Chapter 6

Diffusion of prescription drugs in the United States of America

6.1. Introduction

The issue of costs and benefits of prescription drug promotion has been the subject of ongoing debate between opponents and proponents of the pharmaceutical industry and has long been a focal point in policy discussions between regulatory agencies and the industry. Pharmaceutical marketing has been criticized as wasteful and excessive and for contributing to the overuse, misuse, and misprescription of drugs (Families USA, 2002). However, marketing may also serve as a key communication channel for continuing physician education regarding pharmaceutical products and for exposing consumers to information that may improve health outcomes (Rubin, 2003).

For the pharmaceutical industry innovations are vital and expenditures on Research and Development are very high. For the industry it is important to discover factors that accelerate adoption of newly developed products so that the likelihood of a profitable R&D investment can be maximized. This is also important from the patient's point of view as health outcomes improve via rapid diffusion of new drugs that provide unique and superior benefits. In this study we focus on estimating the effects of the different marketing instruments on the diffusion process of new prescription drugs, and also on the analysis of separate effects of marketing directed at physicians ("push" strategy) and direct-to-consumer advertising ("pull" strategy).

We use a trial-repeat purchase diffusion model that we calibrate for each drug in the category of rhinitis drugs. This category contains only branded products, most of which are introduced during the observational period (1993-2000). We also calibrate the model using data of two other categories: osteoarthritis-rheumatoid-arthritis and asthma. The rhinitis and the asthma categories are quite similar (they both treat the respiratory organs and display seasonal patterns), whereas the osteoarthritis-rheumatoid-arthritis category is completely different from the other

two. The osteoarthritis-rheumatoid-arthritis category and the asthma category are much older than the rhinitis category. Hence, in contrast to the rhinitis category, these do not only contain branded products but also generics.

The trial-repeat purchase diffusion model that we develop is an extension of the model of Hahn et al. (1994). We propose to incorporate the effect of company and competitors' promotional efforts separately. In contrast to other studies that either use aggregate measures for marketing expenditures or expenditures for a single instrument (Lilien, Rao and Kalish 1981, Rao and Yamada 1988, Hahn et al., 1994), our model accommodates heterogeneity in the effects of different marketing instruments. Following existing literature, we assume that the trial rate is time varying and that it depends on marketing expenditures.

However, unlike previous research on trial-repeat diffusion models that assume *a priori* that marketing instruments affect the trial rate through the external influence, we first analyze a family of diffusion models that allows us to detect the appropriate allocation for marketing instruments in the trial rate. We hypothesize that longitudinal relationships exist both for own-brand and competitors' marketing efforts.

We also accommodate so-called cross-sectional effects: we investigate how marketing expenditures affect the basic propensity to try a new product, internal influence and the repeat rate. We investigate these effects by performing a second-stage analysis on the results of the diffusion model, which are obtained separately for multiple brands.

In summary, our contributions are:

- we investigate both longitudinal and cross-sectional effects of marketing expenditures on the diffusion of pharmaceutical products;
- our model accommodates heterogeneity in the effects of the different marketing instruments;
- we do not make *a priori* assumptions about how the allocation of marketing instruments affects the diffusion process. We determine empirically whether the marketing instruments influence the diffusion process through the trial rate (through the internal and/or external influence) or through the repeat rate;
- our model specification accommodates the effects of both own and competitors' marketing expenditures on the diffusion process;
- we separately determine the effect of marketing expenditures on the trial rate ("informative" function) and on the repeat rate ("persuasive" function);
- our results show that the diffusion process is clearly affected by marketing directed at physicians and slightly affected by direct-to-consumer advertising;
- our results provide support for the existence of both longitudinal and cross-

sectional diffusion effects in the market of three drug categories: rhinitis, osteoarthritis-rheumatoid-arthritis and asthma.

The results of this study allow us to perform an in-depth analysis of the impact of the different marketing instruments on the time-varying diffusion parameters. We use a recursive time window approach and show preliminary results. These results suggest that marketing directed at physicians, especially detailing and then physician meetings, affect the longitudinal pattern of the diffusion parameters.

The rest of this chapter is set up as follows. In Section 6.2 we focus on the importance of marketing in the pharmaceutical industry, especially for new products, and the buying decision process in this industry. In Section 6.3 we provide a brief literature review of research addressing diffusion models for non-durable products that accounts for repeat purchases and that analyzes the impact of marketing variables on the diffusion process. We also review existing trial-repeat diffusion models validated on pharmaceutical products. In Section 6.4 we specify our diffusion model. In Sections 6.5 and 6.6 we present the data and the estimation results, respectively. Finally, we discuss our conclusions in Section 6.7.

6.2. The Pharmaceutical Industry

In this section we discuss the main characteristics of the pharmaceutical industry, we point out the relevance of pharma marketing and explain the particular buying decision process in this industry.

The pharmaceutical industry has been the most profitable industry in the USA, as measured by median return on revenue, for each of the 10 years before 2002 (Families USA, 2002). In Table 6.1 we show that the top 9 pharmaceutical companies spend on average 11 percent ($19 billion) of total revenue on R&D. The US General Accounting Office (a research bureau of the US Congress) reports that the US Pharmaceutical firms spent $30.3 billion on R&D in 2001 (US General Accounting Office, 2002). The competitive advantage of a new pharmaceutical product is a temporary impact due to patent expiration. Hence, it is of interest to determine factors that accelerate the diffusion process, for example to recover the R&D costs more quickly. We focus on marketing as a possible means for increasing the speed of diffusion. According to Families USA (2002), "*The drug industry pumps huge sums of money into marketing because it works. Advertising and marketing help drive sales, and top-selling drugs can generate large revenues*" (p.13). Leffler (1981, p.52) points out that "*prescription drugs are one of the most heavily promoted products in the American economy*". In the US, the marketing

expenditures are somewhere between $10 and 20 billion annually (see, e.g. Breitstein, 2002). Pharma marketing expenditures[1] potentially help the pharmaceutical industry to quickly recover the R&D costs by providing useful and beneficial information to physicians and patients. "*This is the role of marketing – providing information to decision makers*" (Rubin, 2003, p.7). Another possible effect of marketing activities is that they might create effective barriers for new entrants so that market shares are protected.

Table 6.1.
2001 Financials for U.S. Corporations Marketing the Top 50 Drugs for Seniors

Company	Revenue (Net Sales in Millions of Dollars)	Percent of Revenue Allocated to:		
		Marketing/ Advertising/ Administration	R & D	Profit (Net Income)
Merck & Co., Inc.	$47,716	13%	5%	15%
Pfizer, Inc.	$32,259	35%	15%	24%
Bristol-Myers Squibb Company	$19,423	27%	12%	27%
Abbott Laboratories	$16,285	23%	10%	10%
Wyeth	$14,129	37%	13%	16%
Pharmacia Corporation	$13,837	44%	16%	11%
Eli Lilly & Co.	$11,543	30%	19%	24%
Schering-Plough Corporation	$9,802	36%	13%	20%
Allergan, Inc.	$1,685	42%	15%	13%
Total* (Dollars in millions)	**$166,678**	**27%** $45,413	**11%** $19,076	**18%** $30,599

* Totals may not add due to rounding.

Source: Families USA (2002)

The instruments of pharmaceutical marketing

When considering the strategic issues facing medical marketers in the pharmaceutical industry we distinguish "push" and "pull" strategies. The pharmaceutical industry has traditionally used a "push" strategy focusing their promotional budget directly on the physicians. The most important promotional

[1] See Rubin (2003) for a detailed explanation of the allocation of pharma marketing expenditures.

instruments that are used with this strategy are detailing, physician meetings and seminars, medical journal advertising, samples and direct mail. Detailing is the name of the promotional activity that consists of sales representatives (detailers) visiting physicians in order to provide information on e.g. appropriate drug usage (efficacy, indications, contra-indications, side effects, etc.), modes of therapy, prices, etc. Physician meetings and seminars involve talks that are organized or sponsored by pharmaceutical companies where experts discuss the treatment of specific diseases or illnesses. Medical journal advertising refers to advertisements for specific pharmaceuticals in medical journals. Samples refer to the free product samples distributed by pharmaceutical firms. Direct mail includes the printed material sent out to physicians as information aids. Among the previous promotional activities, detailing has been and continues to be the primary form of promotion directed at physicians.

Another strategy is to use direct-to-consumer advertising (i.e. promotional activities used by pharmaceutical firms directed at consumer). Direct-to-consumer advertising can be classified as a "pull" strategy. Consumers tend to have positive attitudes toward direct-to-consumer ads. Handlin, Mosca, Forgione and Pitta (2003) show that approximately 10 million people requested an advertised drug from their doctor in 1997. These positive attitudes towards direct-to-consumer advertisements are relevant given that they might lead patients to increase compliance. Due to direct-to-consumer advertising patients are more educated in terms of what types of drugs and treatments are available to them (Butler, 2002). Direct-to-consumer advertising can make someone aware that he or she may have a treatable condition, for example through an advertisement explaining the symptoms of depression (Rubin, 2003). Direct-to-consumer advertising is gaining a relevant place in pharma marketing, particularly in the USA, one of the few countries where direct-to-consumer advertising is allowed. In the USA, direct-to-consumer advertising used to be heavily restricted until 1997, when the Food and Drug Administration (FDA) changed its ruling and relaxed their restrictions significantly.

Parker and Pettijohn (2003) report that direct-to-consumer advertising costs increased from approximately $40 million in 1989 to $160 million in 1994 and to 350$ million by 1995. By 1996 the expenditures on direct-to-consumer advertising was estimated to have doubled to approximately $700 million. The US General Accounting Office shows that expenditures on direct-to-consumer advertising increased by 145 percent between 1997 and 2001. In 1997, direct-to-consumer advertising accounted for 10 percent of total spending on promotion, whereas in 2001 it accounted for almost 14 percent ($2.7 billion dollars, see US General Accounting Office, 2002). Recent research shows that promotional expenditures on direct-to-consumer advertising are concentrated on a small number of medications and that promotions directed to physicians remain dominant, but that direct-to-consumer advertising has become key for a subset of medications (Ma, Stafford,

Cockburn and Finkelstein, 2003). Wittink (2002), in a study of 392 branded drugs, reports that the firms under study invested approximately 8.5 billion dollars on direct-to-physician marketing in 2000 and 2.5 billion on direct-to-consumer advertising.

Pharmaceutical firms use both promotion directed at physicians and direct-to-consumer advertising to inform the market about their products. The use of both strategies has important implications, given that with the "pull" strategy the patients could play a role in the decision what drug is prescribed. However, existing research (Rosenthal, Berndt, Donohue, Frank and Epstein, 2002; Wosinska, 2002; Xie, 2003) reveals that although direct-to-consumer advertising allows patients to be better informed about their health conditions, this does not affect the physician's decision.

The role of pharma marketing on the trial rate (external and internal influences)

Pharma marketing activity can influence the trial rate of the new prescription drugs. In the trial rate of new drugs we differentiate to types of influences: external and internal influence (see Chapter 2). External influence is related to the innovative behavior of physicians, which can be modified by promotional activities developed by firms. Marketing activities of pharmaceutical products may allow for better health care and lower costs as it helps physicians to keep up-to-date with medical treatments, therapies and medications (Mossinghoff, 1992). Internal influence is related to the interpersonal communication among physicians about the new drugs introduced into the market; i.e. the influence resulting from interaction between previous and potential adopters of the new drugs. The more widely a drug is used, the more we can expect this drug to be effective and safe. For example, physicians may assume that the probability of a malpractice suit is lower for an existing brand than for a new drug. Temin (1980) suggest that since physicians do not have access to well-organized data that allow them to make comparisons about the efficacy and risk of substitute drugs, their decisions about drugs are based on the customary behavior of other physicians (Berndt, Pindyck and Azoulay, 2003). Pharma marketing can improve this situation by offering useful information on new pharmaceutical products. Hence, pharma marketing can affect physician's behavior through both types of influences and play a relevant role in the physician's decision to prescribe a new drug.

The role of pharma marketing on the repeat rate

Prescription drugs belong to the category of frequently purchased products where adopters may switch from one product to another substitute product in the short term. Therefore, the role of pharma marketing activities is also very relevant here as these activities may influence a physician's decision to prescribe the same

(new) drug instead of a substitute drug. Pharma marketing may help to a physician that his/her initial choice is the most appropriate for patients' health outcomes, and in that manner reduce the probability of a switch to a competing drug.

Pharma marketing: information or persuasion?

The literature distinguishes two functions for pharma marketing activities: an "informative" and a "persuasive" function (Leffler, 1981; Hurwitz and Caves, 1988; Rizzo, 1999). It is of interest for managers to know their role on the diffusion processes of new pharmaceutical products.

When there are several competing products available to treat a certain disease, a physician may have selected a "preferred product", possibly based on efficacy considerations, severity of side effects, etc[2]. With the introduction of a new alternative the physician may reconsider his/her choice of a preferred drug. The outcome of this reconsideration may be influenced by the amount of information that is provided by the manufacturers of the new product. That is, marketing activities allow physicians to update their prior beliefs about the new product. Hence, pharmaceutical marketing may influence the diffusion process of a new product through the trial rate. This is what some authors call the "informative" function of marketing activities. The role marketing plays in providing information about new treatments and drugs is crucial to health care. This is especially important in the pharmaceutical industry where developing successful new products is of vital importance. If physicians and patients do not have sufficient information about how and why to use a new drug, their interest in the new drug will be low whatever the price. In the USA pharmaceutical industry, which spent over $20 billion promoting and marketing their products in 2002, pharma marketing is the main source of information on new cures, treatments and medications (Manning and Masia, 2003). As pointed out by Rubin (2003, p.12) "*The absence of information would severely restrict access to innovative products. Note that the spread of new medicines has been shown to reduce other types of health care spending such as hospitalization, implying that without information on new medicines, overall health spending might increase*".

The "persuasive" function of pharma marketing refers to the influence that marketing activities have in creating market power for the promoted product. In this sense, the "persuasive" function of pharma marketing builds barriers of entry to competitors into the marketplace. Hurwitz and Caves (1988) find that marketing activities protect the market share of innovators when generics enter the market.

[2] We note that part of this selection procedure may also be performed by health maintenance organizations -HMOs- or insurance companies that select preferred products based on benefit/cost considerations. These preferences show up in formularies (drugs for which the HMO provides full or partial coverage in the form of reimbursements).

Hence, this "persuasive" function influences the diffusion process of a new product through the repeat rate.

Other authors distinguish between the indirect ("informative") and direct ("persuasive") effects of pharma marketing communication. The studies of Erdem and Keane (1996) on laundry detergents, Currie and Park (2002) on prescription of antidepressants, and Ackerberg (2003) on yogurt have found evidence only for the indirect effect ("informative" function) of marketing communication; others like those of Anand and Shachar (2001) on Television shows and Narayanan, Manchanda and Chintagunta (2004) on prescription of antihistamines have found evidence for both the indirect ("informative") and direct ("persuasive") effects[3]. Hence, there is evidence for both the "informative" and "persuasive" function of pharma marketing communication.

The buying process of pharmaceuticals

In the remainder of this section we focus on the buying decision process in the pharmaceutical industry. In this industry we distinguish two processes depending on the kind of drug considered: the buying decision process in the over-the-counter (OTC) drug market and the buying decision process in the prescription drug market.

