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Competitive diffusion of new prescription drugs: The role of pharmaceutical marketing investment

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ABSTRACT

We investigate the impact of marketing interventions on the diffusion of new products in a competitive setting. We develop a family of trial–repeat diffusion models to identify the longitudinal effects of marketing efforts, and complement this with a cross-sectional analysis to identify the between-drug effects. We believe that we are the first to consider both longitudinal and cross-sectional marketing effects in a trial–repeat diffusion context. The models are calibrated on 34 drugs in three therapeutic categories using monthly data. Our longitudinal analyses demonstrate that the trial rate responds positively to increases in own marketing expenditures but is affected negatively by competitors' expenditures. We show how these within-drug analyses provide opportunities for accelerating the diffusion process by reallocating marketing expenditures over time. The cross-sectional analyses demonstrate that pharmaceutical marketing has both an informative and a persuasive influence on the diffusion of new drugs. We find that direct-to-consumer advertising does not affect the trial nor repeat rates during the first months after introduction. We illustrate the managerial relevance of our results and find that a reallocation of marketing budgets does not alter the saturation level, but can help in attaining this level faster. We show that this has a great effect on sales, market share and ROI.

1. Introduction

Product innovation is widely recognized as an important source of competitive advantage and a driver of firm's growth and profitability (Salomo et al., 2007), the stock returns for the Top 25 innovative companies are 134% higher than the others over 5 years (Appel, 2013). In many, if not all, industries, new product development represents a top priority, because modern product life cycles are shrinking, and competition has intensified in the drive to satisfy customer needs (Filippini et al., 2004). However, almost half the resources that U.S. industries devote to product development go to products that fail. The failure rate of new products reaches 90% (Nielsenwire, 2013). Others estimate that only 5% of new consumer products launched are successful in the market (Nobel, 2011). The situation is even more challenging for manufacturers of pharmaceuticals, an industry characterized by long development times, low success rates, high development costs, high capital requirements for manufacturing facilities, delayed feedbacks, and broad uncertainty in sales estimates (Blau et al., 2004; Chiou et al., 2012). Typically, the process of clinical trials and Food and Drug Administration (FDA) approvals required of a pharmaceutical company takes approximately 10 years after filing a patent for a particular molecule. On average, only one or two of every 10,000 substances synthesized in laboratories, will successfully pass all the phases to become marketable drugs (EFPIA, 2012). The average cost of successfully launching a drug to market achieves $1,137 million (Deloitte and Thompson Reuters, 2013). In fact, pharmaceutical firms such as GlaxoSmithKline and Eli Lilly

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outsource much of their research on new products to reduce the amount of dollars that the development and launch of new drugs require (Calantone and Stanko, 2007). The R&D internal rate of return of leading pharmaceutical firms was 7.2% in 2012 (Deloitte and Thompson Reuters, 2013).

As time-based competition increases due to the globalization of business (Harvey and Griffith, 2007), it is important for any manufacturer to discover factors that increase and accelerate adoption of newly developed products, which can increase the profitability and shorten the earn-back period of R&D investments. For innovative pharmaceutical companies this is even more important, because the competitive advantage of a new pharmaceutical product is usually only temporary due to patent expirations. Also from a patient's point of view it is important to accelerate new product adoption as health outcomes may improve as a result of the rapid diffusion of new drugs that provide unique and superior benefits.

Which role plays pharmaceutical marketing in the diffusion process of new drugs? Measuring the effects of pharmaceutical marketing and understanding the importance of their informative and persuasive roles on the diffusion process of new drugs can help manufacturers allocate resources to pharmaceutical marketing more efficiently. In this study, we address these important issues.

Firstly, we therefore develop diffusion models that determine the effects of pharmaceutical marketing expenditures on the diffusion of new drugs. Explicitly including marketing variables in diffusion models not only provides a better description of reality but also offers guidance for managerial decision makers on how to spend their marketing budget (Kalish and Sen, 1986; Mahajan and Muller, 1991; Bass et al., 2000). However, the literature is inconclusive about the appropriate inclusion of marketing variables in diffusion models. Earlier diffusion studies that accommodate the effects of marketing variables can be classified according to the way they include these effects in the model. A first stream of studies models the effects of for example manufacturer's advertising as an external influence of marketing efforts on trial rates, (e.g., Lilien et al. (1981); Hahn et al. (1994)). In contrast, a second group of studies assumes that marketing affects trial through internal influence, such that it affects the interpersonal communication among physicians who already have adopted the new drug and potential adopters (e.g., Simon and Sebastian (1987)). A third group of studies considers both external and internal effects of marketing on trial; this is known as the mixed influence effect of marketing (Parker and Gatignon, 1994). A fourth stream of research considers the possibility that marketing instruments influence the repeat rate (e.g., Mesak and Berg (1995)). However, there is no consensus on which of the four ways is most appropriate to accommodate the effects of marketing efforts in diffusion models. This paper is the first that does not make a priori assumptions about how marketing efforts should be included in diffusion models. Instead, we empirically investigate the most appropriate location of marketing variables in trial–repeat diffusion models, using data of 34 individual new drugs in three different therapeutic categories.

Most studies in these four research streams model longitudinal effects of marketing. Thus, they employ a within-brand analysis, studying how marketing affects the diffusion process of a new product over time. This is important because it creates opportunities for influencing the diffusion process by reallocating marketing expenditures over time. However, other studies also indicate that cross-sectional variation in sales (i.e., across geographical markets or brands) may be much greater than longitudinal variation (Bronnenberg et al., 2007). However, to the best of our knowledge, marketing diffusion literature has largely neglected between-brand analyses.

Secondly, there is an unresolved debate over the years around the potential impact of pharmaceutical marketing on physicians' behaviors (Lim and Kirikoshi, 2008). Pharmaceutical industry spent billions of dollars to promote its new products to physicians (push marketing) and consumers (pull marketing), expecting that this has positive effects on physicians' prescriptions. These promotional activities can generate two types of effects on prescription behavior: it can help physicians making better and informed decisions (informative effect), but it can also create market power (persuasive effect). These two types of effects are central to the debates on the role of pharmaceutical marketing (Ching and Ishihara, 2012). If one observes the pharmaceutical marketing efforts and the diffusion process of the new drugs over time, it is hard to disentangle the informative and persuasive roles of marketing. Hence, we analyze the cross-sectional effects of marketing expenditures and perform a second-stage analysis on the brand-specific estimation results. This analysis allows us to investigate the informative and persuasive roles of pharmaceutical marketing and whether the observed differences in the diffusion parameters between brands result from differences in their average marketing expenditures.

Hence, in this study we focus on exploring the role of pharmaceutical marketing investment on the diffusion process of new prescriptions using both longitudinal and cross-sectional analyses. Our results support the existence of both longitudinal and cross-sectional diffusion effects for all three categories under study. We determine empirically that marketing efforts mainly affect trial rates of new prescription drugs via external and not internal influence. We allow for heterogeneity in the effects of both push and pull marketing strategies. Specifically, from the cross-sectional analysis, we conclude that marketing expenditures affect not only the trial rate but also the repeat rate of the diffusion process of new prescription drugs. We also find that pharmaceutical marketing has both informative and persuasive effects on diffusion. In a simulation study we illustrate the implications of our results. We conclude that an appropriate reallocation of marketing expenditures over time can have a great influence on sales, market share and ROI. In particular, our simulation study shows that reallocating marketing expenditures over time can have large effects on the speed of the diffusion process and may prolong the maturity stage in the product life cycle. The simulation study also shows that the saturation level is unaffected by the total marketing budget.

The remainder of this article is organized as follows: In the next section, we review marketing issues in the pharmaceutical industry, thereafter we specify our trial–repeat diffusion model. Subsequently, we present the data that are used in the empirical study. Next we discuss the estimation results of our longitudinal and cross-sectional analyses. Finally, we summarize and discuss our conclusions. In Appendix A2 we illustrate the managerial implications of our work with simulation studies.

2. Pharmaceutical marketing

In the US, pharmaceutical industry spent $10.7 billion in 2011 to promote its products (excluding expenditures for drug samples), with $6.8 billion (64%) directed to physicians and $3.9 billion (36%) in direct-to-consumer (DTC) advertising (IMS Health, 2013). The push marketing strategy, which targeted mainly the physicians, includes physician meetings and seminars, medical journal advertising, samples, direct mail, and detailing (traditional detailing refers to sales representatives visit physicians to provide information on drugs while e-detailing refers to educating physicians via electronic means). With the pull marketing strategy, pharmaceutical firms attempt to play on consumers' positive attitudes toward direct-to-consumer ads. The marketing efforts developed by the pharmaceutical manufactures can generate both informative and persuasive effects on prescribing decisions. When several competing drugs can treat a disease, a physician may select a preferred product based on the drug's benefits for their patients' health. Physicians (who are the decision makers for the prescription decision) are not perfectly informed about a new drug's benefits prior to its use. Hence, the information provided by pharmaceutical companies about the new drug's efficacy, side effects and so forth (always supervised by the US Food and Drug Administration) reveals crucial in the physician's decision of prescribing a new drug. This information reduces uncertainty about the existing drugs in the marketplace and allows physicians to prescribe the most appropriate drug to their patients. Physicians will eventually learn the true drug's benefits and hence pharmaceutical marketing will not play an informative role in the long run (Narayanan et al., 2005). Thus, the *informative function* of marketing influences the diffusion process of a new drug through the trial rate.

