketing communication (detailing and sampling) as well as aggregate-level marketing efforts (such as DTCA). Third, we allowed the contagion effects to be heterogeneous across physicians. Finally, we controlled for unknown temporal effects by explicitly including time trend variables in our model.

In the paper, we demonstrated the existence of contagion effects among physicians in Manhattan and Indianapolis. We found that this effect exists even in the presence of targeted marketing, DTCA, and time trends. We also tried and ruled out alternative explanations to the extent allowed by our data. Specifically, we ruled out contextual (via the use of data from another market) and correlational effects such as salesperson effects, formulary and other hospital-specific effects, and effects of common marketing instruments such as physician meetings and events. We also ruled out any other common effects that are correlated with the linear and quadratic time terms and checked that these results were not driven by our specific sampling scheme and the specification of the contagion measure. We also found that the effect size is relatively large, especially a few months after the launch of the drug.

There are several limitations of the paper that we hope can be addressed in future research. First, part of the consumption externality could be due to physician interactions via meetings and events directed at physicians. Although we were able to show that the aggregate-level expenditure on such meetings and events did not seem to affect adoption, individual-level meetings and events attendance data could pinpoint these effects. Second, although we have included the DTCA measure in the model, we acknowledge that this variable might not be able to fully capture the impact of patient-induced contagion. Our measure of contagion may be confounded with contagion at the patient level. We have tried to use geographic distance to minimize the impact of the patient-to-patient (and patient-to-physician) contact, but we cannot rule it out. Future research could focus on obtaining patient-level data that tracks interaction among patients and physicians and controls for this effect explicitly. Third, we studied two local markets and one therapeutic category in this paper. Further research can look at other therapeutic categories and different markets (as also a nationally representative sample) to check the robustness of our findings. Fourth, if data on physician adoption of multiple drugs are available, then models that allow for the firm to use this knowledge in targeting marketing resources can be specified. Finally, the model specification can be made richer. For example, the effects of marketing instruments and contagion could vary over time and/or the error terms could be spatially correlated. Future research could also test for asymmetric contagion effects between individual physicians or groups of physicians (e.g., see Nair et al. 2006 for a study of the effectiveness of key opinion leaders in this industry).

[23] A hierarchical model that allows the contagion response parameter to vary by specialty would also pick this up. We are, however, unable to find this effect. This is most likely because the number of specialists in our sample is limited (10%).

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Appendix. The Markov-Chain Monte Carlo Sampler

We describe the full conditional distributions of the unknowns below.

1. The full conditional distribution for β_i is given as

$$p(\beta_i \mid \beta_0, \gamma, V_\beta, y_{it}, x_{it}) \propto l(\beta_i) * \exp((\beta_i - \beta_0) * V_\beta^{-1} * (\beta_i - \beta_0)'),$$

where $l(\beta_i) = \Pr_{iT} \prod_{t=1}^{T-1} (1 - \Pr_{it})$.

The full conditional distribution for β_i is known only up to a proportionality constant. We use the Random Walk Metropolis-Hastings algorithm to generate a candidate on iteration n as $\beta_i^c = \beta_i^{(n-1)} + \breve{\beta}_i$, where $\breve{\beta}_i$ is a draw from a multivariate normal proposal density, $N(0, k\Psi)$. We set ψ to the asymptotic variance-covariance matrix of the β parameters estimated on pooled data (i.e., assuming no physician level differences) using maximum likelihood. The acceptance probability is given by

$$\min\left\{\frac{p(\beta_i^c \mid \bar{\beta}, V_\beta, y_{it}, x_{it})}{p(\beta_i^{(n-1)} \mid \bar{\beta}, V_\beta, y_{it}, x_{it})}, 1\right\},$$

where $p(\cdot \mid \cdot)$ is as given above.

2. The prior distribution for β_0 is $N(\beta_{00}, V_{\beta_0})$. The full conditional distribution for β_0 is given as

$$p(\beta_0 \mid \{\beta_i\}, V_\beta, \beta_{00}, V_{\beta_0}) = N(\hat{\beta}, \hat{V}_\beta),$$

where $\hat{\beta} = \hat{V}_\beta (V_{\beta_0}^{-1} * \beta_{00} + \sum_{i=1}^{I} V_\beta^{-1} * \beta_i)$ and $\hat{V}_\beta = (V_{\beta_0}^{-1} + \sum_{i=1}^{I} V_\beta^{-1})$. We set $\beta_{00} = 0$ and $V_{\beta_0} = \text{diag}(20)$.

3. The prior distribution for γ is $N(\gamma_0, V_\gamma)$. The full conditional distribution for γ is given as

$$p(\gamma \mid \{\beta_i\}, y_{it}, x_{it}) \propto l(\gamma) * \exp((\gamma - \gamma_0) * V_\gamma^{-1} * (\gamma - \gamma_0)'),$$

where $l(\gamma) = \prod_i (\Pr_{iT} \prod_{t=}^{T-1} (1 - \Pr_{it}))$. We set $\gamma_0 = 0$ and $V_\gamma = \text{diag}(20)$. As this full conditional distribution is also known up to a proportionality constant, we use a Random Walk Metropolis-Hastings algorithm similar to the one described above.

4. The full conditional distribution for V_β^{-1} is

$$p(V_\beta^{-1} \mid \{\beta_i\}, \beta_0, \rho, R) = \text{Wishart}\left(\left[\rho * R + \sum_{i=1}^{I} (\beta_i - \beta_0)(\beta_i - \beta_0)'\right]^{-1}, \rho + I\right),$$

where I is the number of customers. We set the prior mean of $V_\beta = (\rho R)^{-1} = \text{diag}(10)$ and the prior degrees of freedom, $\rho = \text{NPAR} + 3$, where NPAR is the dimension of the β vector.

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