The OTC drug market shows the traditional process where the decision maker is also the end-user of the product (Akçura, Gönül and Petrova, 2004). However, in the prescription drug market, the users are not the decision makers. The users, patients, may influence the decision-making process but the physicians prescribe the (prescription) drugs after which patients purchase the drugs. Hence, although the consumers are the users, they are not the decision makers. The decision to choose a specific drug can be influenced by both patients and physicians, but the ultimate decision is made by the physician.

Another important difference between the OTC and prescription drug markets that affects the buying decision process is related to price. Intermediaries such as insurance firms, health maintenance organizations -HMOs- or government agencies pay for more of the cost of the prescription drugs. Hence, one might expect that insured patients have little awareness of the full price, and hence will not be very sensitive to it. In the United States about 87 percent of the population -approximately 221 million adults between the ages of 18 and 64- have some kind of health care insurance (Narayanan, Manchanda and Chintagunta, 2004) that includes coverage of prescription drugs. The physicians, working in the interest of the patient, have no financial stimulus either to be price sensitive and they tend to be ignorant of the full price of specific drugs (Hurwitz and Caves, 1988). Newhouse (1993) finds no evidence that physicians prescribe lower-price drugs to

[3] For more details see Narayanan, Manchanda and Chintagunta (2004).

patients who have less generous insurance coverage. Hellerstein (1998) does report that physicians are somewhat price sensitive for prescription drugs when generics compete with branded drugs. Her study reports evidence that physicians are more likely to prescribe generic than branded drugs when they have a relative large number of patients covered by insurance plans. Real competition in the pharmaceutical industry comes when generics enter the marketplace; generic drugs are about half the full price of branded drugs in the first year after a generic is introduced in the market (Families USA, 2002). Gönül, Carter, Petrova and Srinivasan (2001) find evidence that considerations about drug efficacy and patients' conditions are the primary drivers in the decision process, overriding price concerns. In general, price is a factor of little concern to physicians and patients in the case of health insurance coverage.

6.3. Literature review

The literature on the diffusion of new products reveals the wide acceptance of the Bass model (Mahajan, Muller and Wind, 2000). However, this model is built on assumptions that reduce its applicability (see Chapter 2, Section 2.4), but at the same time invite researchers to extend the model. We focus on two of these assumptions that make the Bass model less appropriate for analyzing diffusion processes of new drugs. The Bass model assumes that each adopter purchases the new product only once (see Chapter 2, Section 2.4.2.4). This assumption reduces the applicability of the model to durables (without a replacement option), and makes it less suitable for non-durables that involve repeated purchases. Another restrictive assumption of the Bass model is that the impact of the marketing variables is implicitly captured by the model parameters (see Chapter 2, Section 2.4.2.9). This assumption makes it impossible to analyze the effect of marketing instruments on the diffusion process of the innovation. In the pharmaceutical industry where firms invest a huge amount of dollars in marketing, the analysis of the effects of pharma marketing is an essential issue to managers.

Several authors like Dodson and Muller (1978), Dolan and Jeuland (1981), Lilien, Rao and Kalish (1981), Jeuland and Dolan (1982), Mahajan, Wind and Sharma (1983), Parker and Gatignon (1994) and Hahn et al. (1994) modified the Bass model to allow for non-durable products and repeat purchases. Several of these applications deal with pharmaceutical products. In this respect Lilien, Rao and Kalish (1981) and Hahn et al. (1994) propose trial-repeat diffusion models with promotional efforts. Rao and Yamada (1988) validate the work of Lilien, Rao and Kalish (1981) on twenty products. Mahajan, Wind and Sharma (1983) focus on the non-uniform nature of the internal influence.

The incorporation of marketing mix variables into diffusion models is important because by measuring the effects of marketing on the diffusion process, marketing strategies for new products may be improved (Mahajan and Muller, 1979; Kalish and Sen, 1986; Mahajan and Wind, 1986; Mahajan, Muller and Bass, 1990; Bass, Jain and Krishnan, 2000). Given the evident importance of marketing variables on the diffusion process of innovations, many authors have proposed diffusion models that incorporate marketing variables and have provided empirical support (see Chapter 2, Section 2.4.2.9). Most of these studies focus on durable products, and the empirical evidence is not conclusive. Concerning the effect of advertising on diffusion rate: Horsky and Simon (1983) and Simon and Sebastian (1987) find that advertising affects the diffusion rate of banking telephoning and telephones, respectively. Horsky and Simon (1983) investigate only one possibility: advertising affects the diffusion rate through the external influence. However, Simon and Sebastian (1987) consider that advertising can affect external and/or internal influence; their results show that, although advertising efforts affect both external and internal influence, the model that considers that advertising only affects the internal influence is slightly superior. Bass, Krishnan and Jain (1994) introduce advertising into the Bass model by assuming separable[4] effects for advertising (i.e. advertising affects both external and internal influence) and find a positive advertising influence on the diffusion rate of high priced durable products. Mesak (1996) considers different options to include advertising: separable and non-separable effects (i.e. advertising impact on the parameter of external influence or the parameter of internal influence); he concludes that advertising affects the diffusion rate through both external and internal influence. Although the empirical findings of these authors are not conclusive, they agree on the positive sign of advertising influence.

Unfortunately, little is known about the effects of the marketing instruments on the diffusion process of frequently purchased products. We have found the empirical work by Parker and Gatignon (1994) on five brands of hair styling mousse, and applications by Lilien, Rao and Kalish (1981), Rao and Yamada (1988) and Hahn et al. (1994) on prescription drugs. In this section we first make some general remarks on these articles and then discuss the last three articles in greater detail.

Parker and Gatignon (1994) study frequently purchased products, but only consider first purchases (trials). They analyze both separable effects and non-separable effects for advertising, and find that advertising affects the trial rate with the expected signs in three out of five brands analyzed. However their results are not conclusive on how to include the effect of marketing in a diffusion model: a different model is selected for each of these three brands. For one brand the

[4] Separable effects and non-separable effects are discussed in Section 2.4.2.9.

preferred model is the one that accommodates the external influence of advertising, for another brand the model that allows for the internal influence is preferred, and for the third the model in which advertising affects both internal and external influence is selected. In the other three papers, the authors consider both first and repeat purchases and, hence, unlike Parker and Gatignon (1994), assume that diffusion rate is divided into a trial rate and a repeat rate. Lilien, Rao and Kalish (1981) find positive and significant, although small, effects of detailing expenditures and the squared company's detailing expenditures on sales. The opposite holds for the competitive detailing effects. Their results also demonstrate that the company's detailing expenditures increase the trial rate through the external influence whereas the competitors' detailing expenditures decrease the repeat rate. The findings of Lilien, Rao and Kalish are supported by Rao and Yamada (1988). Hahn et al. (1994) analyze twenty-one prescription drugs and consider the effects of detailing and medical journal advertising (considering them as an aggregated marketing instrument). They find positive and significant effects for the company's marketing efforts on the trial rate through the external influence.

We now discuss in greater detail the trial-repeat diffusion models developed by
a) Lilien, Rao and Kalish (1981), we refer to this model as the LRK model;
b) Mahajan, Wind and Sharma (1983), the MWS model, and
c) Hahn, Park, Krishnamurthi and Zoltners (1994), the HPKZ model.

a) The LRK model

Lilien, Rao and Kalish (1981) develop a model for the early forecasting of prescription drug sales. Specifically, they propose[5]:

$$\begin{aligned} s_{i,t} = &\left(\beta_{11i} x_{i,t-1} + \beta_{12i} x_{i,t-1}^2\right)\left[m - s_{i,t-1}\right] \\ &+\beta_{2i}\left(s_{i,t-1} - s_{i,t-2}\right)\left[m - s_{i,t-1}\right] + \left(1 - \beta_{3i} x_{c,t-1}\right) s_{i,t-1} \end{aligned} \tag{6.1}$$

where, for brand i ($i = 1, \ldots, N$) in month t ($t = 1, \ldots, T$):

$s_{i,t}$ = sales of brand i in time period t;
$x_{i,t}$ = detailing effort associated with brand i in time period t;
$x_{c,t}$ = detailing effort associated with the competing brands in time period t;
m = total market sales;
$\beta_{11i}, \beta_{12i}, \beta_{2i}, \beta_{3i}$ = parameters to be estimated.

[5] To facilitate comparison among the described models, we use the same terminology.

The LRK-model consists of three components:

(1) $\left(\beta_{11i}x_{i,t-1} + \beta_{12i}x_{i,t-1}^2\right)\left[m - s_{i,t-1}\right]$

(2) $\beta_{2i}\left(s_{i,t-1} - s_{i,t-2}\right)\left[m - s_{i,t-1}\right]$

(3) $\left(1 - \beta_{3i}x_{c,t-1}\right)s_{i,t-1}$.

Term (1) represents triers of brand *i* due to the company's promotional efforts, term (2) triers of brand *i* due to the internal influence and term (3) repeaters of brand *i* as an expression that accounts for the competitors' promotional efforts. Although the LRK-model is developed to describe and analyze the physician's prescription behavior, the estimation is done on the brand sales level. The LRK-model includes the effects of the company's and the competitors' promotional efforts and the internal influence. Although the authors have available data about medical journal advertising, direct mail and detailing expenditures, they only consider the effect of detailing expenditures given the high correlation between the promotional activities.

b) The MWS model

Mahajan, Wind and Sharma (1983) extend the first-purchase non-uniform influence model proposed by Easingwood, Mahajan and Muller (1983) by proposing a repeat-purchase diffusion model -the MWS-model- which specifically considers the non-uniform nature of the internal influence:

$$s_{i,t} = \beta_{1i}\left[m - s_{i,t-1}\right] + \beta_{21i}\left(\frac{s_{i,t-1}}{m}\right)^{\beta_{22i}}\left[m - s_{i,t-1}\right] + \beta_{3i}s_{i,t-1} \qquad (6.2)$$

where, for brand *i* ($i = 1, \ldots, N$) in month *t* ($t = 1, \ldots, T$):

$s_{i,t}$ = sales of brand *i* in time period *t*;

m = total market sales;

$\beta_{1i}, \beta_{21i}, \beta_{22i}, \beta_{3i}$ = parameters to be estimated.

In the MWS-model we distinguish three components:

(1) $\beta_{1i}\left[m - s_{i,t-1}\right]$

(2) $\beta_{21i}\left(\frac{s_{i,t-1}}{m}\right)^{\beta_{22i}}\left[m - s_{i,t-1}\right]$

(3) $\beta_{3i}s_{i,t-1}$.

Term (1) represents the sales effects of brand *i* due to triers that are affected by external influence (company's promotional efforts among others), term (2) is the sales effect of brand *i* that results from internal influence (such as word-of-mouth communication) and term (3) represents the sales effect due to repeat purchases of repeaters of brand *i*. Term (2), the internal influence, represents the time-varying

effect of imitation. The parameter β_{22i} is the non-uniform influence parameter ($\beta_{22i} \geq 0$). This term allows the internal influence effect to increase, decrease and remain constant over the penetration horizon. The incorporation of the non-uniform nature of the internal influence (see also Chapter 2, Section 2.4.2.3, and Chapter 5) makes the model more realistic.

c) The HPKZ model

Hahn et al. (1994) extend the diffusion framework of previous studies and develop a four-segment (triers, non-triers, repeaters and non-repeaters) trial and repeat model -the HPKZ-model- that can be calibrated using aggregate data for frequently purchased products in the early stages of the product life cycle. The HPKZ-model offers the opportunity to estimate the long run average market share of the new product. The HPKZ-model accommodates, although in a different way to the LRK-model, the effect of the internal influence and the company's promotional efforts. The authors specify two versions of the HPKZ-model. In the first version, HPKZ1, promotional spending enters the model as a relative variable. In the second version, HPKZ2, promotional spending is not divided by the total promotional spending:

HPKZ1

$$s_{i,t} = \left(\beta_{11i} + \beta_{12i} \ln\left(\frac{x_{i,t-1}}{x_{i,t-1} + x_{c,t-1}} \right) \right) \left[m - q_{i,t-1} \right] + \beta_{2i} \left(\frac{s_{i,t-1}}{m} \right) \left[m - q_{i,t-1} \right] + \beta_{3i} q_{i,t-1} \tag{6.3}$$

HPKZ2

$$s_{i,t} = \left(\beta_{11i} + \beta_{12i} \ln\left(x_{i,t-1} \right) \right) \left[m - q_{i,t-1} \right] + \beta_{2i} \left(\frac{s_{i,t-1}}{m} \right) \left[m - q_{i,t-1} \right] + \beta_{3i} q_{i,t-1} \tag{6.4}$$

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

where, for brand i ($i = 1, \ldots, N$) in month t ($t = 1, \ldots, T$):

$s_{i,t}$ = sales of brand i in time period t;
$x_{i,t}$ = detailing and advertising efforts associated with brand i in time period t;
$x_{c,t}$ = detailing and advertising efforts associated with the competing brands in time period t;
m = total market sales;
$q_{i,t-1}$ = potential sales of brand i to physicians in the post-trial segments (triers, repeaters and buyers of competing brands that have tried brand i before) at time t-1;
$\beta_{11i}, \beta_{12i}, \beta_{2i}, \beta_{3i}$ = parameters to be estimated.

As in the previous models, we identify three components in the HPKZ-model:

(1) $\left(\beta_{11i} + \beta_{12i} \ln\left(\frac{x_{i,t-1}}{x_{i,t-1} + x_{c,t-1}}\right)\right)\left[m - q_{i,t-1}\right]$ in version HPKZ1

$\left(\beta_{11i} + \beta_{12i} \ln\left(x_{i,t-1}\right)\right)\left[m - q_{i,t-1}\right]$ in version HPKZ2

(2) $\beta_{2i}\left(\frac{s_{i,t-1}}{m}\right)\left[m - q_{i,t-1}\right]$

(3) $\beta_{3i} q_{i,t-1}$.

Term (1) represents triers of brand *i* due to the external influence (which is influenced by the company's and competitors' promotional efforts in version HPKZ1 and only by the company's promotional efforts in version HPKZ2), term (2) represents triers of brand *i* due to the internal influence and term (3) repeaters of brand *i*. One interpretation of parameter β_{3i} is that "*it represents 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). Among the trial-repeat diffusion models, the HPKZ-model has the best fit and forecast ability, and has superior parameter face validity (Hahn et al., 1994). We use this model as the starting point of the development of our model.

6.4. Model specification

In this study, we modify the model of Hahn et al. (1994) to allow for heterogeneity in the effects of the different marketing variables and for differences in the effects of own and competitor's marketing efforts. Previous trial-repeat diffusion models (Lilien, Rao and Kalish, 1981; Hahn et al., 1994) employ a single or aggregated variable to model the effects of marketing instruments on the diffusion process. This aggregation implies that important information is lost about how different types of marketing variables have unique effects. In contrast to previous studies, we consider two promotional strategies: the traditional "push" strategy (medical journal advertising, detailing and physician meetings expenditures) and the "pull" strategy (direct-to-consumer advertising). We extend the HPKZ-model (version HPKZ2) to investigate longitudinal and cross-sectional effects of marketing expenditures on the diffusion of pharmaceuticals. Following the diffusion framework (see also Chapter 2, Section 2.3.1) presented by Hahn et al. (1994), the market is divided into four segments of physicians:

1) non-triers, physicians that are potential prescribers and have never tried the new product;
2) triers, physicians that prescribe the new product for the first time in time period *t*;
3) repeaters, physicians that prescribe the new product earlier and continue prescribing it in time period *t*; and
4) non-repeaters, physicians that have tried the new product in the past, but decided not to prescribe it in time period *t*.