Prescription drugs belong to the category of frequently purchased products where adopters may switch from one product to another substitute product. The role of pharmaceutical marketing must convince the physician that his/her initial choice is the most appropriate for his/her patients' health outcomes, and in that manner reduce the probability of a switch to a competing drug. Hence, the *persuasive function* of pharmaceutical marketing refers instead to the influence that marketing activities have in terms of creating market power for the promoted product. In this way, the *persuasive function* of pharmaceutical marketing builds barriers to entry and influences the diffusion process of a new drug through the repeat rate.[1]

There is an open debate on the informative and persuasive roles of pharmaceutical marketing on physicians' prescriptions (Ching and Ishihara, 2012). Hurwitz and Caves (1988) find that marketing activities protect the market share of innovators when generics enter the market. Currie and Park (2002), studying prescriptions of antidepressants, find evidence for only the *informative function* of marketing communication; in contrast, Leffler (1981), studying antihypertensive drugs, argue that detailing plays informative and persuasive roles; Narayanan et al. (2005) discover evidence of both informative and persuasive effects in their study of prescription antihistamines; and, Ching and Ishihara (2012), using data for angiotensin-converting enzyme inhibitor with diuretic, also find significant evidence for both effects.

[1] From this perspective, persuasive pharmaceutical marketing primarily affects experienced physicians.

3. Research framework and model specification

The Bass model (Bass, 1969) remains the most parsimonious model for diffusion processes in marketing literature and is widely accepted (Mahajan et al., 1990, 2000; Peres et al., 2010). In fact, the majority of the literature stream on diffusion models is based on the framework developed by Bass (Peres et al., 2010; Fok and Franses, 2007), although there is literature that also suggests new avenues for empirical implementation of diffusion models (Cabral, 2012). The empirical application of the Bass model has been tested on many different product categories, especially durable products (Sultan et al., 1990) but also on products such as wind energy (Davies, 2011), photovoltaic systems (Guidolin and Mortarino, 2010) or scientific publications (Fok and Franses, 2007). The model assumes that each adopter purchases the new product only once, which restricts its applicability and makes it less suitable for nondurables that involve repeated purchases. Furthermore, in the Bass model, the model parameters implicitly capture the impact of the marketing variables. This assumption makes it impossible to analyze the effects of marketing instruments on the diffusion process of an innovation. Robinson and Lakhani (Robinson and Lakhani, 1975) include marketing variables in their diffusion model, and many authors since have proposed diffusion models that incorporate marketing variables, though most still focus on durable products. For example, Bass, Krishnan and Jain (Bass et al., 1994) propose the Generalized Bass Model, that extends the Bass model by incorporating price and advertising. Several authors, including Dodson and Muller (1978), Dolan and Jeuland (1981), Lilien et al. (1981), Jeuland and Dolan (1982), Mahajan et al. (1983), Hahn et al. (1994) and Shankar et al. (1998), modify the Bass model to accommodate nondurable products and allow for repeat purchases, with several applications to pharmaceutical products. Both Lilien et al. (1981) and Hahn et al. (1994) propose trial–repeat diffusion models that accommodate the effects of promotional efforts, and Hahn et al. show that, compared with other models, their model achieves the best fit, superior forecast ability, and high parameter face validity. The results from those previous studies reveal the important role of repeat sales in the diffusion process of pharmaceuticals and, hence, lead us to focus on trial–repeat diffusion models. However, there are studies on the diffusion of pharmaceuticals that do not include repeat sales in the diffusion model (Guseo and Guidolin, 2011).

Shankar et al. (1998) also develop a trial–repeat diffusion model to investigate differences in pioneers, innovative late entrants, and non-innovative late entrants' strategies. They extend the Bass model to include competitors' sales, marketing spending, and repeat purchase variables. Furthermore, they follow Hahn et al.'s method (Hahn et al., 1994) to differentiate between trial sales and cumulative sales; however, their model does not include competitive marketing expenditures. Instead, they assume that competitors' sales moderate own marketing effectiveness. They do not

Table 1
Marketing communication characteristics of the trial–repeat diffusion studies.

Marketing communication:	Lilien et al. (1981)	Mahajan et al. (1983)	Rao and Yamada (1988)	Hahn et al. (1994)	Shankar et al. (1998)	Ataman et al. (2008)	This study
"Push" strategy	Detailing	Not included	Detailing	•Detailing •Medical journal advertising	•Detailing •Medical journal advertising	Not included	•Detailing •Medical journal advertising •Physicians' meetings
"Pull" strategy	Not included	Not included	Not included	Not included	Not included	•Features/displays •Advertising	•Direct-to-consumer Advertising
Heterogeneity in the effects of the marketing activities	No	No	No	No	No	Yes	Yes
Competitor's marketing activities (separated from own)	Yes	No	Yes	No	No	Yes	Yes
Extended model	Mixed influence diffusion model	Mixed influence diffusion model	Mixed influence diffusion model	Mixed influence diffusion model	Mixed influence diffusion model	External influence diffusion model	Mixed influence diffusion model
Influence on the trial rate through…							
external influence	Considered	Not considered	Considered	Considered	Not considered	Considered	Considered
internal influence	Not considered	Not considered	Not considered	Not considered	Not considered	Not considered	Considered
both external and internal influence	Not considered	Not considered	Not considered	Not considered	Considered	Not considered	Considered
… is investigated in separate models							

empirically investigate where to locate variables that represent marketing efforts in the model, and they assume that marketing expenditures have the same impact on external and internal influences.

Ataman et al. (2008) extend Hahn et al.'s method (Hahn et al., 1994) by formulating a multivariate Bayesian dynamic linear model of repeat purchase diffusion, which offers several advantages. However, their model does not accommodate internal influence, which greatly reduces multicollinearity problems but also hampers its applicability to the pharmaceutical sector, because network effects such as word of mouth play essential roles in the diffusion of new brands (Peres et al., 2010).

Desiraju et al. (2004) extend Van den Bulte (2000) internal diffusion model to analyze a new category of antidepressant drugs (i.e. second-generation pharmaceuticals). Specifically, they propose a model based exclusively on the internal influence, which means that their model does not accommodate repeat sales.

This discussion indicates that the models proposed by Hahn et al. (1994) and Shankar et al. (1998) provide good starting points for our study. Because we do not focus on late mover advantages but rather on the effects of own and competitive marketing efforts on the diffusion process, we extend Hahn et al.'s simpler model. In Table 1, we summarize how our study compares with earlier empirical studies that employ trial–repeat diffusion models.

Following Hahn et al. (1994), we incorporate three components of new product buying behavior:

1. Innovative behavior, or the adopters' basic tendency to innovate (or innate innovativeness), which is influenced by sources of external communication.
2. Imitating behavior, or the tendency to adopt an innovation as a result of interpersonal influence processes (word-of-mouth or internal communication).
3. Repeat buying.

The sum of the first two components represents the trial rate, which can be multiplied by sales of the effective potential market (m_t, total market potential at time t, minus potential sales to all previous adopters at time $t-1$, q_{t-1}) to arrive at trial sales. We include repeat sales as a separate term in the diffusion model and assume it is a fraction of the potential sales to all physicians that adopted the new product before time t.

Initially, we follow Hahn et al. (1994) and consider the first trial component to be time varying. We assume that it depends on a linear combination of own and competitive marketing expenditures. Subsequently, we also specify models where marketing expenditures (1) affect the second trial component, and (2) affect both components.

Adopters might be either active users (i.e., repeaters) or inactive users (i.e., non-repeaters) of the new product. The first group is more likely to engage in social contagion. Because sales in the previous period provide a proxy for the size of this group, we include this variable in the model. Similar to Hahn et al. (1994), we specify the second component as proportional to the sales of the adopters in the previous time period. Thus, we include $s_{i,t-1}/m_t$ to measure the internal influence effects.

The following diffusion model emerges:

$$s_{i,t} = \left[\beta_{10i} + \beta_{11i}\ln(x_{i\cdot t}) + \beta_{11ci}\ln(x_{c\cdot t}) + \beta_{2i}\left[\frac{s_{i,t-1}}{m_t}\right]\right]\left[m_t - q_{i,t-1}\right] + \beta_{3i}q_{i,t-1} \quad (1)$$

where $q_{i,t} - q_{i,t-1} = s_{i,t} - \beta_{3i}q_{i,t-1}$, $x_{i\cdot t} = \sum_{j=1}^{4} x_{ij,t}$, and $x_{c\cdot t} = \sum_{j=1}^{4} x_{cj,t}$. For brand i ($i = 1, \ldots, N$) in month t ($t = 1, \ldots, T$),

$s_{i,t}$ = sales, or the sum of trial and repeat purchases;
$x_{ij,t}$ = own marketing expenditures on instrument j ($j = 1$ is detailing; $j = 2$ medical journal advertising; $j = 3$ physician meetings; $j = 4$ direct-to-consumer advertising);
$x_{cj,t}$ = competitors' marketing expenditures on instrument j;
m_t = total market potential; and
$q_{i,t-1}$ = potential sales to physicians in the post-trial segments (triers, repeaters, and buyers of competing brands that have tried brand i before).

The parameter β_{10i} indicates the basic propensity to try brand i in the absence of the influence of marketing expenditures and prior adopters. The effect of own (competitors') promotional activities on the trial rate of brand i is captured by β_{11i} (β_{11ci}). The parameter β_{2i} captures the effect of internal influence on the trial rate, and β_{3i} is the repeat rate.[2]

The three components of Model (1) therefore can be expressed as:

a) $(\beta_{10i} + \beta_{11i}\ln(x_{i\cdot t}) + \beta_{11ci}\ln(x_{c\cdot t}))[m_t - q_{i,t-1}]$;
b) $\beta_{2i}\left[\frac{s_{i,t-1}}{m_t}\right][m_t - q_{i,t-1}]$; and
c) $\beta_{3i}q_{i,t-1}$.

The first component of Model (1) represents sales to triers of brand i due to the external influence (which is influenced by the company's and competitors' promotional efforts) – innovative behavior – the second component represents triers of brand i due to the internal influence – imitating behavior or word-of-mouth communication – and the third component repeaters of brand i. Model (1) assumes that marketing expenditures affect the trial rate of the diffusion process through an external influence. However, extant literature remains inconclusive about how to include marketing instruments into a diffusion model. Therefore, unlike the previous studies, we do not assume a priori that marketing expenditures affect external and/or internal influences. A formulation analogous to Eq. (1) that accommodates the effects of marketing expenditures on internal influence appears in Eq. (2), and a model that allows for the effects of marketing expenditures on both internal and external influence can be found in Eq. (3):

$$s_{i,t} = \left[\beta_{10i} + (\beta_{2i} + \beta_{21i}\ln(x_{i\cdot t}) + \beta_{21ci}\ln(x_{c\cdot t}))\left[\frac{s_{i,t-1}}{m_t}\right]\right] \times \left[m_t - q_{i,t-1}\right] + \beta_{3i}q_{i,t-1}, \text{ and} \quad (2)$$

$$s_{i,t} = \left[\beta_{10i} + \beta_{2i}\left[\frac{s_{i,t-1}}{m_t}\right]\right](1 + \beta_{41i}\ln(x_{i\cdot t}) + \beta_{41ci}\ln(x_{c\cdot t})) \times \left[m_t - q_{i,t-1}\right] + \beta_{3i}q_{i,t-1} \quad (3)$$

with $q_{i,t} - q_{i,t-1} = s_{i,t} - \beta_{3i}q_{i,t-1}$.

Our models are based on three restrictive assumptions. First, we do not consider the influence of marketing on the size of the potential market to ensure the tractability of the model estimation. Second, as Hahn et al. (Hahn et al., 1994) argue, "the direct experience coming from product trial should be more influential in the repeat market than indirect experience" (see also Smith and Swinyard (1982)), so we exclude the influence of marketing on the repeat rate, which again enhances the tractability of the model estimation. Third, we do not incorporate price nor distribution channels. Intermediaries such as insurance firms, health maintenance organizations, or government agencies cover most of the cost of the prescription drugs (Manchanda et al., 2005), so that the insured patients likely are not price sensitive and have little awareness of the retail price of their prescription. Physicians, working in the interest of the patients, also do not have a financial stimulus to be price sensitive. Furthermore, they tend to be unaware of the retail price of specific drugs (Narayanan et al., 2005); (Newhouse, 1993). Gönül et al. (2001) find that considerations about drug efficacy and patients' conditions represent the primary drivers in the decision process, clearly overriding price concerns. This is in line with Leeflang and Wieringa (2010), who find that price does not affect prescription behavior. For these reasons, we do not include the price variable in our diffusion models.[3] We also do not consider the distribution variable, because pharmaceutical firms do not differ in the distribution channels they employ.

We estimate six versions of our model that we classify into two subsets (see Table 2). The models in subset 1 accommodate own marketing instruments' effect on the trial rate and assume that the effects of competitive marketing efforts are absent ($\beta_{k1ci} = 0$ for $k = 1, 2, 4$). In subset 2, we consider both own and competitive marketing instruments' effects on the trial rate. Within both subsets, we estimate several versions that are based on the external influence model (Eq. (1); indicated as E in Table 2), the internal influence model (Eq. (2); I in Table 2), and the external and internal influence model (Eq. (3); EI in Table 2).

The external influence formulation – Model 1E – corresponds to the model proposed by Hahn et al. (1994). The external and internal influence formulations – Models 1EI and 2EI – are in line with the Generalized Bass Model proposed by Bass et al. (1994). They introduce advertising and price into the Bass model by assuming that marketing efforts affect the trial rate through external as well as internal influence. In the case of Models 1E, 1I, 2E and 2I, the marketing variables are assumed to moderate the diffusion process through external or internal influence.

To employ an aggregated variable to model the effects of marketing instruments on the diffusion process implies that important information is lost about how different types of marketing variables have unique effects. We also explore disaggregated marketing effects by proposing several versions of our models (Eq. (1), Eq. (2) and Eq. (3)) that allow for

[2] The parameter β_{3i} can also be interpreted as "the long-run average market share of the new product because the total market is represented by the repeat market after the trial market is saturated" (Hahn et al., 1994, p.229).

[3] Cf. [16, p. 246].

Table 2
Versions of the proposed model in its external, internal and external and internal influence formulations.

———————— Subset 1 ————————

External influence (Model 1E)

$$s_{i,t} = \left[\beta_{10i} + \beta_{11i}\ln(x_{i\cdot t}) + \beta_{2i}\left[\frac{s_{i,t-1}}{m_t}\right]\right]\left[m_t - q_{i,t-1}\right] + \beta_{3i}q_{i,t-1}$$

Internal influence (Model 1I)

$$s_{i,t} = \left[\beta_{10i} + (\beta_{2i} + \beta_{21i}\ln(x_{i\cdot t}))\left[\frac{s_{i,t-1}}{m_t}\right]\right]\left[m_t - q_{i,t-1}\right] + \beta_{3i}q_{i,t-1}$$

External and internal influence (Model 1EI)

$$s_{i,t} = \left[\beta_{10i} + \beta_{2i}\left[\frac{s_{i,t-1}}{m_t}\right]\right](1 + \beta_{41i}\ln(x_{i\cdot t}))\left[m_t - q_{i,t-1}\right] + \beta_{3i}q_{i,t-1}, \text{ where } x_{i\cdot t} = \sum_{j=1}^{4} x_{ij,t}$$

———————— Subset 2 ————————

External influence (Model 2E)

$$s_{i,t} = \left[\beta_{10i} + \beta_{11i}\ln(x_{i\cdot t}) + \beta_{11ci}\ln(x_{c\cdot t}) + \beta_{2i}\left[\frac{s_{i,t-1}}{m_t}\right]\right]\left[m_t - q_{i,t-1}\right] + \beta_{3i}q_{i,t-1}$$

Internal influence (Model 2I)

$$s_{i,t} = \left[\beta_{10i} + (\beta_{2i} + \beta_{21i}\ln(x_{i\cdot t}) + \beta_{21ci}\ln(x_{c\cdot t}))\left[\frac{s_{i,t-1}}{m_t}\right]\right]\left[m_t - q_{i,t-1}\right] + \beta_{3i}q_{i,t-1}$$

External and internal influence (Model 2EI)

$$s_{i,t} = \left[\beta_{10i} + \beta_{2i}\left[\frac{s_{i,t-1}}{m_t}\right]\right](1 + \beta_{41i}\ln(x_{i\cdot t}) + \beta_{41ci}\ln(x_{c\cdot t}))\left[m_t - q_{i,t-1}\right] + \beta_{3i}q_{i,t-1}, \text{ where } x_{i\cdot t} = \sum_{j=1}^{4} x_{ij,t} \text{ and } x_{c\cdot t} = \sum_{j=1}^{4} x_{cj,t}$$

For all models: $q_{i,t} - q_{i,t-1} = s_{i,t} - \beta_{3i}q_{i,t-1}$.

heterogeneity in the effects of the different marketing variables apart from differences in the effects of own and competitor's marketing efforts (see Table A1 in Appendix A1). However, the estimation of these versions suffers from multicollinearity of the marketing instruments (see e.g. Lilien et al. (1981); Gatignon et al. (1990); Hahn et al. (1994); Rizzo (1999)). Multicollinearity between the expenditures for the different instruments can be explained by the fact that new pharmaceutical products typically receive strong promotional support using all available instruments during market introduction, and much less in later periods. Consequently, for a given drug, the longitudinal pattern for the different marketing instruments is quite similar. Hence, we decided to discard the models in subsets 3 and 4 (Table A1 in Appendix A1), and continued investigating the different versions in subsets 1 and 2 (Table 2).

We use three criteria to select the most appropriate model: (1) the Akaike Information Criterion to select the most parsimonious specification with the best goodness-of-fit values, (2) the parameter stability measures proposed by Golder and Tellis (1998), and (3) the face validity of the estimates. We do not consider predictive validity because our model is descriptive rather than predictive, and sales forecasts in any future period require knowledge of the size of the potential market in the same period, which in turn depends on the value of sales in that period.