Our model incorporates three characteristics of new product buying behavior: the innovative trial, the imitative trial, and the repeat buying. The innovative and imitative buying behaviors are modeled following Bass diffusion model (see Chapter 2). In a first stage of the diffusion process, the new product is discovered and adopted by a small group of innovative consumers (innovators) who influence others (imitators). This social interaction between the previous adopters and the potential adopters helps to explain the phase of rapid expansion in the diffusion process of innovations (Rogers, 1962, 1995). Hence, we face two kinds of behavior among those who adopt an innovation:

1) innovative behavior, which involves the adopters' basic tendency to innovate (or the adopters' innate innovativeness). This behavior is influenced by sources of external communication;
2) imitating behavior, which involves the tendency to adopt an innovation on the bases of interpersonal influence processes (word-of-mouth or internal communication).

The behavioral theory behind the traditional diffusion models suggests that a time-lag exists between the adoptions by members of a social system and that these adoptions are influenced by experience. Thus, the rate of trial purchases at time *t* is proportional to: i) the size of the market yet to be penetrated and ii) a linear function of previous penetration.

Since we are concerned with the longitudinal effects of both own and competitor's expenditures on the different marketing instruments and also with differentiating between marketing directed at physicians -"push" effect- and direct-to-consumer advertising -"pull" effect-, we propose the following model, which is an extended version of the HPKZ-model:

$$s_{i,t} = \left[\beta_{10i} + \sum_{j=1}^{4} \beta_{1ji} \ln\left(x_{ij,t}\right) + \sum_{j=1}^{4} \beta_{1jci} \ln\left(x_{cj,t}\right) + \beta_{2i} \left[\frac{s_{i,t-1}}{m_t} \right] \right] \left[m_t - q_{i,t-1} \right] + \beta_{3i} q_{i,t-1} \tag{6.5}$$

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

where, for brand i ($i = 1, \ldots, N$) in month t ($t = 1, \ldots, T$):

$s_{i,t}$ = sales of brand i in time period t (sales from trial and from repeat purchases);

$x_{ij,t}$ = own marketing expenditures on instrument j ($j = 1$: direct-to-consumer advertising; $j = 2$: detailing; $j = 3$: medical journal advertising; $j = 4$: physician meetings) in time period t;

$x_{cj,t}$ = competitors' marketing expenditures on instrument j in time period t;

m_t = total market sales in time period t;

$q_{i,t-1}$ = potential sales of brand i to physicians in the post-trial segments (triers, repeaters and buyers of competing brands that have tried brand i before) at time t-1;

$\beta_{10i}, \beta_{1ji}, \beta_{1jci}, \beta_{2i}, \beta_{3i}$ = parameters to be estimated.

In this model, $s_{i,t-1} / m_t$ is included to measure the internal influence effects: we assume that the prescribers of brand i at time t-1 are likely to influence non-prescribers to try this brand in time period t. The variable $q_{i,t-1}$ is included to measure the repeat rate. The parameters in the model can be interpreted as follows. The parameter β_{10i} indicates the basic propensity to try brand i without the influence of prior buyers or marketing expenditures. The effect of own (competitors') promotional activities on the trial rate of brand i is captured by β_{1ji} (β_{1jci}). The parameter β_{2i} captures the effect of internal influence on the trial rate, and β_{3i} is the repeat rate[6]. The proposed model has three components:

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

$$(2)\ \beta_{2i}\left[\frac{s_{i,t-1}}{m_t}\right]\left[m_t - q_{i,t-1}\right]$$

$$(3)\ \beta_{3i} q_{i,t-1}.$$

Term (1) represents triers of brand i due to external influence, which is determined by the basic propensity to buy at a given period and by own and cross marketing expenditures. Term (2) refers triers of brand i due to internal influence as a result of internal influence. Term (3) represents to the repeaters of brand i.

Equation (6.5) assumes that marketing expenditures affect the diffusion process through the trial rate and more specifically through the external influence (i.e. assuming non-separable effects for marketing expenditures). However, extant literature is inconclusive on how to include marketing instruments in a diffusion

[6] The model represented by Equation (6.5) is expressed in terms of sales, which are directly observable from aggregate data. However, when the model is expressed in terms of consumers (i.e. physicians), it is easy to see that $\hat{\beta}_{3i}$ is a repeat rate, the fraction of post-trial buyers who repurchase brand i in time period t. (see Hahn et al., 1994, pp. 227-228).

model (see also Section 6.2). For that reason, we do not assume *a priori* whether marketing expenditures affect external and/or internal influence. A formulation analogous to Equation (6.5) that accommodates internal influence is presented in Equation (6.6), and a model that allows for both internal and external influence is given in Equation (6.7.):

$$s_{i,t} = \left[\beta_{10i} + \left(\beta_{2i} + \sum_{j=1}^{4} \beta_{2ji} \ln\left(x_{ij,t}\right) + \sum_{j=1}^{4} \beta_{2jci} \ln\left(x_{cj,t}\right) \right) \left[\frac{s_{i,t-1}}{m_t} \right] \right] \left[m_t - q_{i,t-1} \right] + \beta_{3i} q_{i,t-1} \quad (6.6)$$

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

$$s_{i,t} = \left[\beta_{10i} + \beta_{2i} \left[\frac{s_{i,t-1}}{m_t} \right] \right] \left(1 + \sum_{j=1}^{4} \beta_{4ji} \ln\left(x_{ij,t}\right) + \sum_{j=1}^{4} \beta_{4jci} \ln\left(x_{cj,t}\right) \right) \left[m_t - q_{i,t-1} \right] + \beta_{3i} q_{i,t-1} \quad (6.7)$$

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

Our model -in all of its formulations: Equations (6.5), (6.6) or (6.7)- has two important limitations.

Firstly, the formulations assume that marketing affects the trial rate but not the repeat rate. Although theoretically both options are possible, we do not consider the influence of marketing on the repeat rate using the same argument as Hahn et al. (1994): "… *the direct experience coming from product trial should be more influential in the repeat market than indirect experience"* (see also Smith and Swinyard, 1982).

Secondly, none of the formulations incorporate price. There are some reasons that have led us to take this decision. Firstly, we have the arguments shown in Section 6.2 that can be synthesized in the following sentence "*Physicians are typically more concerned about product quality and side-effects than they are about price. Thus, price tends to be less important for the diffusion of a new prescription drug. However, it can have an impact on new product diffusion of more price sensitive pharmaceutical products or other product categories*" (Hahn et al., 1994, p. 246). Secondly, price is a very difficult concept to define in the pharmaceutical industry. Although manufacturers determine drugs' prices (list prices), different patients can pay different prices for the same prescription drug, depending on the health plans that they have with HMOs or insurance companies. These companies can negotiate discounts from pharmacies (retailers) or from manufacturers (rebates), and patients with health plans pay co-payments or only a part of the price. Hence, it is difficult to specify a "unique" price concept to incorporate into our model.

We estimate thirteen versions of our model. The versions differ with respect to the three formulations of the model (external influence formulation, see Equation (6.5), internal influence formulation, see Equation (6.6), and both external and internal influence formulation, see Equation (6.7)) and restrictions that we apply to these models. We now discuss the different versions.

- Model version 1: for this version we employ the following restrictions: $\beta_{11i} = \beta_{12i} = \beta_{13i} = \beta_{14i} = 0$ and $\beta_{11ci} = \beta_{12ci} = \beta_{13ci} = \beta_{14ci} = 0$ in the external influence formulation, $\beta_{21i} = \beta_{22i} = \beta_{23i} = \beta_{24i} = 0$ and $\beta_{21ci} = \beta_{22ci} = \beta_{23ci} = \beta_{24ci} = 0$ in the internal influence formulation and $\beta_{41i} = \beta_{42i} = \beta_{43i} = \beta_{44i} = 0$ and $\beta_{41ci} = \beta_{42ci} = \beta_{43ci} = \beta_{44ci} = 0$ in the external and internal influence formulation, the longitudinal effects of marketing expenditures are not accommodated in the model. With these restrictions, the three formulations reduce to one model. This version is close to the traditional diffusion models where no promotional efforts are considered.

- Model versions 2E, 2I and 2EI: for these versions we employ the following restrictions:
(1) $\beta_{11i} = \beta_{12i} = \beta_{13i} = \beta_{14i}$ in the external influence formulation, $\beta_{21i} = \beta_{22i} = \beta_{23i} = \beta_{24i}$ in the internal influence formulation and $\beta_{41i} = \beta_{42i} = \beta_{43i} = \beta_{44i}$ in the external and internal influence formulation, own marketing instruments have the same effect on the trial rate, and (2) $\beta_{11ci} = \beta_{12ci} = \beta_{13ci} = \beta_{14ci} = 0$ in the external influence formulation, $\beta_{21ci} = \beta_{22ci} = \beta_{23ci} = \beta_{24ci} = 0$ in the internal influence formulation and $\beta_{41ci} = \beta_{42ci} = \beta_{43ci} = \beta_{44ci} = 0$ in the external and internal influence formulation, the longitudinal effects of competitors' marketing expenditures are not accommodated in the model. The external influence formulation -model 2E- corresponds to HPKZ2, the second specification of the model proposed by Hahn et al. (1994).

- Model versions 3E, 3I and 3EI: for these versions we employ the following restrictions:
$\beta_{11i} = \beta_{12i} = \beta_{13i} = \beta_{14i}$ and $\beta_{11ci} = \beta_{12ci} = \beta_{13ci} = \beta_{14ci}$ in the external influence formulation, $\beta_{21i} = \beta_{22i} = \beta_{23i} = \beta_{24i}$ and $\beta_{21ci} = \beta_{22ci} = \beta_{23ci} = \beta_{24ci}$ in the internal influence formulation and $\beta_{41i} = \beta_{42i} = \beta_{43i} = \beta_{44i}$ and $\beta_{41ci} = \beta_{42ci} = \beta_{43ci} = \beta_{44ci}$ in the external and internal influence formulation, we assume that all the marketing instruments have the same effect on the trial rate. In contrast to the previous versions, these versions allow for both own-brand and competing-brands promotional effects.

- Model versions 4E, 4I and 4EI: for these versions we employ the following restrictions:
$\beta_{12i} = \beta_{13i} = \beta_{14i}$ and $\beta_{12ci} = \beta_{13ci} = \beta_{14ci}$ in the external influence formulation $\beta_{22i} = \beta_{23i} = \beta_{24i}$ and $\beta_{22ci} = \beta_{23ci} = \beta_{24ci}$ in the internal influence formulation and $\beta_{42i} = \beta_{43i}$

$= \beta_{44i}$ and $\beta_{42ci} = \beta_{43ci} = \beta_{44ci}$ in the external and internal influence formulation. With these assumptions, 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 the previous ones 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.

- Model versions 5E, 5I and 5EI: these are unrestricted versions of (6.5), (6.6) and (6.7).
These versions are the most flexible versions as they allow for heterogeneity in the effects of the different marketing variables.

In Table 6.2 we present an overview of the versions of the model we consider.

Table 6.2.
Versions of the proposed model in its external, internal and external and internal influence formulations.

---------- Model 1 ----------

$$s_{i,t} = \left[\beta_{10i} + \beta_{2i}\left[\frac{s_{i,t-1}}{m_t}\right]\right]\left[m_t - q_{i,t-1}\right] + \beta_{3i} q_{i,t-1}$$

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

---------- Model 2 ----------

External influence (Model 2E)

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

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

Internal influence (Model 2I)

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

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

External and internal influence (Model 2EI)

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

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

where $x_{i\bullet t} = \sum_{j=1}^{4} x_{ij,t}$

Table 6.2.
Versions of the proposed model in its external, internal and external and internal influence formulations (continued).

---------- Model 3 ----------

External influence (Model 3E)

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

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

Internal influence (Model 3I)

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

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

External and internal influence (Model 3EI)

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

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

where $x_{i\bullet t} = \sum_{j=1}^{4} x_{ij,t}$ and $x_{c\bullet t} = \sum_{j=1}^{4} x_{cj,t}$

---------- Model 4 ----------

External influence (Model 4E)

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

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

Internal influence (Model 4I)

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

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

External and internal influence (Model 4EI)

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

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

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

Table 6.2.
Versions of the proposed model in its external, internal and external and internal influence formulations (continued).

---------- Model 5 ----------

External influence (Model 5E)

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

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

Internal influence (Model 5I)

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

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

External and internal influence (Model 5EI)

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

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

In Table 6.3 we present an overview of the marketing instruments that are incorporated in the LRK, the MWS, in the HPKZ models, and in our model.

Table 6.3.
Marketing communication characteristics of the trial-repeat diffusion models.

	Lilien, Rao and Kalish model (1981)	Mahajan, Wind and Sharma model (1983)	Hahn, Park, Krishnamurthi and Zoltners model (1994)	Proposed model
Marketing Communication:				
-"Push" strategy	Detailing	Not included	Detailing Medical journal advertising	Detailing Medical journal advertising Physician meetings
-"Pull" strategy	Not included	Not included	Not included	Direct-to-consumer advertising
-Heterogeneity in the effects of the marketing activities	No	No	No	Yes
-Competitor's marketing activities (separated from own)	Yes	No	No	Yes
-Influence on the trial rate through…				
…the external influence	Considered	Not considered	Considered	Considered
…the internal influence	Not considered	Not considered	Not considered	Considered
…both the external and internal influences(1)				Considered

(1): This indicates that separate models are proposed to test each alternative on the trial rate.

6.5. Sample, data and measurement of the variables

We use monthly US data on a category of prescription drugs, "rhinitis" category, to estimate the versions of the proposed model. Rhinitis is a reaction that occurs in the eyes, nose and throat when airborne irritants -allergens- trigger the release of histamine; histamine causes inflammation and fluid production in the fragile linings of nasal passages, sinuses, and eyelids. All the drugs in the category are functionally equivalent, i.e. they all treat the same medical symptoms. This category is highly competitive containing only branded products with small to moderate differences in price positioning[7]. The category is also characterized by a

[7] The average category price (in constant dollars) is 34.26$, the lowest average price is 25.67 (*Zyrec Syrup*) and highest price is 47.28$ (*Claritin*).

large number of introductions: it contains 16 products, 14 of which are introduced in the observational period (1993-2000). Thus, it is an appropriate category to validate the proposed model since we observe almost the complete category (87.5% of the drugs) from its inception (see Table 6.4). These introductions are accompanied by extensive marketing expenditures. The introductions differ in terms of instruments used and in terms of "money spent" per instrument. This allows us to study the effect of pharmaceutical marketing on diffusion of new drugs without the possible disturbance of differences in markets, differences in "seriousness" of the treated disease, etc.

Table 6.4.
Rhinitis category.