In a second-stage analysis, we determine the cross-sectional effects of marketing expenditures on the trial rate and the repeat rate when we account for different effects of different marketing strategies.

4. Sample, data, and measurement of the variables

We use monthly US data pertaining to three categories of pharmaceuticals that belong to the "Top-10 markets" of prescription drugs in the United States in 2000: rhinitis, osteoarthritis and rheumatoid arthritis (ORA), and asthma. Rhinitis is a reaction that occurs in the eyes, nose, and throat when airborne irritants (allergens) trigger the release of histamines, which then cause inflammation and fluid production in the fragile linings of nasal passages, sinuses, and eyelids. All drugs in the category are functionally equivalent in that they all treat the same medical symptoms. This category is highly competitive and contains only branded drugs; no generic drugs entered the market during the observation period, but of the 16 branded drugs in this category, 14 were introduced during the observation period (1993–2000). The ORA and asthma categories contain a lower proportion of new drugs (9 out of 20 and 11 out of 26, respectively), but the asthma category resembles the rhinitis category in terms of symptoms and seasonal patterns, whereas the ORA category is completely different. The ORA and asthma categories contain both branded and generic items.[4]

The data describe sales and expenditures for detailing, medical journal advertising, physician meetings, and direct-to-consumer (DTC) advertising. Expenditures on advertising directed at physicians are greater than DTC expenditures, and detailing is the primary promotional activity directed at physicians. However, for the majority of the brands, expenditures on DTC advertising are higher than expenditures on medical journal advertising and physician meetings. The historical data come from syndicated secondary data sources.

Prior to parameter estimation, we deflate the promotional expenditures by the consumer price index (1982 = 1.00) to obtain the real expenditures for each drug in constant dollars. The seasonal adjustment (we use the Census X-11 seasonal adjustment method) removes seasonal patterns in the sales time series, such as demand fluctuations due to allergy seasons. Expenditures for promotional activities in a period influence the drug's sales in the following period, so we lag the promotional activities by 1 month, similar to Lilien et al. (1981) and Hahn et al. (1994). However, because Gönül et al. (2001) and Berndt et al. (2003) use marketing stock variables with a different number of lags to account for carryover effects in the promotional activities, we also estimate versions of the model using stock variables with a different number of lags, but

[4] Details and descriptive statistics about the drugs analyzed are available from the authors upon request.

the option of one lag offers the best results.[5] By applying natural logarithms to the marketing variables, we account for diminishing returns to scale for marketing actions in the model.

5. The impact of pharmaceutical marketing on the diffusion of new prescription drugs

In this section we explore the longitudinal effects of pharmaceutical marketing investment on the diffusion processes of new drugs. We estimate six versions of our model (see Table 2) using the iterative ordinary least squares procedure developed by Hahn et al. (1994).[6] Models 1EI and 2EI suffer from multicollinearity and hence the specifications adopting a GBM like approach to capturing the effects of marketing investments were never selected as the best model. In order to conserve space, we do not show the estimation results of these model specifications (these are available from the authors upon request). Instead, we compare the external formulations of Models 1 and 2 with the corresponding internal formulations. Table 3 summarizes the model selection criteria.

Focusing on Criterion 1, the Akaike Information Criterion reveals that, although differences are very small, Model 2E is preferred for the majority of the drugs, especially for the Rhinitis category. Model 2E is preferred in 16 out of 34 cases (47%) followed by Model 2I in 8 out of 34 cases (24%).[7]

Criterion 2 is used to evaluate parameter stability. Following Golder and Tellis (1998) we estimate each model repeatedly, starting with a short data series (T = 87 periods) and adding one additional period on every iteration. We use the two measures of parameter stability proposed by these authors: one captures fluctuations from the overall mean–STAB1–and another captures period to period fluctuations–STAB2. Higher values of STAB1 indicate that the model presents greater parameter stability, and lower values of STAB2 indicate that the model presents greater parameter stability. Table 3 shows that: i) $\hat{\beta}_{10i}$ and $\hat{\beta}_{3i}$ of model 1E are more stable than those of model 1I, but not for $\hat{\beta}_{2i}$; ii) $\hat{\beta}_{10i}$ of model 2E is more stable than that of model 2I, the opposite happens with $\hat{\beta}_{2i}$, and there is no conclusive result on $\hat{\beta}_{3i}$; and, iii) $\hat{\beta}_{21i}$ of model 2E is more stable than that of model 1E, $\hat{\beta}_{11i}$ of model 2I is more stable than that of model 1I, and there is no conclusive result on $\hat{\beta}_{10i}$, $\hat{\beta}_{2i}$ and $\hat{\beta}_{3i}$. In general, an external formulation yields a slightly larger parameter stability than an internal formulation – see i) and ii). Furthermore, these results also show that the inclusion of competitor's marketing efforts yields a slightly larger parameter stability than assuming only own marketing efforts – see iii). Hence, although to a lesser extent, model 2E is the preferred specification for the majority of the drugs for three categories analyzed.

Also according to Criterion 3 (face validity of the estimates) the external influence models perform better than the internal influence models (76% and 73% of the cases, respectively). The last five lines of Table 3 also show that if we compare the models focusing only on the basic diffusion parameter estimates ($\hat{\beta}_{10i}$, $\hat{\beta}_{2i}$ and $\hat{\beta}_{3i}$), the external formulations and in particular Model 2E are preferred. The face validity of the estimates is very relevant in our study, because for the cross-sectional analysis (described in the following section), we use the estimates of the selected diffusion model from the longitudinal analysis. Again, with this criterion, Model 2E is preferred.

Although no model dominates the others for all branded items (Parker and Gatignon, 1994), we conclude that Model 2E (the model in which own and competitive marketing affect the diffusion pattern via external influence) is the most appropriate model for most items. We provide the estimation results from Model 2E for the three different categories in Tables 4, 5, and 6, each of which consists of five columns-blocks. Column-block 1 shows the identification numbers of the brands (presented in order of brand introduction). Column-block 2 in each table contains the estimates for the trial rate parameters that capture external influence (β_{10i}, β_{11i} and β_{11ci} in Model 2E) and the internal influence parameter (β_{2i}). In column-block 3, we present the estimates for the repeat rate parameter (β_{3i}); column-block 4 reveals the average market share of each brand. Because market share should approach the repeat rate, this column is an indication for the face validity of the repeat rate estimates. Finally, the fifth column-block presents the goodness-of-fit statistics (mean absolute deviation [MAD], mean absolute percentage error [MAPE], and r, or the correlation between the observed and the estimated values of the dependent variables[8]). Tables 4–6 only include parameter estimates that differ significantly from 0, and the results are obtained after mean-centering the marketing variables to make the estimates for β_{10i} comparable across brands. These estimates therefore represent the basic propensity to try a new product in conditions with average marketing expenditures and the absence of internal influence effects. We perform several residual diagnostic checks and conclude that neither the homoskedasticity nor the nonnormality assumption is violated. For some brands, the residuals show positive autocorrelation. We reestimate the models for these brands using generalized least squares and observe only minor changes in the estimation results.

The estimation results for Model 2E for the rhinitis category in Table 4 indicate that β_{10i} estimates are all significant and within the expected range. The significant estimates for the effect of own marketing expenditures on the trial rate (β_{11i}) are positive except for brand 8, and for five brands the estimate is not significant. Competitors' marketing expenditures generally have a negative effect on the trial rate ($\hat{\beta}_{11ci}<0$); however, for five brands, the estimate is insignificant. The significant estimates of β_{2i} are within the expected range. The estimates

[5] Specifically, we tried marketing stock variables with a different number of lags (0, 1, 2, …, 12) and different lag specifications (e.g., exponentially declining weights, parabola weights).

[6] We also extended a naive model (without marketing variables) by considering a time-varying repeat rate, allowing the repeat rate to depend on the number of competitors in the market. The results were similar to those of a naive model with a constant repeat rate. Extending Model 1 in a similar fashion, resulted in a smaller number of significant estimates due to increased multicollinearity. Hence, we decided to retain the model specifications with a static repeat rate.

[7] We also employ likelihood ratio tests to identify the most parsimonious specification among the nested models, considering a naive model (without marketing variables), Model 1 and Model 2 (Ramanathan, 1993). The null hypothesis of these tests corresponds to the most restricted model under consideration; the alternative hypothesis to the less restricted model. None of the models dominate the others in all cases. However, theses test in general also select Model 2E as the best model.

[8] We report r instead of R^2 or adjusted-R^2 because the models under consideration do not have an intercept term (Judge et al., 1985).

Table 3
Model selection criteria.