Brand ID	Name	Month of introduction
1	*Allegra*	August 1996
2	*Allegra-D*	January 1998
3	*Astelin*	January 1997
4	*Atrovent Nasal Spra*	December 1995
5	*Beconase*	before January 1993
6	*Claritin*	April 1993
7	*Claritin D*	November 1994
8	*Claritin Syrup*	October 1996
9	*Flonase*	December 1994
10	*Nasacort*	January 1993
11	*Nasarel*	October 1995
12	*Nasonex*	October 1997
13	*Rhinocort*	June 1994
14	*Vancenase*	before January 1993
15	*Zyrtec*	January 1996
16	*Zyrtec Syrup*	November 1996

The data set contains the following information for each drug: sales, price[8] and expenditures for detailing, medical journal advertising, physician meetings and direct-to-consumer advertising. Expenditures on advertising directed at physicians are higher than direct-to-consumer advertising, and detailing is the primary promotional activity directed at physicians. However, for the majority of the brands, expenditures on direct-to-consumer advertising are higher than expenditures on medical journal advertising and physician meetings, respectively.

[8] Given that patient may need different dosages of a drug, price refers to the complete treatment course instead of per dose.

Figure 6.1 presents real monthly sales data of the new brands. Marketing variables are defined in terms of constant dollars and sales are seasonally adjusted[9] prior to estimating each of the parameters. 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) allows for removing jumps in the sales time series that are not the result of marketing actions or inter-personal communication. As expenditures for promotional activities in a period influence the drug's sales in the following period, we have lagged the promotional activities by one month. The same number of lags is taken by Lilien, Rao and Kalish (1981) and Hahn et al. (1994). Although authors such as Gönül et al. (2001), Van den Bulte and Lilien (2001) and Berndt, Pindyck and Azoulay (2003) use marketing stock variables with a different number of lags to account for carryover effects in the promotional activities, the specification of our model already incorporates the lagged effects of marketing instruments. We also estimated versions of the model using stock variables with a different number of lags, but the option of one lag offered the best results. Natural logarithms are applied to marketing variables[10] to incorporate diminishing returns to scale for marketing actions in the model (Hahn et al., 1994).

[9] We apply a seasonal adjustment following Lilien, Rao and Kalish (1981) and Rao and Yamada (1988) instead of using the approach proposed by Hahn et al. (1994) of dividing sales by the growth of the usage rate over time. We proceed in this way for the following two reasons: i) the approach proposed by Hahn et al. (1994) does not deseasonalize our data, and ii) this approach takes out the market growth, so that category expansion effects would be removed from the data.

[10] We consider the marketing variables in a "mean deviation form".

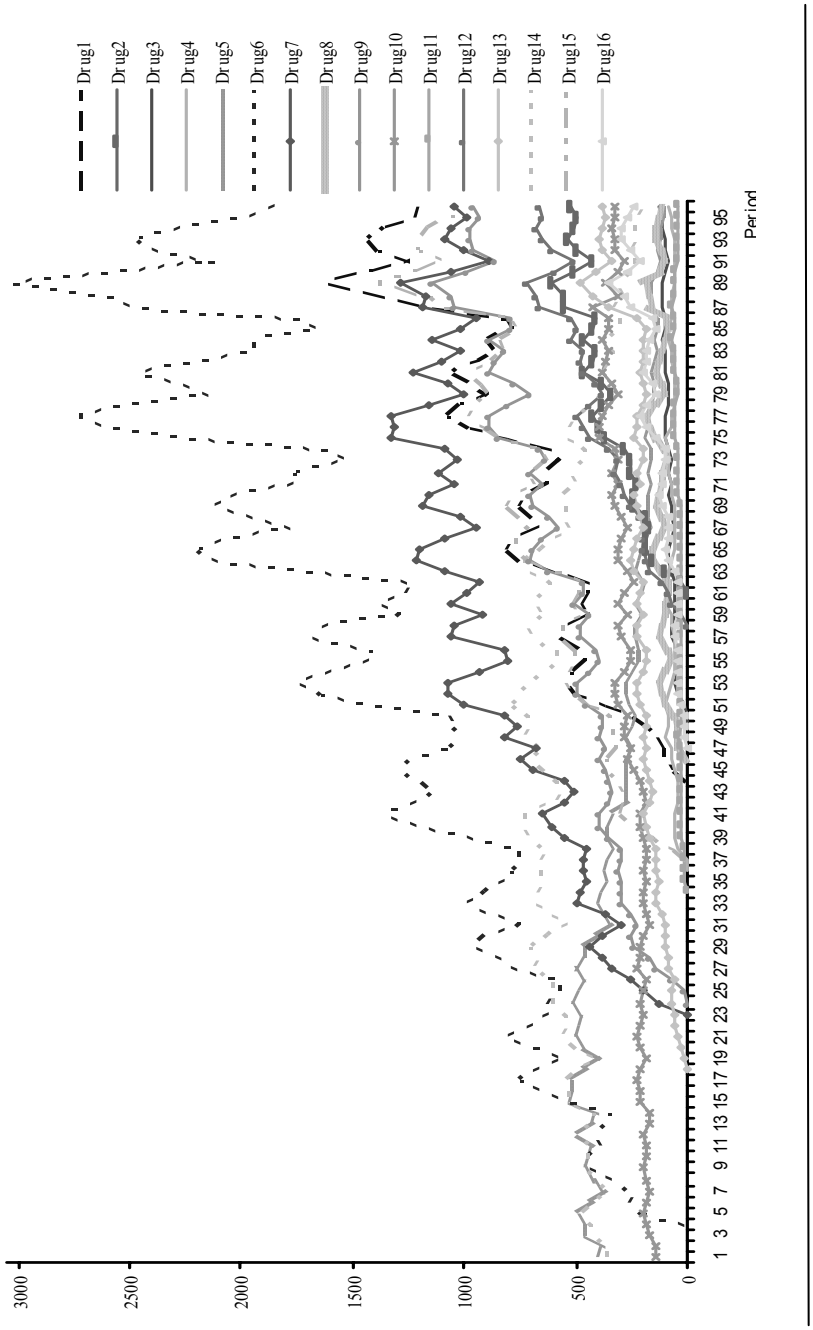


Figure 6.1.
Real monthly data sales for the new drugs.

In Sections 6.2 and 6.4 we discussed some reasons for not including price in our model. Looking at the data of the rhinitis category we find three more arguments. Firstly, there are no generic drugs within this category (see Section 6.2). Secondly, we observe small differences in price positioning among the drugs. This seems to indicate that if there are no big differences in the price of competing drugs, factors other than price, such as advertising, are used by firms to compete. Price does not seem to be a key factor in this category to influence the switching from one branded drug to another. Thirdly, multicollinearity problems appeared when we included both price and the other marketing instruments in the model.

Although we focus on the rhinitis category, an especially interesting category given the large number of introductions (14 out of 16 drugs) in the period 1993-2000, we also calibrate the model on another two categories: osteoarthritis-rheumatoid-arthritis and asthma. These two categories together with rhinitis belong to the "Top-10 market" of prescription drugs in the US in 2000; i.e. the 10 major categories of prescription drugs that have the prescription drugs with the largest amount of sales among those with annual sales of 25$ million or more in the US in 2000. Although osteoarthritis-rheumatoid-arthritis and asthma categories have a lower proportion of new drugs than rhinitis (9 out of 20 drugs and 11 out of 26 drugs, respectively), they are also appropriate for calibration of our model for three reasons. First, these two categories use the same promotional instruments as rhinitis: detailing, medical journal advertising, physician meetings and also direct-to-consumer advertising. Second, it is interesting to analyze the results for other categories with similar and different characteristics to rhinitis. This is the case for osteoarthritis-rheumatoid-arthritis and asthma. The asthma category resembles the rhinitis category in terms of symptoms and seasonal patterns, whereas the osteoarthritis-rheumatoid-arthritis is completely different. Third, in contrast to the rhinitis category, the osteoarthritis-rheumatoid-arthritis category and the asthma category contain both branded drugs and generic drugs. Table 6.5 shows code (identification number of the drug), name, kind of drug (branded or generic drug) and introduction month for each drug within each category.

Table 6.5.
Osteoarthritis-rheumatoid-arthritis and asthma categories.

osteoarthritis-rheumatoid-arthritis				asthma			
Code	Name	kind	Month of introduction	Code	Name	kind	Month of introduction
1	*Arthrotec*	branded	January 1998	1	*Accolate*	branded	October 1996
2	*Celebrex*	branded	January 1999	2	*Aerobid*	branded	before January 1993
3	*Daypro*	branded	January 1993	3	*Albuterol Aerosol*	generic	January 1996
4	*Diclofenac Sodium*	generic	August 1995	4	*Albuterol Neb Soln*	generic	before January 1993
5	*Etodolac*	generic	March 1997	5	*Albuterol Oral*	generic	before January 1993
6	*Ibuprofen*	generic	before January 1993	6	*Atrovent Inh/Neb Sol*	branded	before January 1993
7	*Indomethacin*	generic	before January 1993	7	*Azmacort*	branded	before January 1993
8	*Ketoprofen*	generic	before January 1993	8	*Beclovent*	branded	before January 1993
9	*Lodine*	branded	before January 1993	9	*Combivent*	branded	June 1997
10	*Methylprednis Tabs*	generic	before January 1993	10	*Cromolyn Sod*	generic	May 1994
11	*Naprelan*	branded	April 1996	11	*Flovent*	branded	July 1996
12	*Naproxen*	generic	September 1993	12	*Intal*	branded	before January 1993
13	*Oruvail*	branded	October 1993	13	*Ipratropium Bromide*	generic	June 1996
14	*Piroxicam*	generic	before January 1993	14	*Maxair*	branded	before January 1993
15	*Prednisolone*	generic	before January 1993	15	*Proventil Aerosol*	branded	before January 1993
16	*Prednisone*	generic	before January 1993	16	*Proventil Oral*	branded	before January 1993
17	*Relafen*	branded	before January 1993	17	*Pulmicort Turbuhale*	branded	October 1997
18	*Sulindac*	generic	before January 1993	18	*Serevent*	branded	March 1994
19	*Vioxx*	branded	May 1999	19	*Serevent Diskus*	branded	December 1997
20	*Voltaren*	branded	before January 1993	20	*Singulair*	branded	March 1998
				21	*Theo-Dur*	branded	before January 1993
				22	*Theophylline SR*	generic	before January 1993
				23	*Uniphyl*	branded	before January 1993
				24	*Vanceril*	branded	before January 1993
				25	*Ventolin*	branded	before January 1993
				26	*Volmax*	branded	October 1993

6.6. Empirical results

6.6.1. Longitudinal effects of marketing instruments

We estimated the thirteen versions of the proposed model -Equations (6.5), (6.7) and (6.8)- (see Table 6.2) using the iterative OLS procedure[11] of Hahn et al. (1994). The estimation of models 2EI, 3EI, 4EI and 5EI -external and internal influence formulation- suffers from multicollinearity of the marketing instruments. Hence, we discard the external and internal influence formulation and compare the external with the internal formulations of model 2, 3, 4 and 5 using the Akaike Information Criterion and taking into account the face validity of the estimates. After selecting the best formulation -external or internal influence-, the likelihood ratio tests allow us to identify the most parsimonious specification (Ramanathan, 1993) between the nested versions of the models. The restricted model is defined as the null hypothesis and the unrestricted model as the alternative hypothesis. Table 6.6 summarizes the hypotheses for the likelihood ratio tests.

Table 6.6.
Hypotheses for the likelihood ratio tests (external influence formulation)(1).

Null hypothesis	Alternative hypothesis			
	Model 2	Model 3	Model 4	Model 5
Model 1	$\beta_{11i} = 0$	$\beta_{11i} = 0$ $\beta_{11ci} = 0$	$\beta_{11i} = \beta_{12i} = 0$ $\beta_{11ci} = \beta_{12ci} = 0$	$\beta_{11i} = \beta_{12i} = \beta_{13i} = \beta_{14i} = 0$ $\beta_{11ci} = \beta_{12ci} = \beta_{13ci} = \beta_{14ci} = 0$
Model 2		$\beta_{11ci} = 0$	$\beta_{12i} = 0$ $\beta_{11ci} = \beta_{12ci} = 0$	$\beta_{12i} = \beta_{13i} = \beta_{14i} = 0$ $\beta_{11ci} = \beta_{12ci} = \beta_{13ci} = \beta_{14ci} = 0$
Model 3			$\beta_{12i} = 0$ $\beta_{12ci} = 0$	$\beta_{12i} = \beta_{13i} = \beta_{14i} = 0$ $\beta_{12ci} = \beta_{13ci} = \beta_{14ci} = 0$
Model 4				$\beta_{13i} = \beta_{14i} = 0$ $\beta_{13ci} = \beta_{14ci} = 0$

(1): Identical hypotheses are used for the internal influence formulation.

The estimation of models 4E, 4I, 5E and 5I suffers from multicollinearity of the marketing instruments; the models as a whole are highly significant whereas the number of significant estimates is small. Other researchers find the same. For

[11] Since $q_{i,t-1}$ is not directly observeable from the data and as it is therefore not posible to estimate the parameters, we follow the procedure proposed by Hahn et al. (1994) to calculate it.

example Lilien, Rao and Kalish (1981), Gatignon, Weitz and Bansal (1990), Hahn et al. (1994), and Rizzo (1999) also report that marketing activities are highly correlated in pharmaceutical markets. We discard models 4 and 5, and continue investigating models 1, 2 and 3. The Akaike Information Criterion (Table 6.7) reveals that, although differences are very small, the external influence formulation is the most appropriate for the majority of the drugs for both models 2 and 3. Model 2E is preferred to model 2I in 11 out of 14 cases (79%) and Model 3E is preferred to model 3I in 10 out of 14 cases (71%). Face validity is quite similar for the external and internal influence formulations.

Table 6.7.
Akaike Information Criterion.

Brand code	model 2			model 3		
	model 2E	model 2I	preferred model	model 3E	model 3I	preferred model
10	9.70	9.71	model 2E	9.72	9.64	model 3I
6	11.63	11.62	model 2I	11.25	11.26	model 3E
13	8.38	8.46	model 2E	7.90	8.31	model 3E
7	12.27	12.28	model 2E	11.43	11.63	model 3E
9	9.73	9.75	model 2E	9.19	9.32	model 3E
11	3.97	4.04	model 2E	3.85	3.97	model 3E
4	5.55	5.55	model 2E	5.57	5.55	model 3I
15	9.67	9.68	model 2E	9.68	9.69	model 3E
1	10.65	10.66	model 2E	10.52	10.57	model 3E
8	5.95	5.96	model 2E	5.87	5.89	model 3E
16	7.02	7.01	model 2I	7.06	7.04	model 3I
3	7.83	7.84	model 2E	7.62	7.70	model 3E
12	8.59	8.67	model 2E	8.62	8.72	model 3E
2	10.80	10.79	model 2I	10.73	10.70	model 3I

The estimation results of models 1, 2E and 3E are shown in Tables 6.8, 6.9 and 6.10, respectively. These tables are divided into five column-blocks. Column-block 1 shows the identification numbers of the brands (the results are presented in order of introduction). Brands 5 and 14 were already on the market at the start of the observational period, so we cannot study their entire diffusion process. For that reason, those brands are not present in the tables. Column-block 2 contains the trial rate parameters, differentiating between external (β_{10i} in model 1, β_{10i} and β_{11i} in model 2E, and β_{10i}, β_{11i} and β_{11ci} in model 3E) and internal influence (β_{2i}). Column-block 3 shows the repeat rate parameter (β_{3i}). Column-block 4 presents the average market share of each brand. This column can be used to judge the face validity of the repeat rate, as the market share should approach the repeat rate. Column-block 5 presents the goodness-of-fit statistics (mean absolute deviation -MAD-, mean absolute percentage error -MAPE- and the correlation between the real and the

estimated values of the dependent variables[12] -r-). All of the values in Tables 6.8, 6.9 and 6.10 are significantly different from zero ($\alpha = 0.0001$, $\alpha = 0.001$, $\alpha = 0.05$ or $\alpha = 0.10$). The results in Table 6.8, 6.9 and 6.10 are obtained using the marketing variables in a "mean deviation form": this creates values for β_{10i} that are comparable across brands and that can be interpreted as the basic propensity to try the new product (or innate innovativeness) when the marketing variables are at their mean level and when there is no internal influence. We performed several residual diagnostic checks, and concluded that neither the homoskedasticity, nor the non-normality assumption is violated. For some brands, the residuals show positive autocorrelation. We re-estimated the models for these brands using GLS, and observed only minor changes in the estimation results.

Table 6.8.
Estimation results of model 1.

Brand code	Trial rate		Repeat rate	Average market share (in units)	MAD	MAPE	r
	External influence $\hat{\beta}_{10i}$	Internal influence $\hat{\beta}_{2i}$	$\hat{\beta}_{3i}$				
10	-0.05***	1.31***	0.09***	0.04	23.38	9.03	0.92
6			0.32***	0.20	63.83	4.76	0.99
13	-0.03***	2.06***	0.03***	0.03	12.37	6.29	0.97
7		0.78*	0.16***	0.13	72.40	8.53	0.97
9	0.01°	0.64***	0.10***	0.09	18.88	3.37	0.99
11		1.02***	0.003**	0.01	1.10	2.91	0.99
4	0.01***	0.84***	0.001*	0.01	1.66	2.53	0.95
15	0.04***	0.19°	0.13***	0.10	13.80	2.07	0.99
1	0.03**		0.19***	0.11	21.80	2.96	0.99
8	0.01***	0.65***	0.01°	0.02	2.07	1.82	0.97
16	0.002*	0.60***	0.05***	0.02	2.09	1.61	0.99
3	0.004**	0.56***	0.01*	0.01	2.97	3.81	0.90
12	0.01***	0.53***	0.09***	0.06	4.62	1.18	0.99
2	0.01*	0.66**	0.06***	0.05	8.71	2.49	0.95

***: p≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05 ; °: p<0.1

[12] We show r instead of R^2 or adjusted-R^2 since the proposed models do not have an intercept term (Judge et al., 1985, pp. 30-31).

The estimates in Table 6.8 correspond to outcomes of an earlier meta study conducted by Sultan, Farley and Lehmann (1990). They found that values for β_{10i} are positive and (much) smaller than the internal influence coefficient ($\hat{\beta}_{2i}$). This is the case for all but two of the significant estimates for β_{10i}. The significant estimates of β_{2i} are also within the expected range (positive and larger than β_{10i}). Also, the estimates of β_{3i} are reasonable, as they typically closely resemble the average market share of each brand. All of the differences between $\hat{\beta}_{3i}$ and the average market share are equal to or smaller than 0.08, except for brand 6, where the difference equals 0.12. The values for r, SSR and MAPE suggest that model 1 describes the diffusion process of the drugs in the rhinitis category quite well.

Table 6.9.
Estimation results of model 2E.

Brand code	Trial rate			Repeat rate	Average market share (in units)	MAD	MAPE	r
	External influence		Internal influence					
	$\hat{\beta}_{10i}$	$\hat{\beta}_{11i}$	$\hat{\beta}_{2i}$	$\hat{\beta}_{3i}$				
10	0.04***		1.32***	0.09***	0.04	22.80	8.80	0.92
6		-0.03*		0.31***	0.20	61.30	4.57	0.99
13	0.04***	0.01***	1.65***	0.03*	0.03	8.84	4.49	0.99
7	0.07*		0.88*	0.16**	0.13	72.76	8.57	0.98
9	0.07***		0.59***	0.10***	0.09	18.36	3.27	0.99
11	0.01***	0.0003***	0.91***	0.004**	0.01	0.85	2.24	0.99
4	0.01***		0.83***	0.001***	0.01	1.66	2.53	0.95
15	0.05***		0.19°	0.13***	0.10	13.99	2.10	0.99
1	0.04***			0.18***	0.11	21.80	2.96	0.99
8	0.02***	-0.001*	0.57***	0.01***	0.02	1.87	1.65	0.98
16	0.01***		0.59***	0.05***	0.02	2.09	1.62	0.99
3	0.01***		0.54***		0.01	3.05	3.91	0.90
12	0.03***	0.002*	0.42***	0.09***	0.06	4.38	1.11	0.99
2	0.04***		0.65**	0.06*	0.05	8.50	2.43	0.95

***: p≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05; °: p<0.1

The significant estimates of β_{10i} in Table 6.9 are also within the expected range. Three out of the five significant estimates for the effect of own marketing expenditures on the trial rate show a positive although small value; for two brands the estimated parameter is significant although negative; for nine brands, the estimated parameter turned out not to be significant. The significant estimates of β_{2i} are also within the expected range. The significant estimates of β_{3i} are

reasonable. All of the differences between $\hat{\beta}_{3i}$ and the average market share are equal or smaller than 0.07, except for brand 6, which is 0.11. The values of r, SSR and MAPE indicate that model 2E also describes the adoption of the drugs in the rhinitis category well.

Table 6.10.
Estimation results of model 3E.

Brand code	Trial rate				Repeat rate	Average market share (in units)	MAD	MAPE	r
	External influence			Internal influence					
	$\hat{\beta}_{10i}$	$\hat{\beta}_{11i}$	$\hat{\beta}_{11ci}$	$\hat{\beta}_{2i}$	$\hat{\beta}_{3i}$				
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}: $p<0.1$

The results for model 3E provoke the following comments. The estimates of β_{10i} in Table 6.10 are all significant and within the expected range. All of the significant estimates for the effect of own marketing expenditures on the trial rate have the positive sign, except for brand 8, which is negative; for five brands, the estimated parameter turned out not to be significant. Competitors' marketing expenditures generally have a negative effect on the trial rate; for five brands, the estimated parameter turned out not to be significant. The significant estimates of β_{2i} are also within the expected range. Also, the values of $\hat{\beta}_{3i}$ are reasonable. All of the differences between $\hat{\beta}_{3i}$ and the average market share are equal or smaller than 0.08, except for brand 6, which is 0.12.

Results of the likelihood ratio tests are shown in Table 6.11. The second and third column of this table show the results of the likelihood ratio test for comparing

model 1 -restricted model- and model 2E -unrestricted model-. For brand 10, the restriction, $\hat{\beta}_{11i} = 0$, does not lead to a significant decrease of fit (χ^2=1.48). Hence, model 1 is retained. For brand 6, χ^2=6.06 is significant and this indicates that model 2E is a significant improvement over model 1. Therefore, model 2E is retained.

Table 6.11.
Likelihood ratio tests.

Brand code	Restricted model: mod.1 Unrestricted model: mod.2E		Restricted model: mod.1 Unrestricted model: mod.3E		Restricted model: mod.2E Unrestricted model: mod.3E	
	χ^2	Retained model	χ^2	Retained model	χ^2	Retained model
10	1.48	model 1	1.66	model 1	0.18	model 2E
6	6.06*	model 2E	42.90*	model 3E	36.84*	model 3E
13	50.41*	model 2E	90.32*	model 3E	39.91*	model 3E
7	1.07	model 1	64.91*	model 3E	63.85*	model 3E
9	1.67	model 1	43.65*	model 3E	41.98*	model 3E
11	24.09*	model 2E	33.69*	model 3E	9.60*	model 3E
4	0.15	model 2E	0.68	model 1	0.54	model 2E
15	1.19	model 1	2.64	model 1	1.45	model 2E
1	0.50	model 1	9.18*	model 3E	8.68*	model 3E
8	7.36*	model 2E	13.07*	model 3E	5.71*	model 3E
16	0.02	model 1	0.24	model 1	0.22	model 2E
3	0.42	model 1	12.56*	model 3E	12.14*	model 3E
12	6.62*	model 2E	7.41*	model 3E	0.79	model 2E
2	0.05	model 1	4.74^{o}	model 3E	4.68*	model 3E

*: Significant at 0.05 level; ^{o}: Significant at 0.1 level.

Although there is not a model that has been retained for all the brands, in general terms model 3E is revealed as the preferred model (see Table 6.12) given that model 3E is a significant improvement over model 1 and model 2E in 71% and 64% of the cases, respectively.

Table 6.12.
Retained model -general terms-(1)

	Relative frequency
model 1 vs. model 2E	
model 1 is retained	8/14
model 2E is retained	6/14
model 1 vs. model 3E	
model 1 is retained	4/14
model 3E is retained	10/14
model 2E vs. model 3E	
model 2E is retained	5/14
model 3E is retained	9/14

(1): see Section 6.6.1 for an explanation about why external and internal formulations (EI models) are discarded.

We repeat the previous analyses for osteoarthritis-rheumatoid-arthritis and asthma categories. Again multicollinearity of the marketing instruments leads us to discard models 4 and 5, and continue investigating models 1, 2 and 3. The detailed results for these two categories are in Appendix 6A. Tables 6.13 and 6.14 show the summarized results for the three categories analyzed. Table 6A.1 (Appendix 6A) shows that, for the osteoarthritis-rheumatoid-arthritis category, internal influence formulation is preferred for model 2 in 6 out of 9 of the cases (67%) and for model 3 in 5 out of 9 of the cases (56%). Table 6A.2 (Appendix 6A) shows that, for the asthma category, external influence formulation is preferred for model 2 in 5 out of 10 of the cases (50%) and for model 3 in 8 out of 10 of the cases (56%)[13]. Face validity reveals that the external influence formulation shows more significant estimates with right signs and expected values. Face validity of the estimates is very relevant given that for the cross-section analysis (that we conduct in the following section) we use the estimates of the selected diffusion model in the longitudinal analysis carried out in this section. Table 6.13 summaries results for the three categories analyzed.

[13] Although within the asthma category there are 11 new drugs, the estimation method does not converge for drug 19. Hence, we retain the estimation results for 10 new drugs.

Table 6.13.
External vs. internal influence formulation.

	Akaike Information Criterion shows…… as the preferred formulation	Face validity shows…… as the preferred formulation
Rhinitis		
model 2	*External* influence formulation (in 11 out 14 of the cases)	*External* influence formulation and Internal influence formulation
model 3	*External* influence formulation (in 10 out 14 of the cases)	*External* influence formulation and Internal influence formulation
Osteoarthritis-rheumatoid-arthritis		
model 2	Internal influence formulation (in 6 out 9 of the cases)	*External* influence formulation
model 3	Internal influence formulation (in 5 out 9 of the cases)	*External* influence formulation
Asthma		
model 2	*External* influence formulation (in 5 out 10 of the cases)	*External* influence formulation
model 3	*External* influence formulation (in 8 out 10 of the cases)	*External* influence formulation

Table 6.13 shows that the external influence formulation is, in general terms, more appropriate than the internal influence formulation to describe the diffusion processes of the new drugs in the three categories analyzed. Appendix 6A shows the estimation results of models 1, 2E and 3E for osteoarthritis-rheumatoid-arthritis (Tables 6A.3, 6A.4 and 6A.5, respectively) and asthma (Tables 6A.6, 6A.7 and 6A.8, respectively). Appendix 6A also shows the likelihood ratio tests for osteoarthritis-rheumatoid-arthritis and asthma (Tables 6A.9 and 6A.10, respectively). Table 6.14 summaries the results of the likelihood ratio tests for the three categories analyzed. This table reveals that, in general terms, model 3E is the preferred model given that model 3E is a significant improvement over model 1 and model 2E in the three analyzed categories.

Table 6.14.
Retained model in each drug category -general terms-

	Rhinitis	Osteoarthritis-rheumatoid-arthritis	Asthma
	Relative frequency	Relative frequency	Relative frequency
model 1 vs model 2E			
model 1 is retained	8/14	5/9	7/10
model 2E is retained	6/14	4/9	3/10
model 1 vs model 3E			
model 1 is retained	4/14	0/9	4/10
model 3E is retained	10/14	9/9	6/10
model 2E vs model 3E			
model 2E is retained	5/14	0/9	4/10
model 3E is retained	9/14	9/9	6/10

From the results in this section we conclude that longitudinal effects of marketing instruments matter. The estimates of β_{1ji} and β_{1jci} show that the external influence on the trial rate varies over time: it responds to changes in both own and competitors' marketing expenditures. Own marketing expenditures increase the trial rate and competitors' marketing expenditures decrease the trial rate. We find that marketing efforts by the drug manufacturers are key drivers of the diffusion processes of the new drugs introduced into the market. In the next section we investigate whether there exist systematic differences in the parameter estimates between the brands. That is, we perform a second-stage cross-sectional analysis on the parameter estimates that we obtained in this section.

6.6.2. Cross-sectional effects of marketing instruments

It is of interest to investigate whether the basic propensity to try the new product - $\hat{\beta}_{10i}$ -, the physicians' internal influence parameter - $\hat{\beta}_{2i}$ - and the repeat rate[14] - $\hat{\beta}_{3i}$ - are influenced by the average own "marketing-pressure" during the introduction. In Section 6.2 we distinguished two functions of marketing communication that play a relevant role in the pharmaceutical industry: the "informative" and "persuasive" functions of pharma marketing. The "informative" function reveals that pharmaceutical marketing may influence the diffusion process

[14] Hahn at al. (1994, p. 235) refer to $\hat{\beta}_{3i}$ as "*the effectiveness of consumers' product trial or direct product experience on repeat purchase*".

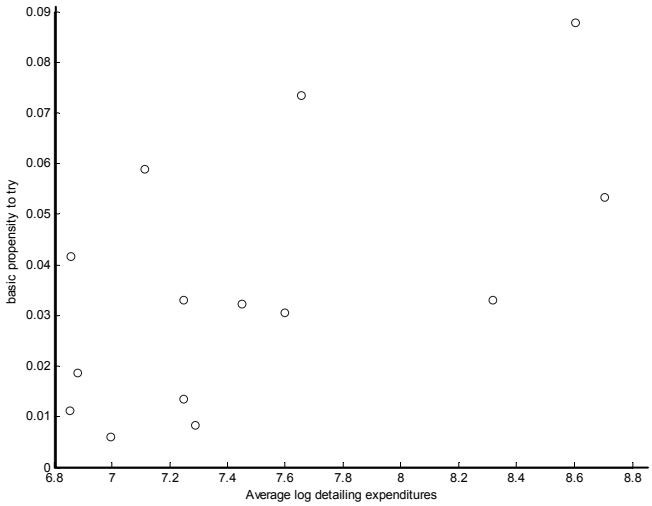
of a new product through the trial rate and the "persuasive" function through the repeat rate. We first investigate the trial rate ($\hat{\beta}_{10i}$ and $\hat{\beta}_{2i}$) during the first months after introduction and then the repeat rate ($\hat{\beta}_{3i}$) in two periods: 1) during the first months after introduction, and 2) during the complete period, given that the "persuasive" function refers to the influence that marketing activities have in creating market power for the promoted product.