Criterion 1: Akaike Information Criterion[a]

Rhinitis category					ORA category					Asthma category				
Brand code	Mod.1E	Mod.1I	Mod.2E	Mod.2I	Brand code	Mod.1E	Mod.1I	Mod.2E	Mod.2I	Brand code	Mod.1E	Mod.1I	Mod.2E	Mod.2I
10	9.70	9.71	9.72	**9.64**	3	11.27	10.53	10.63	**10.25**	26	7.29	7.32	**7.13**	7.35
6	11.63	11.62	**11.25**	11.26	12	11.99	11.92	11.22	**10.97**	18	**7.62**	7.77	7.64	7.78
13	8.38	8.46	**7.90**	8.31	13	9.77	**9.49**	9.58	9.49	10	7.18	7.20	**7.15**	7.17
7	12.27	12.28	**11.43**	11.63	4	11.23	11.18	**10.12**	11.08	3	10.43	10.39	10.38	**10.32**
9	9.73	9.75	**9.19**	9.32	11	7.56	**6.55**	6.97	6.58	13	**5.23**	**5.23**	5.25	5.26
11	3.97	4.04	**3.85**	3.97	5	7.44	7.45	**7.05**	7.18	11	8.94	8.84	8.80	**8.62**
4	**5.55**	**5.55**	5.57	**5.55**	1	8.29	8.24	8.16	**7.95**	1	7.45	7.44	**7.42**	7.43
15	**9.67**	9.68	9.68	9.69	2	11.79	12.24	**11.73**	12.09	9	**7.50**	7.54	7.54	7.57
1	10.65	10.66	**10.52**	10.57	19	10.39	10.52	**10.34**	10.48	17	**8.09**	8.12	8.15	8.17
8	5.95	5.96	**5.87**	5.89						20	7.73	7.69	**7.33**	7.58
16	7.02	**7.01**	7.06	7.04										
3	7.83	7.84	**7.62**	7.70										
12	**8.59**	8.67	8.62	8.72										
2	10.80	10.79	10.73	**10.70**										

Criterion 2: Measures of stability of parameters estimates (mean values)

	β_{10}	β_{11}	β_{11c}	β_2	β_{21}	β_{21c}	β_3
STAB1[b]							
Model 1E	**48.076**			21.673	*−3.193*		97.908
Model 1I	*47.079*	*4.509*		**46.821**			97.818
Model 2E	40.896			14.513	**2.234**	−9.055	**171.053**
Model 2I	39.776	**6.673**	−8.606	*43.497*			*142.677*
STAB2[c]							
Model 1E	0.019			0.430	*0.252*		**0.028**
Model 1I	*0.019*	*0.202*		*0.054*			0.078
Model 2E	**0.018**			0.120	**0.241**	0.099	0.034
Model 2I	0.021	**0.160**	0.080	**0.041**			*0.031*

Criterion 3: Face validity

	Relative frequency[d]							
	Rhinitis category		ORA category		Asthma category		Overall	
All the parameters in the model								
Model 1E: $\beta_{10}\ \beta_{11}\ \beta_2\ \beta_3$	41/56	(73%)	*27/36*	*(75%)*	**28/40**	**(70%)**	*96/132*	*(73%)*
Model 1I: $\beta_{10}\ \beta_{21}\ \beta_{21c}\ \beta_3$	33/56	(59%)	24/36	(67%)	26/40	(65%)	83/132	(63%)
Model 2E: $\beta_{10}\ \beta_{11}\ \beta_{11c}\ \beta_2\ \beta_3$	**55/70**	**(79%)**	**36/45**	**(80%)**	*34/50*	*(68%)*	**125/165**	**(76%)**
Model 2I: $\beta_{10}\ \beta_2\ \beta_{21}\ \beta_{21c}\ \beta_3$	*54/70*	*(77%)*	31/45	(69%)	30/50	(60%)	115/165	(70%)
Basic diffusion parameters: $\beta_{10}\ \beta_2\ \beta_3$								
Model 1E	*38/42*	*(90%)*	*24/27*	*(89%)*	*26/30*	*(87%)*	*88/99*	*(89%)*
Model 1I	31/42	(74%)	21/27	(77%)	23/30	(77%)	75/99	(76%)
Model 2E	**39/42**	**(93%)**	**26/27**	**(96%)**	**27/30**	**(90%)**	**92/99**	**(93%)**
Model 2I	*38/42*	*(90%)*	23/27	(85%)	24/30	(80%)	85/99	(86%)

[a] Lowest AIC values (per brand within each category) are bold-faced.
[b] STAB1 $= \frac{\text{Mean}(\hat{\beta})}{\text{Stand.dev.}(\hat{\beta})}$, $\hat{\beta}$ represents the estimates. Highest STAB1 values are bold-faced; 2nd highest STAB1 values are in italics.
[c] STAB2 $= \sum \left| \frac{\hat{\beta}_t - \hat{\beta}_{t-1}}{\text{mean}\left(\hat{\beta}\right)} \right| \frac{1}{K}$, $\hat{\beta}$ represents the estimates and K the number of estimation periods. Lowest STAB2 values are bold-faced; 2nd lowest STAB2 values are in italics.
[d] Amount of significant estimates within the expected range vs total number of parameters in the model. Highest relative frequencies are bold-faced; 2nd highest relative frequencies values are in italics.

in Table 4 thus agree with outcomes of a meta-study by Sultan et al. (1990), who find that estimates for β_{10i} are positive and (much) smaller than the estimated internal influence coefficient ($\hat{\beta}_{2i}$), as is the case for all estimates for β_{10i}. Finally, the estimates for β_{3i} are significant, except for brand 4, and the values of $\hat{\beta}_{3i}$ are very similar to the average market share, as we expected.

We obtain similar estimation results of Model 2E for the ORA and asthma categories (see Tables 5 and 6). We observe the following exceptions: For the ORA category, only three significant estimates emerge for the effect of own marketing expenditures on the trial rate, one of which (brand 1) has the wrong sign. Eight out of nine estimates for competitors' marketing expenditures are significant, and one (brand 13) has a positive sign. In the asthma category, all four significant estimates for the effect of own marketing expenditures on the trial rate are positive, whereas competitors' marketing expenditures indicate a negative and significant effect on the trial rate for three brands but a positive and significant effect for two brands.

Table 4
Estimation results of Model 2E: Rhinitis category.

Brand code	Trial rate				Repeat rate				
	External influence			Internal influence		Average market share	MAD	MAPE	r
	$\hat{\beta}_{10i}$	$\hat{\beta}_{11i}$	$\hat{\beta}_{11ci}$	$\hat{\beta}_{2i}$	$\hat{\beta}_{3i}$	(in units)			
10	0.04^{***}			1.29^{***}	0.01^{***}	0.03	22.81	8.81	0.92
6	0.09^{*}	0.02^{o}	-0.07^{***}		0.32^{***}	0.20	47.74	3.56	0.99
13	0.03^{***}	0.01^{***}	-0.01^{***}	1.47^{***}	0.04^{***}	0.03	7.17	3.65	0.99
7	0.07^{***}	0.02^{o}	-0.07^{***}	1.47^{***}	0.17^{***}	0.13	42.48	5.01	0.98
9	0.06^{***}	0.003^{**}	-0.02^{***}	0.63^{***}	0.11^{***}	0.09	13.53	2.41	0.99
11	0.01^{***}	0.0004^{***}	-0.0003^{*}	0.99^{***}	0.004^{***}	0.01	0.76	1.99	0.99
4	0.01^{***}			0.84^{***}		0.01	1.66	2.54	0.95
15	0.05^{***}			0.24^{*}	0.13^{***}	0.10	13.84	2.08	0.99
1	0.03^{***}	0.01^{*}	-0.01^{*}		0.19^{***}	0.11	19.59	2.66	0.99
8	0.02^{***}	-0.001^{**}	0.001^{*}	0.53^{***}	0.01^{***}	0.02	1.74	1.53	0.98
16	0.01^{***}			0.61^{***}	0.05^{***}	0.02	2.11	1.63	0.99
3	0.01^{***}	0.002^{*}	-0.003^{**}	0.39^{*}	0.02^{***}	0.01	3.28	4.20	0.93
12	0.03^{***}	0.002^{*}		0.40^{***}	0.09^{***}	0.06	4.38	1.11	0.99
2	0.03^{***}		-0.01^{*}	0.61^{**}	0.07^{***}	0.05	9.01	2.57	0.95

*** $p \leq 0.0001$.
** $p \leq 0.001$.
* $p \leq 0.05$.
^{o} $p < 0.1$.

6. The role of pharmaceutical marketing on the trial and repeat rates

In the previous section we consider the *within-brand*, longitudinal effects of marketing expenditures on the diffusion process for each brand separately. In this section we investigate *between-brand* differences in the diffusion process, and focus on how these are caused by differences in marketing expenditures across brands. These cross-sectional analyses allow us to explore the *informative* and *persuasive functions* of pharmaceutical marketing on the diffusion process of the analyzed new prescription drugs. We investigate whether $\hat{\beta}_{10i}$ (basic propensity to try the new product), $\hat{\beta}_{2i}$ (internal influence parameter), and $\hat{\beta}_{3i}$ (repeat rate) depend on the average marketing support during the introduction period of the brand's life cycle. We also consider whether marketing expenditures explain differences in the estimates of the trial rate parameters ($\hat{\beta}_{10i}$ and $\hat{\beta}_{2i}$) during the first 12 months after introduction (informative effects). By focusing on the first year after introduction, we acknowledge that marketing expenditures are significantly higher during this period than later in the product life cycle for most brands. In addition, we expect the effects of marketing variables on trial to be most pronounced during the introduction phase. To determine the *persuasive function* of marketing, we evaluate the relationship between the repeat rate ($\hat{\beta}_{3i}$) and marketing efforts during the first 12 months after introduction. In addition, because the repeat rate relates to creating market power (which also has a longer-term perspective), we analyze its relationship with marketing expenditures over the complete period.