We first explore the aforementioned relationships using scatter plots and correlation analysis. Subsequently we employ regression analysis. Analogous to the longitudinal analysis in Section 6.6.1 we present a detailed analysis for the rhinitis category and then provide a summary of the analyses of the osteoarthritis-rheumatoid-arthritis category and the asthma category.

Effects on the propensity to try and internal influence (trial rate)

Figure 6.2 shows a scatter plot of the estimated values of $\hat{\beta}_{10i}$ and the mean level of the own detailing expenditures during the first 12 months after introduction[15].

Figure 6.2.
Scatter plot of the basic propensity to try ($\hat{\beta}_{10i}$) and the mean of the log of detailing level.



[15] In Figure 6.2 and in the rest of this section, the estimation results of model 3E are used.

The correlation coefficient of the scatter plot in Figure 6.2 is significant with a confidence level of 95% (ρ = 0.60; p-value = 0.02), which indicates that the basic propensity to try the new product is positively affected by a high level of detailing during the first 12 months after introduction. The other marketing instruments show a similarly positive although not significant correlation with $\hat{\beta}_{10i}$, except physician meetings, which show a positive and significant correlation with $\hat{\beta}_{10i}$ (see Table 6.15).

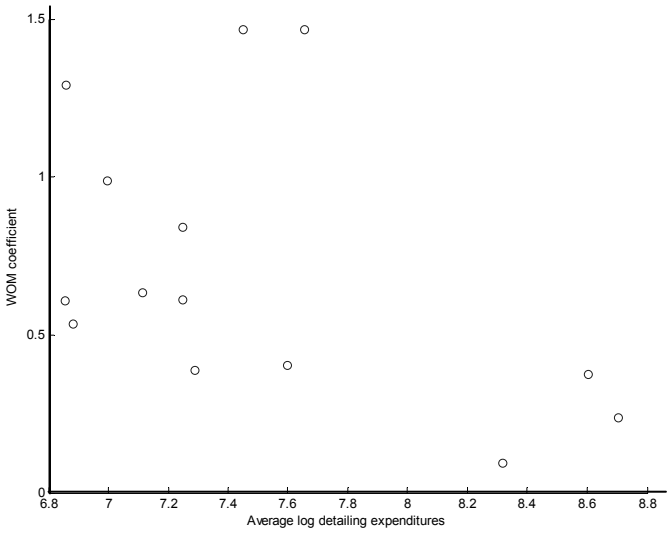
Table 6.15.
Correlation between marketing instruments and the basic propensity to try ($\hat{\beta}_{10i}$)

	ρ	p-value
detailing	0.60*	0.02
medical journal advertising	0.30	0.29
physician meetings	0.59*	0.03
direct-to-consumer advertising	0.10	0.73

*: p≤ 0.05

Figure 6.3 shows a scatter plot of the estimated values of $\hat{\beta}_{2i}$ and the mean level of the own detailing expenditures during the first 12 months after introduction.

Figure 6.3.
Scatter plot of the WOM coefficient ($\hat{\beta}_{2i}$) and the mean of the log of detailing level.



The correlation coefficient of the scatter plot of $\hat{\beta}_{2i}$ and the mean detailing level (see Figure 6.3) is not significant (ρ = -0.45; p-value = 0.10). We also do not find a significant correlation between physician meeting expenditures and $\hat{\beta}_{2i}$. This indicates that internal influence is not significantly affected by detailing or physician meeting expenditures during the first 12 months after introduction. The other two marketing instruments show a negative and significant correlation with $\hat{\beta}_{2i}$ (see Table 6.16). A possible explanation for the fact that detailing and physician meeting expenditures are not significantly correlated to $\hat{\beta}_{2i}$ is that these marketing activities provide sufficient information to physicians and internal influence loses importance. The negative correlation of $\hat{\beta}_{2i}$ and the other two instruments, medical journal advertising and direct-to-consumer advertising, is harder to explain. These results appear to be driven by a few brands that spend relatively large amounts on these instruments and have small estimates for β_{2i}.

Table 6.16.
Correlation between marketing instruments and the internal influence coefficient ($\hat{\beta}_{2i}$)

	ρ	p-value
detailing	-0.45	0.10
medical journal advertising	-0.55*	0.04
physician meetings	-0.36	0.21
direct-to-consumer advertising	-0.61*	0.02

*: p≤ 0.05

In order to check the insights revealed by the correlation analysis, we regress $\hat{\beta}_{10i}$ and $\hat{\beta}_{2i}$ on the marketing instruments and also on the order of entry of the brands into the marketplace. Seemingly Unrelated Regression (SUR) corrected by the approach developed by Wittink (1977) is used because this estimation method properly accounts for heteroscedasticity[16]. The SUR results (see Table 6.17) show that marketing activity and order of entry affect the trial rate of the new brands[17]. The marketing activity has a positive and significant effect on the basic propensity to try but a negative and significant effect on the internal influence. This suggests that the more marketing activity, the higher the propensity to try and the lower the

[16] Correction for heteroscedasticity is needed given that the dependent variables of the regressions are the parameter estimates from different brands (Wittink, 1977).
[17] We carried out this analysis considering the significant and not significant estimates of β_{2i}, and we repeat the same analysis by changing the not significant estimates to zeros. The results do not change.

effectiveness of the physicians' internal influence. As we mentioned earlier, the negative effect of marketing activity on internal influence seems to be driven by a few brands that spend relatively large amounts on marketing instruments and have small estimates for β_{2i}. This has to be further investigated. The order of entry effects are negative and significant for $\hat{\beta}_{10i}$ and $\hat{\beta}_{2i}$. This suggests that drugs launched early have advantages on the impact of the propensity to try and the internal influence, revealing the order of entry as an important strategic variable. As we expected, drugs launched early have a better opportunity to occupy a preferential position on the physicians' product space[18].

Table 6.17.
SUR results for the basic propensity to try ($\hat{\beta}_{10i}$) and the internal influence coefficient ($\hat{\beta}_{2i}$) -Aggregate marketing expenditures-

	$\hat{\beta}_{10i}$	$\hat{\beta}_{2i}$
constant	0.02	1.95***
marketing expenditures	0.002*	-0.05**
order of entry	-0.003*	-0.06*
R^2 (%)	51.56	66.41
R^2-adjusted (%)	42.75	60.30

***: p≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05

Due to multicollinearity in the marketing instruments, the separated effects of expenditures in detailing, medical journal advertising, physician meetings and direct-to-consumer advertising could not be detected. Hence, we are not able to determine the impact of each marketing instrument on the trial rate. However, Table 6.18 shows the effects of the expenditures on direct-to-physician marketing -"push" strategy- and direct-to-consumer advertising -"pull" strategy- on $\hat{\beta}_{10i}$ and $\hat{\beta}_{2i}$. The expenditures in direct-to-physician marketing have a positive and significant effect on $\hat{\beta}_{10i}$ and a negative although slightly significant effect on $\hat{\beta}_{2i}$. However, the expenditures in direct-to-consumer advertising have a non-significant effect on $\hat{\beta}_{10i}$ but a negative although slightly significant effect on $\hat{\beta}_{2i}$. This suggests that direct-to-physician marketing has a relevant impact on $\hat{\beta}_{10i}$ and a

[18] We also tried a regression which considers, apart from marketing expenditures and order of entry, the cross effect between these variables; however, results did not improve nor show additional significant information. We also introduced a cross effect variable in subsequent regressions but, again, results did not improve nor show additional significant information.

slightly significant impact on $\hat{\beta}_{2i}$ during the first 12 months after introduction, but direct-to-consumer advertising only has a demonstrable impact (slightly significant) on internal influence.

Table 6.18.
SUR results for the basic propensity to try ($\hat{\beta}_{10i}$) and the internal influence coefficient ($\hat{\beta}_{2i}$) -Disaggregate marketing expenditures-

	$\hat{\beta}_{10i}$	$\hat{\beta}_{2i}$
constant	0.01	1.84***
direct-to-physician marketing expenditures	0.01*	-0.04^{o}
direct-to-consumer advertising expenditures	-0.002	-0.06^{o}
order of entry	-0.003*	-0.06*
R^2 (%)	63.15	66.80
R^2-adjusted (%)	52.10	56.84

***: p≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05; ^{o}: p< 0.1

Our results show that, during the first 12 months after introduction:

i) the "informative" function of marketing activities is confirmed;
ii) the higher the marketing activities, the higher the propensity to try new brands;
iii) the higher the marketing activities, the lower the effectiveness of the physicians' internal influence.
iv) direct-to-consumer advertising expenditures ("pull" strategy) influence the propensity to try but have no demonstrable impact on internal influence.
v) the sooner a new drug enters the market, the higher the propensity to try and the higher the effectiveness of the physicians' internal influence.

Effects on the repeat rate

The cross-sectional effect of marketing expenditures is also related to the "persuasive" function of pharma marketing. This "persuasive" function influences the diffusion of a new product through the repeat rate. Table 6.19 shows the coefficients of the correlation between the average expenditures on each marketing instrument and the repeat rate during the first 12 months after introduction and for the complete period.

Table 6.19.
Correlation between marketing instruments and the repeat rate ($\hat{\beta}_{3i}$)

	12-month period		complete period	
	ρ	*p*-value	ρ	*p*-value
detailing	0.76*	0.002	0.76*	0.002
medical journal advertising	0.37	0.19	0.69*	0.01
physician meetings	0.62*	0.02	0.64*	0.01
direct-to-consumer advertising	0.23	0.43	0.75*	0.002

*: p≤ 0.05

Figures 6.4 and 6.5 show the scatter plots of the repeat rate and the mean detailing level for the 12-month and complete periods, respectively.

Figure 6.4.
Scatter plot of the repeat rate ($\hat{\beta}_{3i}$) and the mean of the log of detailing level (12-month period)

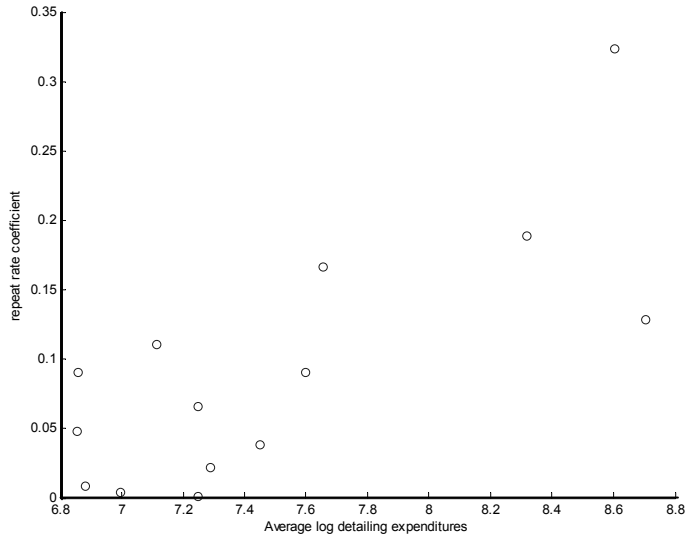


Figure 6.5.
Scatter plot of the repeat rate ($\hat{\beta}_{3i}$) and the mean of the log of detailing level (complete period)

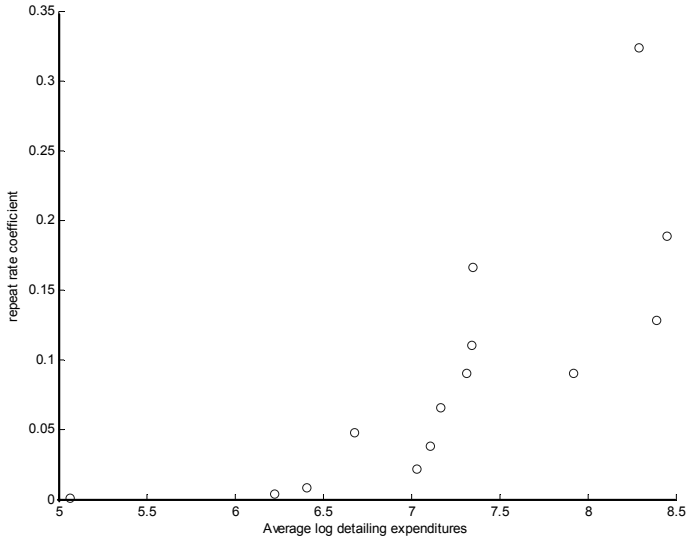


Table 6.19 shows that:

i) only direct-to-physician marketing (specifically, detailing and physicians meetings) is significantly correlated with the repeat rate shortly after introduction of the new product. However, for the complete period, all marketing instruments, including direct-to-consumer advertising, are significantly correlated with the repeat rate and show a high correlation; and
ii) the correlation between marketing instruments and the repeat rate increases when we consider the complete period.

Hence, results suggest that all marketing instruments are important in creating market power for the promoted drugs, although expenditures on direct-to-physician marketing ("push" strategy) show a more rapid influence on physicians than expenditures on direct-to-consumer advertising ("pull" strategy). Furthermore, expenditures on direct-to-consumer advertising appear to be important in creating market power for the promoted drugs. These results show the relevant role that a "pull" strategy plays in pharma marketing.

The SUR results (see Table 6.20) show that, both for the first 12 months after introduction and for the complete period, marketing expenditures have a positive and significant effect on the repeat rate. These results show that marketing activity has a rapid persuasive influence on physicians allowing pharmaceutical companies to protect themselves from competitor's products. The effect of the order of entry of the new brands into the marketplace is negative and significant for the 12-month period, but negative although not significant for the complete period. The results also show that an early entrance in the market creates barriers of entry during the first year after introduction. However, the order of entry loses importance for the complete period. This could be because, over time other aspects (such as the availability of the drug in a certain geographical area) are more important than the age of a drug (i.e. the time that the drug has been in the marketplace) to physicians' repeat prescription decisions. Furthermore, results show that the repeat rate is stronger related to marketing activities for the complete period than to marketing activities for the beginning of the period. This is expected given that the repeat rate is conceptually closer to the long run average market share of the new product.

Due to multicollinearity in the marketing instruments, we are not able to investigate the impact of each marketing instrument on the repeat rate. However, for the 12-month period (see Table 6.21), results reveal that the effect of direct-to-physician marketing is positive and significant and the effect of direct-to-consumer advertising is not significant[19]. Table 6.21 also shows the results for the complete period. The results seem to indicate, although not significant at the 5% level, that "pull" strategy becomes a barrier of entry for the complete period.

[19] We carried out this analysis (Tables 6.20 and 6.21) considering the significant and not significant estimates of β_{3i}, and we repeat the same analysis by changing the non significant estimates to zeros. The results do not change.