Because not all the rhinitis, ORA, and asthma categories have sufficient new brand introductions during the data observation period, we do not perform separate category-level analyses but instead pool data of the three categories to determine cross-sectional marketing effects. We regress $\hat{\beta}_{10i}$, $\hat{\beta}_{2i}$, and $\hat{\beta}_{3i}$ on the mean of the log of marketing efforts. Previous studies detect that the entry strategy into the marketplace affects the diffusion process of new products. Rao and Bass (1985) and

Table 5
Estimation results of Model 2E: ORA category.

Brand code	Trial rate				Repeat rate				
	External influence			Internal influence		Average market share	MAD	MAPE	r
	$\hat{\beta}_{10i}$	$\hat{\beta}_{11i}$	$\hat{\beta}_{11ci}$	$\hat{\beta}_{2i}$	$\hat{\beta}_{3i}$	(in units)			
3	0.04^{***}		-0.05^{***}	0.89^{***}	0.06^{***}	0.03	38.96	11.63	0.92
12	0.13^{***}		-0.13^{***}	0.80^{***}	0.14^{***}	0.10	44.00	4.67	0.96
13	0.02^{***}	0.001^{*}	0.02^{**}	1.91^{***}	0.02^{***}	0.01	20.63	18.12	0.93
4	0.03^{***}		-0.03^{***}	1.21^{***}	0.03^{***}	0.03	18.61	7.77	0.88
11	0.02^{***}		-0.003^{***}	0.99^{***}	0.01^{**}	0.01	3.40	3.33	0.98
5	0.02^{***}		-0.003^{***}	0.55^{***}	0.02^{***}	0.02	2.91	1.73	0.93
1	0.03^{***}	-0.003^{*}	-0.003^{*}	1.05^{***}	0.01^{**}	0.02	3.65	1.77	0.98
2	0.11^{***}	0.05^{*}	-0.02o		0.20^{***}	0.17	14.18	0.88	0.98
19	0.08^{***}			0.47^{***}	0.20^{***}	0.12	5.09	0.43	0.99

*** $p \leq 0.0001$.
** $p \leq 0.001$.
* $p \leq 0.05$.
^{o} $p < 0.1$.

Table 6
Estimation results of Model 2E: Asthma category.

Brand code	Trial rate				Repeat rate	Average market share	MAD	MAPE	r
	External influence			Internal influence					
	$\hat{\beta}_{10i}$	$\hat{\beta}_{11i}$	$\hat{\beta}_{11ci}$	$\hat{\beta}_{2i}$	$\hat{\beta}_{3i}$	(in units)			
26	0.004^{***}	0.001^{**}	-0.002^{*}		0.03^{***}	0.02	4.85	9.47	0.89
18	0.03^{***}	0.003^{***}		0.32^{***}	0.07^{***}	0.04	7.42	2.11	0.99
10	0.02^{***}		0.002^{*}	1.12^{***}	0.01^{***}	0.01	4.29	4.97	0.93
3	0.18^{***}		0.04^{*}	0.22^{**}	0.28^{***}	0.24	19.53	1.02	0.99
13	0.01^{***}			0.79^{***}	0.02^{***}	0.01	1.20	1.36	0.99
11		0.003^{*}	-0.01^{*}		0.28^{***}	0.05	7.10	1.87	0.99
1	0.03^{***}			0.96^{***}	0.02^{***}	0.02	3.19	1.80	0.98
9	0.02^{***}			0.56^{***}	0.06^{***}	0.03	3.05	1.18	0.99
17	0.01^{***}			0.44^{*}	0.03^{*}	0.01	3.28	6.48	0.85
20	0.02^{***}	0.003^{**}	-0.01^{**}	0.20^{**}	0.18^{***}	0.06	2.33	0.52	0.99

*** $p \leq 0.0001$.
** $p \leq 0.001$.
* $p \leq 0.05$.

Schmalensee (1982) suggest that only trial sales reflect order-of-entry effects, whereas Kalyanaram and Urban (1992) find order-of-entry effects for both trial and repeat sales in consumer good markets. In the pharmaceutical markets, Shankar et al. (1998) find that innovative late entrants diffuse faster than the pioneer. We follow existing studies and include an order-of-entry variable. We investigate both aggregate and disaggregate marketing efforts by distinguishing between the effects of direct-to-physician marketing expenditures (push strategy) and DTC advertising expenditures (pull strategy). Because the ORA and asthma categories have both branded and generic new drugs, we include a dummy variable (labeled "generic drug") that takes the value of 0 if the new product is a generic item and 1 if it is a branded item. We use OLSDV pooling and account for heteroskedasticity between brands and categories (Wittink, 1977). Table 7 shows the results.

The first three columns of Table 7 show the effects of the expenditures on direct-to-physician marketing ("push" strategy), the expenditures on DTC advertising ("pull" strategy), the order of entry, and the kind of drug (branded or generic) on $\hat{\beta}_{10i}$, $\hat{\beta}_{2i}$, and $\hat{\beta}_{3i}$ during the 12 months after introduction. The effects of the marketing expenditures shown in the first three columns refer to the *informative function* as well as to the *persuasive function* of marketing during the first 12 months after introduction. The expenditures on direct-to-physician marketing have a positive and significant effect on $\hat{\beta}_{10i}$ and $\hat{\beta}_{3i}$ but an insignificant effect on $\hat{\beta}_{2i}$. In addition, DTC expenditures have insignificant effects on $\hat{\beta}_{10i}$, $\hat{\beta}_{2i}$, and $\hat{\beta}_{3i}$. Our results thus suggest that in the 12 months after introduction, direct-to-physician marketing mainly affects the adoption rate through $\hat{\beta}_{10i}$. We find no significant effect of DTC advertising, which is in line with the meta-analysis of Kremer et al. (1992).

Furthermore, order of entry has a negative (though marginally significant) effect on $\hat{\beta}_{2i}$, which indicates physicians' preference for early entrants. This may be explained by the fact that physicians have more experience with earlier entrants, which increases perceptions that the drug is effective and safe (Berndt et al., 2003). This may make them somewhat reluctant toward trying later entrants. The coefficient for the "generic drug" variable is negative and significant for $\hat{\beta}_{10i}$, so new generic drugs tend to prompt a greater propensity to try than do newly introduced branded drugs.

In the last two columns of Table 7, we report two additional analyses of the repeat rate. The last column shows the effects of aggregate marketing expenditures (i.e. persuasive role), the order of entry, and the kind of drug on $\hat{\beta}_{3i}$ using data of complete data observation period. For this analysis, it is not possible to separate the effects of the push

Table 7
Pooled results for the basic propensity to try ($\hat{\beta}_{10i}$), internal influence coefficient ($\hat{\beta}_{2i}$), and repeat rate ($\hat{\beta}_{3i}$).

	Informative effects			Persuasive effects	
	12-Month period			12-Month period	Complete period
	$\hat{\beta}_{10i}$	$\hat{\beta}_{2i}$	$\hat{\beta}_{3i}$	$\hat{\beta}_{3i}$	$\hat{\beta}_{3i}$
Constant – Rhinitis category	0.08^{**}	1.10^{***}	0.10^{*}	0.11^{o}	0.09^{*}
Constant – ORA category	0.06^{*}	1.21^{***}	0.4	0.04	0.06
Constant – Asthma category	0.06^{*}	0.73^{*}	0.10^{o}	0.10^{*}	0.10^{*}
Mean log marketing expenditures				0.01^{*}	0.01^{***}
Direct-to-physician	0.005^{*}	−0.01	0.01^{o}		
Direct-to-consumer	0.001	−0.07	0.005		
Order of entry	−0.002	-0.04^{o}	−0.003	−0.004	−0.004
Generic drug	-0.10^{*}	0.13	0.13	-0.12^{o}	-0.14^{*}
R^2 (%)	31.46	37.34	18.29	18.00	41.06

*** $p \leq 0.0001$.
** $p \leq 0.001$.
* $p \leq 0.05$.
^{o} $p < 0.1$.

and pull strategies due to multicollinearity, So we cannot compare the parameter estimates of the 12-month period analysis and the complete-period analysis of the repeat rate. To facilitate this comparison, we present the results of the analysis with aggregate marketing expenditures for the 12-month period in the preceding column.

Focusing on the analysis with disaggregate marketing expenditures, we find that expenditures on DTC advertising have an insignificant effect on $\hat{\beta}_{3i}$, which implies that only marketing directed at physicians affects the repeat rate. When we analyze the complete period, the marketing efforts have a positive and significant impact on the repeat rate, and generic new drugs experience a higher repeat rate.

Because of the many new brands in the rhinitis category, we analyze this category separately and derive estimation results similar to those of the pooled analysis, except with regard to the order of entry effect on the trial rate. These results indicate that the sooner a new branded drug enters the market, the higher the propensity to try it ($\hat{\beta}_{10i} = -0.003$; p-value ≤ 0.05), and the greater the effectiveness of the physicians' internal influence ($\hat{\beta}_{10i} = -0.06$; p-value ≤ 0.05).