Table 6.20.
SUR results for the repeat rate ($\hat{\beta}_{3i}$)
-Aggregate marketing expenditures-

	$\hat{\beta}_{3i}$ 12-month period	$\hat{\beta}_{3i}$ complete period
constant	-0.004	-0.05
marketing expenditures	0.01*	0.01***
order of entry	-0.01*	-0.01
R^2 (%)	46.75	69.28
R^2-adjusted (%)	37.07	63.70

***: p≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05

Table 6.21.
SUR results for the repeat rate ($\hat{\beta}_{3i}$)
-Disaggregate marketing expenditures-

	$\hat{\beta}_{3i}$ 12-month period	$\hat{\beta}_{3i}$ complete period
constant	-0.12	-0.03
direct-to-physician marketing expenditures	0.02*	0.01^{o}
direct-to-consumer advertising expenditures	-0.003	0.01^{o}
order of entry	-0.01^{o}	-0.01
R^2 (%)	56.48	69.46
R^2-adjusted (%)	43.42	60.29

*: p≤ 0.05; ^{o}: p< 0.1

6.6.2.1. Pooled cross-sectional analysis

We also carried out the cross-sectional analysis for the osteoarthritis-rheumatoid-arthritis and asthma categories. Because of the small number of new drugs in each category, the SUR estimation method does not show significant estimates, except for $\hat{\beta}_{3i}$ in osteoarthritis-rheumatoid-arthritis. To overcome this problem we pool the three categories and carry out a pooled SUR estimation method by allowing the constant term to vary across categories. Given that the osteoarthritis-rheumatoid-arthritis and asthma categories have branded and generic new drugs, we include the dummy "kind" (kind = 1 if the new product is a branded drug; kind = 0 if the new product is a generic drug) in the system. Tables 6.22 through 6.25 show the results.

Table 6.22 shows the effects of the marketing expenditures, the order of entry and the kind of drug (branded or generic drug) on $\hat{\beta}_{10i}$ and $\hat{\beta}_{2i}$ during the first 12

months after introduction. Table 6.23 shows the same effects but differentiates between direct-to-physician marketing and direct-to-consumer advertising. Again, due to multicollinearity in the marketing instruments, separate effects could not be investigated.

Table 6.22.
Pooled SUR results for the basic propensity to try ($\hat{\beta}_{10i}$) and the internal influence coefficient ($\hat{\beta}_{2i}$) -Aggregate marketing expenditures-

	$\hat{\beta}_{10i}$	$\hat{\beta}_{2i}$
constant – rhinitis category	0.08**	1.13**
constant – osteoarthritis-rheumatoid-arthritis category	0.06**	1.30***
constant – asthma category	0.06**	0.80*
marketing expenditures	0.004*	-0.03
order of entry	-0.002	-0.05*
kind	-0.08*	0.38
R^2 (%)	29.80	34.35
R^2-adjusted (%)	16.80	22.19

***: p≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05

Table 6.23.
Pooled SUR results for the basic propensity to try ($\hat{\beta}_{10i}$) and the internal influence coefficient ($\hat{\beta}_{2i}$) -Disaggregate marketing expenditures-

	$\hat{\beta}_{10i}$	$\hat{\beta}_{2i}$
constant – rhinitis category	0.08**	1.10***
constant – osteoarthritis-rheumatoid-arthritis category	0.06*	1.21***
constant – asthma category	0.06*	0.73*
direct-to-physician marketing expenditures	0.005*	-0.01
direct-to-consumer advertising expenditures	0.001	-0.07
order of entry	-0.002	-0.04^{o}
kind	-0.10*	0.13
R^2 (%)	31.46	37.34
R^2-adjusted (%)	15.64	22.88

***: p≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05; ^{o}: p< 0.1

The results for $\hat{\beta}_{10i}$ and $\hat{\beta}_{2i}$ in the pooled cross-sectional analysis with aggregate marketing expenditures are similar to those when we differentiate between marketing directed at physicians and direct-to-consumer advertising. The coefficient for the variable "kind" is negative and significant for $\hat{\beta}_{10i}$. This indicates

that generic new drugs tend to have a higher propensity to try than newly introduced branded drugs.

Table 6.24 shows the effects of the marketing expenditures, the order of entry and the kind of drug (branded or generic drug) on $\hat{\beta}_{3i}$ during the first 12 months after introduction and for the complete period. Table 6.25 shows the same effects but differentiates between direct-to-physician marketing and direct-to-consumer advertising during the first 12 months after introduction. Due to the correlation between both types of strategies we are not able to estimate their separate effects for the complete period.

Table 6.24.
Pooled SUR results for the repeat rate ($\hat{\beta}_{3i}$)
-Aggregate marketing expenditures-

	$\hat{\beta}_{3i}$ 12-month period	$\hat{\beta}_{3i}$ complete period
constant – rhinitis category	0.11^{o}	0.09*
constant – osteoarthritis-rheumatoid-arthritis category	0.04	0.06
constant – asthma category	0.10*	0.10*
marketing expenditures	0.01*	0.01***
order of entry	-0.004	-0.004
kind	-0.12^{o}	-0.14*
R^2 (%)	18.00	41.06
R^2-adjusted (%)	2.82	30.14

***: p≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05; ^{o}: p< 0.1

Table 6.25.
Pooled SUR results for the repeat rate ($\hat{\beta}_{3i}$)
-Disaggregate marketing expenditures-

	$\hat{\beta}_{3i}$ 12-month period
constant – rhinitis category	0.10*
constant – osteoarthritis-rheumatoid-arthritis category	0.04
constant – asthma category	0.10^{o}
direct-to-physician marketing expenditures	0.01^{o}
direct-to-consumer advertising expenditures	0.005
order of entry	-0.003
kind	-0.14^{o}
R^2 (%)	18.29
R^2-adjusted (%)	-0.01

**: p≤ 0.001; *: p≤ 0.05; ^{o}: p< 0.1

The cross-section analyses for the three analyzed categories show similar results for the repeat rate during the first 12 months after introduction, whether we consider aggregate marketing expenditures or differentiate between marketing directed at physicians and direct-to-consumer advertising. We also get the same results for the complete period. As with the propensity to try, the results seem to confirm that, during the first 12 months after introduction, generic new drugs have advantages on the repeat rate. This result is also shown for the complete period.

In summary, the cross-sectional analysis for the three categories analyzed confirms the conclusions of the longitudinal analysis for the rhinitis category. The results of the pooled SUR analysis indicate that:

i) the "informative" and "persuasive" functions of marketing activities exist;
ii) the higher the marketing activities, the higher the propensity to try new brands;
iii) marketing activities have no demonstrable impact on the effectiveness of physicians' internal influence. A possible explanation for this could be that detailing, medical journal advertising and/or physician meetings provide sufficient information to physicians and internal influence loses importance.
iv) the higher the marketing activities, the higher the repeat rate (for the 12-month and complete periods);
v) marketing activities have a different impact on the trial rate during the first 12 months after introduction. The impact of marketing activities is higher on the repeat rate than on the propensity to try;
vi) direct-to-consumer advertising ("pull" strategy) seems neither to affect the internal influence, the propensity to try nor the repeat rate during the first 12 months after introduction;
vii) generic new drugs have advantages on the impact of the propensity to try and the repeat rate;
viii) order of entry has no demonstrable impact on the repeat and trial rates when the three categories are considered. Order of entry seems to be more important in the 'young' rhinitis category than in the other two 'older' categories, where a number of generic drugs compete with branded drugs.

6.6.3. Investigating a new approach: a recursive window approach

Our results show that marketing expenditures have an impact on the diffusion speed of pharmaceutical products. Multicollinearity problems do not allow for simultaneous investigation of the effect of marketing on all three diffusion parameters (external influence, internal influence and the repeat rate). In this subsection we employ a new approach that allows us to analyze the impact of the different marketing instruments on the diffusion parameters over time.

We start by considering the following extension of model 1 where the diffusion parameters are considered as time-varying parameters:

$$s_{i,t} = \left[\beta_{10i,t} + \beta_{2i,t} \left[\frac{s_{i,t-1}}{m_t} \right] \right] \left[m_t - q_{i,t-1} \right] + \beta_{3i,t} q_{i,t-1} \tag{6.8}$$

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

We estimate the extended model 1 -Equation (6.8)- by dividing the complete period of study (i.e. a time window of 96 observations) into a number of time windows. We try two approaches: 1) a recursive time window approach, and 2) a rolling time window approach. The recursive time window approach keeps the window origin fixed at the first time period in the sample and successively adds observations, thereby increasing the number of observations one by one. The rolling time window approach uses a fixed number of observations within each window and estimates the model in every window before moving on to the next (Swanson and White, 1997; Tashman, 2000). These two approaches are appealing from a diagnostic perspective, but also from a descriptive perspective given that we can use data available prior to a certain time to evaluate the marketing actions during that period. However, there is a characteristic of the rolling approach that makes this approach less appropriate than the recursive one: the rolling window removes the initial information (first observations) of the sample. This information is particularly relevant to represent the data generating process when we are analyzing the diffusion process of new products. Nevertheless, we investigate both approaches.

We estimate the model in Equation (6.8) using the recursive windows approach where the first window has 15 monthly observations (the minimum number of observations to properly estimate the model), we also use the rolling windows approach with windows of 25 monthly observations[20]. We employ scatter plots to explore the relationship between the estimates of the time-varying diffusion parameters ($\hat{\beta}_{10i,t}$, $\hat{\beta}_{2i,t}$ and $\hat{\beta}_{3i,t}$) and the mean level of expenditures on the different marketing instruments in each window: detailing, medical journal advertising, physician meetings, direct-to-consumer advertising, aggregate marketing (by adding all marketing instruments) and direct-to-physician marketing (by adding detailing, medical journal advertising and physician meetings). Results for the rolling window approach do not show any insights on the role that marketing actions play on the diffusion processes of the new drugs. However, the scatter plots using the recursive time window reveal interesting patterns. For

[20] The rolling window approach requires the choice of a window size taking into account that small sizes suffer from low test power and large sizes may not pick up changes early and late in the sample. We tried different window sizes (Pauwels and Hanssens, 2004).

example, a positive correlation between $\hat{\beta}_{10i,t}$ and expenditures on detailing for some drugs.

Appendix 6B presents several plots and summary tables of some of the new drugs in the rhinitis category that show the temporal pattern of the diffusion parameters, the temporal pattern of the different marketing instruments and the correlation among them. In general terms, for the complete category (14 new branded drugs), the plots show that $\hat{\beta}_{10i,t}$ and $\widehat{\beta}_{3i,t}$ follow the same temporal tendency as the mean level of expenditures on marketing directed at physicians, especially detailing. However, $\hat{\beta}_{2i,t}$ shows contrary behavior to $\hat{\beta}_{10i,t}$ and $\widehat{\beta}_{3i,t}$.

The scatter plots and the correlation analysis reveal that, in general terms, $\hat{\beta}_{10i,t}$ and $\widehat{\beta}_{3i,t}$, show a positive and significant correlation with the mean level of expenditures on direct-to-physician marketing, particularly on detailing and physician meetings. There is no clear pattern for medical journal advertising nor for direct-to-consumer advertising (although for direct-to-consumer advertising there seems to be a positive correlation). The results show the opposite pattern for $\hat{\beta}_{2i,t}$, which is consistent with our results for the longitudinal and the cross-sectional analysis. In future research we plan to investigate these patterns in more detail further.

6.7. Conclusions

In this study, we investigate both longitudinal and cross-sectional effects of marketing expenditures on the diffusion of new pharmaceuticals in the "rhinitis"-category. We model the diffusion process of fourteen new brands that are introduced within the observational period. We also model the diffusion processes of new drugs in another two categories, one (asthma) similar and another (osteoarthritis-rheumatoid-arthritis) quite different from rhinitis.

We employ a family of trial-repeat diffusion models that allows us (1) to detect the appropriate allocation for marketing instruments in the trial rate, (2) to accommodate heterogeneity in the effects of the different marketing instruments, and (3) to accommodate a time-varying trial rate that is influenced by both own and competitors' marketing expenditures. In a second-stage analysis we determine the cross-sectional effects of marketing expenditures on the trial rate and on the repeat rate.

Using eight years of US monthly data we find support for the hypothesis that changes in the trial rate are positively related to changes in own marketing expenditures. We also find support for the hypothesis that the trial rate is negatively affected by competitors' marketing expenditures. The results indicate that, in general terms, the external influence formulation of the proposed trial-repeat diffusion model is the most appropriate to incorporate marketing variables for the three categories analyzed.

Besides these longitudinal effects we also find that cross-sectional differences in marketing expenditures are significantly affecting the trial rate and the repeat rate of the diffusion processes of new pharmaceutical products. Specifically, our results show that the mean level of marketing expenditures is positively related to the basic propensity to try the new product and to the repeat rate but apparently not related to the internal influence. These results hold for all three drug categories analyzed, except that in the case of rhinitis, marketing expenditures are negatively related to internal influence. These results imply that the basic propensity to try brands with a high level of marketing expenditures is larger than that of brands with a lower level of marketing expenditures. In addition, such effects exist for the repeat rate as well. However, the relationship is either non existent or the opposite for the internal influence. We argue that marketing expenditures both have an informative and persuasive influence on the diffusion of new products in the category under study. These results are in line with those of Narayanan, Manchanda and Chintagunta (2004) who find evidence for the presence of both indirect (informative) and direct (persuasive) effects of advertising. Furthermore, order of entry is confirmed to play an important role in the launch strategy of the new products in the rhinitis category, which has only branded drugs. However, when we consider the three categories, order of entry becomes less important. In this analysis we also find that generics tend to have a higher basic propensity to try, a larger effect of internal influence and a higher repeat rate than branded drugs.

Finally, we investigate the longitudinal patterns further using a recursive window approach. Preliminary results for the rhinitis category show that the marketing directed at physicians, especially for detailing, but also for physician meetings, is a key factor in determining the temporal pattern of the diffusion parameters. There seems not to be a clear pattern, for medical journal advertising, and for direct-to-consumer advertising. Apart from these preliminary results, which need future research, we are also investigating the effect of the different marketing instruments on the number of triers and repeaters of prescription drugs.

Appendix 6A. Some results of other categories

Tables 6A.1 and 6A.2 show the Akaike Information Criterion for models 2E, 2I, 3E and 3I for osteoarthritis-rheumatoid-arthritis and for asthma, respectively.

Table 6.A1.
Akaike Information Criterion -osteoarthritis-rheumatoid-arthritis-.

Brand code	model 2			model 3		
	model 2E	model 2I	preferred model	model 3E	model 3I	preferred model
3	11.27	10.53	model 2I	10.63	10.25	model 2I
12	11.99	11.92	model 2I	11.22	10.97	model 2I
13	9.77	9.49	model 2I	9.58	9.49	model 2I
4	11.23	11.18	model 2I	10.12	11.08	model 2E
11	7.56	6.55	model 2I	6.97	6.58	model 2I
5	7.44	7.45	model 2E	7.05	7.18	model 2E
1	8.29	8.24	model 2I	8.16	7.95	model 2I
2	11.79	12.24	model 2E	11.73	12.09	model 2E
19	10.39	10.52	model 2E	10.34	10.48	model 2E

Table 6.A2.
Akaike Information Criterion -asthma-.