In summary, marketing activities increase the propensity to try new brands (i.e. the information effect). We find that they affect the adoption rate through external influence, and not via internal influence (Model 2E is a more appropriate model than 2I). Marketing activities also increase the repeat rate (i.e. the persuasion effect) where marketing activities aimed at physicians have a stronger effect on the repeat rate than on the propensity to try. Thus, we find that both the *informative* and *persuasive functions* of marketing activities exist. Furthermore, the results show that DTC advertising ("pull" strategy) seems to affect neither the internal influence nor the propensity to try; it also does not affect the repeat rate during the first 12 months after introduction. On average, generic new drugs create greater propensities to try and repeat rates than do branded drugs. Order of entry appears to have no significant impact on the repeat and trial rates when we analyze the three categories jointly, but category-specific analyses[9] shows that it becomes more important in the comparatively younger rhinitis category than in the other two "older" categories, in which many generic drugs compete with branded drugs.

7. Summary and conclusions

We explore the role of pharmaceutical marketing investment on the diffusion process of new prescription drugs. We employ a family of trial–repeat diffusion models that enable us to determine the appropriate location for marketing instruments in the trial rate, as well as accommodate a time-varying trial rate influenced by both own and competitors' marketing expenditures. We employ the trial–repeat diffusion model to decompose total sales into a trial and a repeat component. In a second-stage analysis, we determine the cross-sectional effects of marketing expenditures on the trial rate and repeat rate by accounting for different effects of marketing strategies. We also disentangle the informative and persuasive roles of marketing.

Using 8 years of US monthly data pertaining to 34 drugs in three therapeutic categories, we find support for the hypothesis that changes in the trial rate relate positively to changes in own marketing expenditures. We also find support for the hypothesis that competitors' marketing expenditures negatively affect the trial rate. These results mirror those of previous research in this area (Lilien et al., 1981; Hahn et al., 1994). Moreover, they indicate that for the three categories under study, the most appropriate way to include marketing variables in a trial–repeat diffusion model is through an external influence formulation.

Pharmaceutical industry is a very competitive setting where new drugs are continuously introducing and pharmaceutical marketing can incentivize their diffusion processes. Incorporating marketing instruments into the diffusion model improves our understanding of the diffusion process of prescription drugs. Managers can improve the trial rate of their new drugs by investing in their own marketing instruments, but they must note that their trial rate will decrease with competitors' marketing expenditures.

Cross-sectional differences in marketing expenditures significantly affect the trial and repeat rates of the diffusion processes of new pharmaceutical products. A pooled analysis of the three focal drug categories demonstrates that direct-to-physician marketing affects both trial and repeat rates, whereas DTC advertising has no effect on the adoption neither the repeat rate during the first 12 months after introduction (Kremer et al., 1992). We also find that generics tend to prompt a higher basic propensity to try and a higher repeat rate than do branded drugs. Furthermore, in the rhinitis category, which contains many new and only branded drugs, the order of entry appears to play an important role in the launch strategy of the new products, such that earlier entrance creates barriers against the entry of future competitors.

These results agree with the findings of Leffler (1981), Narayanan et al. (2005) and Ching and Ishihara (2012), who find both informative (indirect) and persuasive (direct) effects of pharmaceutical marketing. Thus, the differentiation between a push strategy such as direct-to-physician marketing and a pull strategy such as DTC advertising represents a relevant consideration for managers.

Pharmaceutical companies can increase consumers' (physicians') propensity to try new brands and their repeat rate by intensifying marketing activities, which not only stimulates trial of new pharmaceutical drugs but also protects the new drug's market share from competitors' marketing activities. During the first 12 months after the introduction of a new drug, managers should concentrate their efforts mainly on direct-to-physician activities, that allow pharmaceutical companies to increase the trial rate. These efforts also create entrance barriers through the increase of the repeat rate of their new drugs.

This finding is supported by a simulation study that is shown in Appendix A2. We also show that a small market share brand is that doubling the marketing expenditures and employing an exponential declining spending strategy elongate its maturity stage, and hence significantly increase its ROI. This effect is not found in the case of a large market brand. Our simulation study discusses other situations where we show that employing an appropriate marketing spending strategy has a great influence on sales, market share, ROI and the length of the stage of product life cycles. However, managers should take into account that the increase of the total marketing budget does not affect the saturation level.

[9] The category-specific analyses are not shown here due to space limitations; the results are available upon request from the first author.

Although our diffusion model relaxes some restrictive assumptions of previous trial–repeat diffusion models, several limitations remain. First, we assume that the number of physicians in the product category is fixed. Relaxing this assumption would make the model considerably more complex (Hahn et al., 1994). Second, we assume all consumers purchase in the product category in each period (i.e., all physicians prescribe at least one drug within the rhinitis, ORA, and asthma categories), which is not particularly problematic for monthly data. Third, the model assumes that all physicians belong to the same class, even though Lilien et al. (1981) point out that this assumption can be relaxed by constructing a series of parallel processes for each class of doctors. Fourth, we do not consider the effect of promotional efforts on the repeat rate but instead obtain those effects only through a second-stage analysis. Fifth, multicollinearity problems, which are almost always manifest in models that study the effect of different marketing expenditures on pharmaceutical sales, prohibit a more disaggregated investigation into the effects of the different marketing instruments (Simon and Sebastian, 1987; Hahn et al., 1994). Finally, we do not have data on the number of free drug samples that are distributed to physicians. There is evidence that these can affect prescription trial of new drugs (Joseph and Mantrala, 2009).

Further research should develop models of the effects of marketing on all three diffusion parameters simultaneously. Applying new approaches, research could analyze the impact of the different marketing instruments on the diffusion parameters over time. In this respect, recursive window regression might be an appropriate technique (Pauwels and Hanssens, 2007).

Appendix A1

Table A1 shows six additional versions of Model (1). For the versions in subset 3 (Models 3E, 3I and 3EI), we assume that the marketing instruments that are aimed at the physician all have the same effect on the trial rate. However, these specifications accommodate differences in the effects of the consumer-directed and physician-directed instruments. These versions are more flexible than those included in subsets 1 and 2 (see Table 2) because they disaggregate promotional efforts from both own and competing drugs into "pull" and "push" effects. In these specifications, direct-to-consumer advertising represents the "pull" effect and detailing, medical journal advertising and physician meetings represent the "push" effect. The versions in subset 4 (Models 4E, 4I and 4EI) are the most flexible versions as they allow for heterogeneity in the effects of the different marketing variables.

Appendix A2

We illustrate the implications of our results in a simulation study, in which we focus on the effects of an increase and the reallocation of a given marketing budget over time. The three panels on the left of Fig. A.1 (a, c, and e) display the effect of a reallocation of a relatively small marketing budget over time, whereas those on the right (b, d, and f) reveal the same analysis with a marketing budget that is twice as large.

The solid line in Panel a of Fig. A.1 shows the observed marketing expenditures of an arbitrary brand introduced in the middle of the observation period, which owns a small market share in a large market. The dotted and dashed lines in Panel a represent two different marketing expenditure strategies, namely, a uniform spending pattern and a spending pattern that heavily supports the introduction of a new brand and then allows expenditures to decline exponentially in subsequent periods, respectively. All marketing expenditure strategies have the same budget, so the sum of the expenditures over

Table A1
Additional versions of the proposed model in its external, internal and external and internal influence formulations.

———————— Subset 3 ————————

External influence (Model 3E)

$$s_{i,t} = \left[\beta_{10i} + \beta_{11i}\ln(x_{i1,t}) + \beta_{12i}\ln(x_{i\cdot t}) + \beta_{11ci}\ln(x_{c1,t}) + \beta_{12ci}\ln(x_{c\cdot t}) + \left(\beta_{2i}\left[\tfrac{s_{i,t-1}}{m_t}\right]\right)\right][m_t - q_{i,t-1}] + \beta_{3i}q_{i,t-1}$$

$$q_{i,t} - q_{i,t-1} = s_{i,t} - \beta_{3i}q_{i,t-1}$$

Internal influence (Model 3I)

$$s_{i,t} = \left[\beta_{10i} + (\beta_{2i} + \beta_{21i}\ln(x_{i1,t}) + \beta_{22i}\ln(x_{i\cdot t}) + \beta_{21ci}\ln(x_{c1,t}) + \beta_{22ci}\ln(x_{c\cdot t}))\left[\tfrac{s_{i,t-1}}{m_t}\right]\right][m_t - q_{i,t-1}] + \beta_{3i}q_{i,t-1}$$

$$q_{i,t} - q_{i,t-1} = s_{i,t} - \beta_{3i}q_{i,t-1}$$

External and internal influence (Model 3EI)

$$s_{i,t} = \left[\beta_{10i} + \beta_{2i}\left[\tfrac{s_{i,t-1}}{m_t}\right]\right](1 + \beta_{41i}\ln(x_{i1,t}) + \beta_{42i}\ln(x_{i\cdot t}) + \beta_{41ci}\ln(x_{c1,t}) + \beta_{42ci}\ln(x_{c\cdot t}))[m_t - q_{i,t-1}] + \beta_{3i}q_{i,t-1}$$

$$q_{i,t} - q_{i,t-1} = s_{i,t} - \beta_{3i}q_{i,t-1}$$

where $x_{i\cdot t} = \sum_{j=2}^{4} x_{ij,t}$ and $x_{c\cdot t} = \sum_{j=2}^{4} x_{cj,t}$

———————— Subset 4 ————————

External influence (Model 4E)

$$s_{i,t} = \left[\beta_{10i} + \sum_{j=1}^{4}\beta_{1ji}\ln(x_{ij,t}) + \sum_{j=1}^{4}\beta_{1jci}\ln(x_{cj,t}) + \beta_{2i}\left[\frac{s_{i,t-1}}{m_t}\right]\right][m_t - q_{i,t-1}] + \beta_{3i}q_{i,t-1}$$

$$q_{i,t} - q_{i,t-1} = s_{i,t} - \beta_{3i}q_{i,t-1}$$

Internal influence (Model 4I)

$$s_{i,t} = \left[\beta_{10i} + \left(\beta_{2i} + \sum_{j=1}^{4}\beta_{2ji}\ln(x_{ij,t}) + \sum_{j=1}^{4}\beta_{2jci}\ln(x_{cj,t})\right)\left[\tfrac{s_{i,t-1}}{m_t}\right]\right][m_t - q_{i,t-1}] + \beta_{3i}q_{i,t-1}$$

$$q_{i,t} - q_{i,t-1} = s_{i,t} - \beta_{3i}q_{i,t-1}$$

External and internal influence (Model 4EI)

$$s_{i,t} = \left[\beta_{10i} + \beta_{2i}\left[\tfrac{s_{i,t-1}}{m_t}\right]\right]\left(1 + \sum_{j=1}^{4}\beta_{4ji}\ln(x_{ij,t}) + \sum_{j=1}^{4}\beta_{4jci}\ln(x_{cj,t})\right)[m_t - q_{i,t-1}] + \beta_{3i}q_{i,t-1}$$

$$q_{i,t} - q_{i,t-1} = s_{i,t} - \beta_{3i}q_{i,t-1}$$

time remains the same for the observed, exponentially declining, and uniform strategies.

Panel c of Fig. A.1 shows the changes in the diffusion patterns as a result of the reallocation of marketing expenditures. The solid line indicates the fitted diffusion pattern with the observed marketing expenditures, whereas the dotted and dashed diffusion patterns correspond to the uniformly distributed and exponentially decaying marketing spending patterns, respectively. Several studies indicate that a monotonic declining spending pattern is optimal for new products (Horsky and Simon, 1983; Horsky and Mate, 1988). We observe that a heavily supported introduction speeds the diffusion process considerably (Panel c, dashed line), but the subsequent lower level of marketing expenditures leads to a lower sales level at the end of the observational period compared with the two alternative allocations of the same marketing budget. Because the observed expenditures are approximately uniformly distributed over time, its diffusion pattern is very similar to that of the uniform allocation, except with regard to the start of the diffusion pattern. Specifically, the initial diffusion is slightly faster for the uniform spending pattern, due to a small initial delay in the observed expenditures. However, this shift does not have a permanent effect on the level of sales.

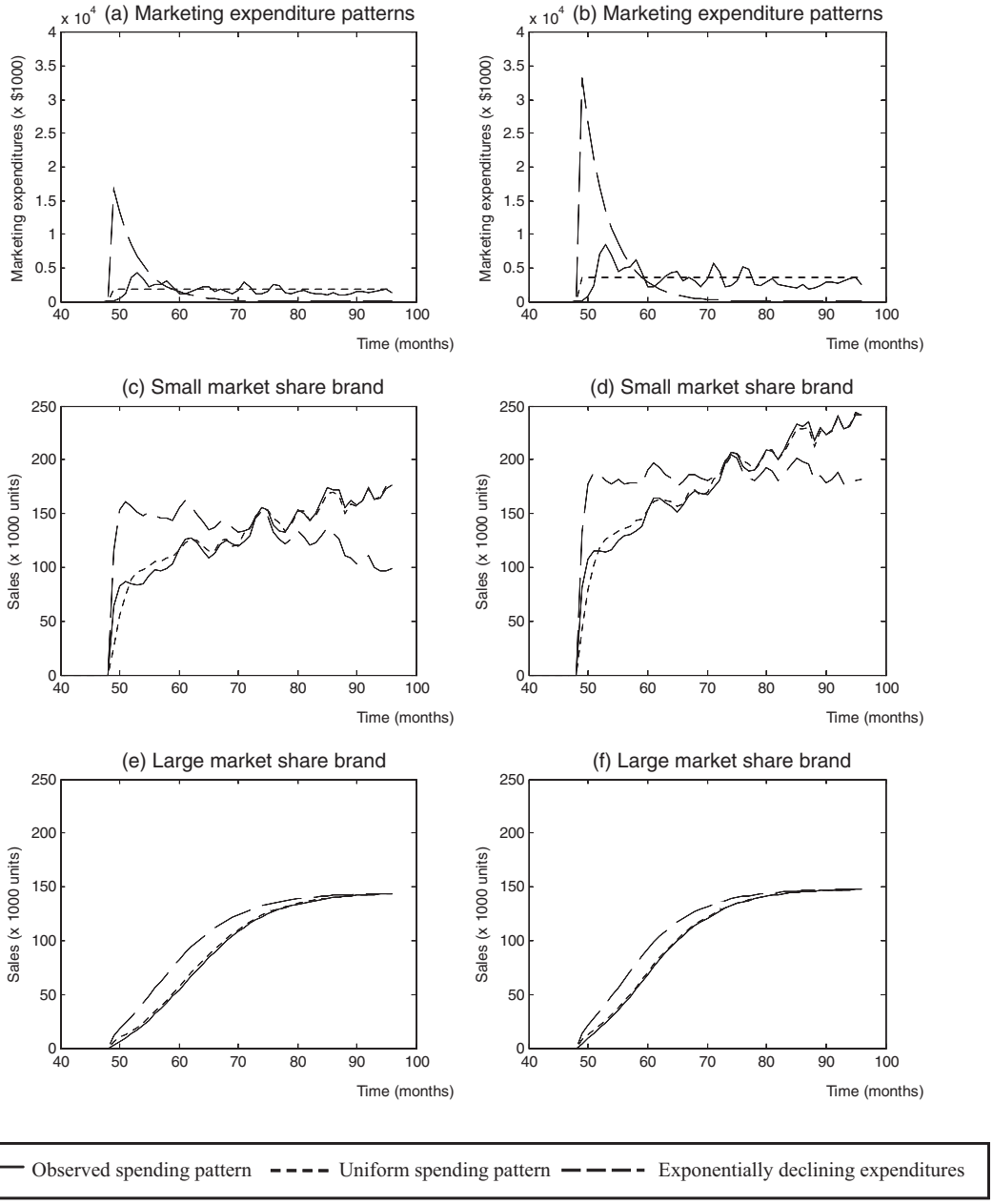

Fig A.1. Effect of different marketing expenditure patterns and different marketing budgets on the diffusion of brands with small and large market shares.

The decaying marketing expenditure pattern ultimately leads to a lower level of sales because the focal brand owns only a small market share. For a small market share brand, $m_t - q_{i,t-1}$ remains relatively large, so the relative sales effect of the first component in Eq. (1) later in the product life cycle is relatively larger than it would be for a brand with a larger market share.

In Panel e of Fig. A.1, we use the same reallocation of marketing expenditures over time but simulate a brand with a larger market share in a smaller market. We observe that the positive effect of strong support during the introduction is not followed in this case by a decline due to falling marketing expenses subsequently.

In the right-hand side panels, we reiterate the simulation but increase the marketing budget to twice its original size (cf. Panels a and b). When we recompute the coefficients of the diffusion process according to the results of the cross-sectional analysis, Panel d shows that the absolute sales level of the small market share brand benefits from the increase in the marketing budget. After 4 years on the market, the sales levels are 40–75% higher, depending on the spending strategy. With the exponentially declining spending strategy, doubling the marketing budget elongates the maturity stage of the small market share brand.

Panel f also shows that increasing the marketing budget does not have a significant effect on the long-run sales level of the larger market share brand; absolute sales in month 96 are only about 3% higher as a result of doubling the marketing expenditures, irrespective of the spending strategy. It therefore may be tempting to conclude that neither increasing the marketing budget nor the spending strategy influences large market share brands.

However, this conclusion is not valid when we consider cumulative sales levels. Due to a steeper initial sales increase in Panel f cumulative sales shortly after the introduction are two to three times greater for an exponentially declining spending pattern (dashed line in Panel f) compared with the sales levels of the original budget and spending pattern (solid line in Panel e). For example, for the small market share brand, the cumulative sales after 4 months with a smaller marketing budget and observed spending pattern equal 31.6 units, whereas the cumulative sales are 96.9 units with the larger budget and the exponentially decaying spending pattern. This effect becomes smaller over time, but after 4 years on the market, the percentage difference in cumulative sales remains as great as 19%. Doubling the budget but maintaining the original spending pattern leads to a 9% increase in cumulative sales after 4 years and a 10% increase with the uniform spending pattern. Therefore, in this case, the return on investment (ROI) more than doubles with a spending strategy that accounts for the effect of marketing variables on diffusion parameters.

This simulation study shows that (1) a reallocation of a given marketing budget over time can have large effects on the speed of diffusion; (2) the size of this effect is moderated by the market share of the brand; and (3) increasing the total marketing budget does not affect the saturation level.

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