Brand code	model 2			model 3		
	model 2E	model 2I	preferred model	model 3E	model 3I	preferred model
26	7.29	7.32	model 2E	7.13	7.35	model 2E
18	7.62	7.77	model 2E	7.64	7.78	model 2E
10	7.18	7.20	model 2E	7.15	7.17	model 2E
3	10.43	10.39	model 2I	10.38	10.32	model 2I
13	5.23	5.23	model 2I	5.25	5.26	model 2E
11	8.94	8.84	model 2I	8.80	8.62	model 2I
1	7.45	7.44	model 2I	7.42	7.43	model 2E
9	7.50	7.54	model 2E	7.54	7.57	model 2E
17	8.09	8.12	model 2E	8.15	8.17	model 2E
20	7.73	7.69	model 2I	7.33	7.58	model 2E

Tables 6A.3, 6A.4 and 6A.5 show the estimation results of models 1, 2E and 3E, respectively, for the osteoarthritis-rheumatoid-arthritis category.

Table 6A.3.
Estimation results of model 1 -osteoarthritis-rheumatoid-arthritis-.

Brand code	Trial rate: External influence $\hat{\beta}_{10i}$	Trial rate: Internal influence $\hat{\beta}_{2i}$	Repeat rate $\hat{\beta}_{3i}$	Average market share (in units)	MAD	MAPE	r
3	-0.06***	2.01***	0.06***	0.03	54.87	16.39	0.82
12		0.63**	0.13***	0.10	88.11	9.34	0.93
13	-0.01***	1.83***	0.02***	0.01	23.69	20.81	0.92
4	-0.02*	1.75***	0.02***	0.03	34.60	14.44	0.63
11	0.002°	1.17***		0.01	4.82	4.72	0.96
5	0.01***	0.76***	0.01*	0.02	3.87	2.30	0.89
1	0.01***	1.08***		0.02	3.58	1.74	0.98
2	0.08***	0.30*	0.19***	0.17	18.95	1.18	0.95
19	0.02***	0.56***	0.20***	0.12	6.21	0.53	0.99

***: p≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05 ; °: p<0.1

Table 6A.4.
Estimation results of model 2E -osteoarthritis-rheumatoid-arthritis-.

Brand code	Trial rate: External influence $\hat{\beta}_{10i}$	Trial rate: influence $\hat{\beta}_{11i}$	Trial rate: Internal influence $\hat{\beta}_{2i}$	Repeat rate $\hat{\beta}_{3i}$	Average market share (in units)	MAD	MAPE	r
3	0.04***	0.01***	1.23***	0.06*	0.03	50.15	14.98	0.86
12	0.08***	0.01***		0.14***	0.10	65.97	7.00	0.93
13	0.02***		1.80***		0.01	23.76	20.87	0.92
4	0.05***	-0.002*	1.86***	0.02***	0.03	35.14	14.67	0.60
11	0.02***	-0.001°	1.20***	0.001***	0.01	4.65	4.55	0.96
5	0.02***		0.77***	0.01***	0.02	3.75	2.23	0.89
1	0.03***		1.08***	0.01***	0.02	3.56	1.73	0.98
2	0.11***	0.07**		0.19***	0.17	14.32	0.89	0.97
19	0.08***		0.48***	0.20***	0.12	5.22	0.44	0.99

***: p≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05; °: p<0.1

Table 6A.5.
Estimation results of model 3E -osteoarthritis-rheumatoid-arthritis-.

Brand code	Trial rate				Repeat rate	Average market share (in units)	MAD	MAPE	r
	External influence			Internal influence					
	$\hat{\beta}_{10i}$	$\hat{\beta}_{11i}$	$\hat{\beta}_{11ci}$	$\hat{\beta}_{2i}$	$\hat{\beta}_{3i}$				
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^{*}	-0.02^{o}		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≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05; ^{o}: p<0.1

Tables 6A.6, 6A.7 and 6A.8 show the estimation results of models 1, 2E and 3E, respectively, for the asthma category.

Table 6A.6.
Estimation results of model 1 -asthma-.

Brand code	Trial rate		Repeat rate	Average market share (in units)	MAD	MAPE	r
	External influence	Internal influence					
	$\hat{\beta}_{10i}$	$\hat{\beta}_{10i}$	$\hat{\beta}_{10i}$				
26	0.01^{***}	0.29^{*}	0.01^{***}	0.02	5.69	11.11	0.84
18	0.02^{***}	0.32^{***}	0.07^{***}	0.04	8.20	2.33	0.99
10		1.09^{***}	0.01^{***}	0.01	4.51	5.23	0.93
3	0.09^{***}	0.26^{***}	0.29^{***}	0.24	19.31	1.01	0.99
13	0.001^{**}	0.82^{***}	0.02^{***}	0.01	1.17	1.34	0.99
11	0.003^{o}		0.48^{***}	0.05	7.07	1.86	0.99
1	0.003^{*}	0.99^{***}	0.02^{***}	0.02	3.30	1.87	0.98
9	0.004^{***}	0.60^{***}	0.06^{***}	0.03	3.29	1.27	0.99
17	0.002^{*}	0.47^{*}	0.03^{*}	0.01	3.42	6.75	0.85
20	0.01^{***}	0.20^{**}	0.18^{***}	0.06	2.99	0.67	0.99

***: p≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05 ; ^{o}: p<0.1

Table 6A.7.
Estimation results of model 2E -asthma-.

Brand code	Trial rate			Repeat rate	Average market share (in units)	MAD	MAPE	r
	External influence		Internal influence					
	$\hat{\beta}_{10i}$	$\hat{\beta}_{11i}$	$\hat{\beta}_{2i}$	$\hat{\beta}_{3i}$				
26	0.01^{***}	0.001^{*}		0.02^{***}	0.02	5.38	10.51	0.86
18	0.03^{***}	0.003^{***}	0.32^{***}	0.07^{***}	0.04	7.39	2.10	0.99
10	0.01^{***}		1.07^{***}	0.01^{***}	0.01	4.55	5.28	0.93
3	0.16^{***}		0.25^{***}	0.29^{***}	0.24	19.46	1.02	0.99
13	0.01^{***}		0.81^{***}	0.02^{***}	0.01	1.17	1.33	0.99
11				0.64^{**}	0.05	6.48	1.70	0.99
1	0.03^{***}		0.97^{***}		0.02	3.23	1.82	0.98
9	0.02^{***}		0.55^{***}	0.06^{***}	0.03	3.04	1.17	0.99
17	0.01^{***}		0.44^{*}	0.03^{*}	0.01	3.29	6.49	0.85
20	0.02^{***}		0.25^{***}	0.17^{***}	0.06	2.96	0.66	0.99

***: p≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05

Table 6A.8.
Estimation results of model 3E -asthma-.

Brand code	Trial rate				Repeat rate	Average market share (in units)	MAD	MAPE	r
	External influence			Internal influence					
	$\hat{\beta}_{10i}$	$\hat{\beta}_{11i}$	$\hat{\beta}_{11ci}$	$\hat{\beta}_{2i}$	$\hat{\beta}_{3i}$				
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≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05

Tables 6A.9 and 6A.10 show the results of the likelihood ratio tests for osteoarthritis-rheumatoid-arthritis and asthma, respectively.

Table 6A.9.
Likelihood ratio tests -osteoarthritis-rheumatoid-arthritis-.

	Restricted model: model 1 Unrestricted model: model 2E		Restricted model: model 1 Unrestricted model: model 3E		Restricted model: model 2E Unrestricted model: model 3E	
Brand code	χ^2	Retained model	χ^2	Retained model	χ^2	Retained model
3	24.98*	model 2E	85.47*	model 3E	60.48*	model 3E
12	37.94*	model 2E	111.75*	model 3E	73.81*	model 3E
13	0.04	model 1	19.61*	model 3E	19.56*	model 3E
4	2.66	model 1	75.78*	model 3E	78.45*	model 3E
11	1.88	model 1	48.25*	model 3E	46.37*	model 3E
5	0.00	model 1	23.21*	model 3E	23.21*	model 3E
1	2.26	model 1	7.30*	model 3E	5.03*	model 3E
2	12.80*	model 2E	16.49*	model 3E	3.69^{o}	model 3E
19	2.62	model 1	5.23^{o}	model 3E	2.61	model 2E

*: Significant at 0.05 level; ^{o}: Significant at 0.1 level.

Table 6A.10.
Likelihood ratio tests -asthma-.

	Restricted model: model 1 Unrestricted model: model 2E		Restricted model: model 1 Unrestricted model: model 3E		Restricted model: model 2E Unrestricted model: model 3E	
Brand code	χ^2	Retained model	χ^2	Retained model	χ^2	Retained model
26	11.00*	model 2E	26.47*	model 3E	15.47*	model 3E
18	23.92*	model 2E	24.23*	model 3E	0.31	model 2E
10	1.85	model 1	6.21*	model 3E	4.37*	model 3E
3	0.83	model 1	5.69^{o}	model 3E	4.86*	model 3E
13	0.03	model 1	1.06	model 1	1.03	model 2E
11	0.14	model 1	9.59*	model 3E	9.73*	model 3E
1	0.48	model 1	3.55	model 1	3.08^{o}	model 3E
9	2.95^{o}	model 2E	3.08	model 1	0.13	model 2E
17	1.05	model 1	1.05	model 1	0.002	model 2E
20	2.12	model 1	17.66*	model 3E	15.55*	model 3E

*: Significant at 0.05 level; ^{o}: Significant at 0.1 level.

Appendix 6B. Some results from the recursive window approach

Figures 6B.1 to 6B.4 show, for some drugs in the rhinitis category, the following plots:

i) in the first line: the temporal pattern of the mean level of expenditures on detailing, medical journal advertising, physician meetings and direct-to-consumer advertising,
ii) in the first column: the temporal pattern of the estimates of the time-varying diffusion parameters -the basic propensity to try ($\hat{\beta}_{10i,t}$), the internal influence coefficient ($\hat{\beta}_{2i,t}$) and the repeat rate ($\hat{\beta}_{3i,t}$)-, and
iii) in the other lines and columns: the scatter plots of the estimates of the time-varying diffusion parameters and the mean level of expenditures on each marketing instrument.

Figures 6B.5 to 6B.10 show, for some drugs in the rhinitis category, the following plots:

i) in the first line: the temporal pattern of the mean level of expenditures on direct-to-consumer advertising, direct-to-physician marketing and aggregate marketing,
ii) in the first column: the temporal pattern of the estimates of the time-varying diffusion parameters -the basic propensity to try ($\hat{\beta}_{10i,t}$), the internal influence coefficient ($\hat{\beta}_{2i,t}$) and the repeat rate ($\hat{\beta}_{3i,t}$)-, and
iii) in the other lines and columns: the scatter plots of the estimates of the time-varying diffusion parameters and the mean level of expenditures on each marketing instrument.

In general terms, we show that $\hat{\beta}_{10i,t}$ and $\hat{\beta}_{3i,t}$ follow the same temporal tendency as the mean level of expenditures on detailing, direct-to-physician marketing and aggregate marketing. This is not the common pattern for the other marketing instruments. This seems to indicate that detailing expenditures are relevant in determining the temporal tendency of these diffusion parameters. However, $\hat{\beta}_{2i,t}$ follows the opposite pattern to $\hat{\beta}_{10i,t}$ and $\hat{\beta}_{3i,t}$. Table 6B.1 summarizes these results.

Table 6B.1
Temporal patterns of the time-varying diffusion parameters and the mean of the log of expenditures on the marketing instruments

	Brand 1	Brand 4	Brand 9	Brand 12
$\hat{\beta}_{10i,t}$	increasing	almost constant with a slight decreasing tendency	decreasing at the beginning and increasing at the end	decreasing at the beginning and increasing at the end
$\hat{\beta}_{2i,t}$	decreasing	almost constant with a slight increasing tendency	increasing at the beginning and decreasing at the end	increasing at the beginning and decreasing at the end
$\hat{\beta}_{3i,t}$	increasing	decreasing	decreasing at the beginning and increasing at the end	decreasing at the beginning and increasing at the end
Average log detailing expenditures	slight increasing tendency	decreasing	slight increasing tendency	slight increasing tendency
Average log medical journal advertising expenditures	decreasing at the beginning and increasing at the end	decreasing	decreasing at the beginning with a slight increase at the end	decreasing
Average log physician meetings Expenditures	increasing	decreasing	decreasing at the beginning and increasing at the end	slight increasing tendency
Average log direct-to-consumer advertising expenditures	slight increasing tendency	increasing at the beginning and then decreasing	increasing	increasing
Average log direct-to-physician marketing expenditures	slight increasing tendency	decreasing	decreasing	decreasing with a slight increase at the end
Average log marketing expenditures	increasing	decreasing	slight decreasing tendency	increasing

Table 6B.2 summaries the results from the scatter plots and shows the correlation coefficients with the level of significance. In general terms, these results show a positive and significant correlation between $\hat{\beta}_{10i,t}$ and the mean level

of expenditures on detailing, physician meetings, direct-to-physician marketing and aggregate marketing. This reveals that high values of $\hat{\beta}_{10i,t}$ are related to high levels of expenditures on direct-to-physician marketing, particularly on detailing and physician meetings. There is not a clear pattern for medical journal advertising nor direct-to-consumer advertising. The results show the same pattern for $\hat{\beta}_{3i,t}$ but the opposite for $\hat{\beta}_{2i,t}$.

Table 6B.2
Correlation between the time-varying diffusion parameters and the mean level of expenditures on the marketing instruments

	Average log detailing expenditures	Average log medical journal adv. expenditures	Average log physician meetings expenditures	Average log direct-to-consumer advertising expenditures	Average log direct-to-physician marketing expenditures	Average log marketing expenditures
Brand 1						
$\hat{\beta}_{10i,t}$	0.95***	0.52**	0.91***	0.78***	0.93***	0.91***
$\hat{\beta}_{2i,t}$	-0.94***	-0.52**	-0.88***	-0.78***	-0.91***	-0.90***
$\hat{\beta}_{3i,t}$	0.88***	0.50*	0.82***	0.74***	0.85***	0.85***
Brand 4						
$\hat{\beta}_{10i,t}$	0.76***	0.71***	0.62***	0.44**	0.73***	0.71***
$\hat{\beta}_{2i,t}$	-0.91***	-0.94***	-0.86***	-0.49**	-0.92***	-0.88***
$\hat{\beta}_{3i,t}$	0.78***	0.89***	0.77***	0.26^{o}	0.84***	0.75***
Brand 9						
$\hat{\beta}_{10i,t}$	0.35*	0.84***	0.39*	-0.71***	0.88***	0.88***
$\hat{\beta}_{2i,t}$	-0.72***	0.21	-0.64***	-0.20	0.05	-0.05
$\hat{\beta}_{3i,t}$	0.54***	0.05	0.73***	-0.08	0.22^{o}	0.28*
Brand 12						
$\hat{\beta}_{10i,t}$	0.82***	-0.77***	0.79***	0.77***	-0.50***	0.68**
$\hat{\beta}_{2i,t}$	-0.80***	0.77***	-0.78***	-0.76***	0.51*	-0.67**
$\hat{\beta}_{3i,t}$	0.76***	-0.76***	0.74***	0.74***	-0.53*	0.63**

***: p≤ 0.0001; **: p≤ 0.001; *: p≤ 0.05 ; ^{o}: p<0.1

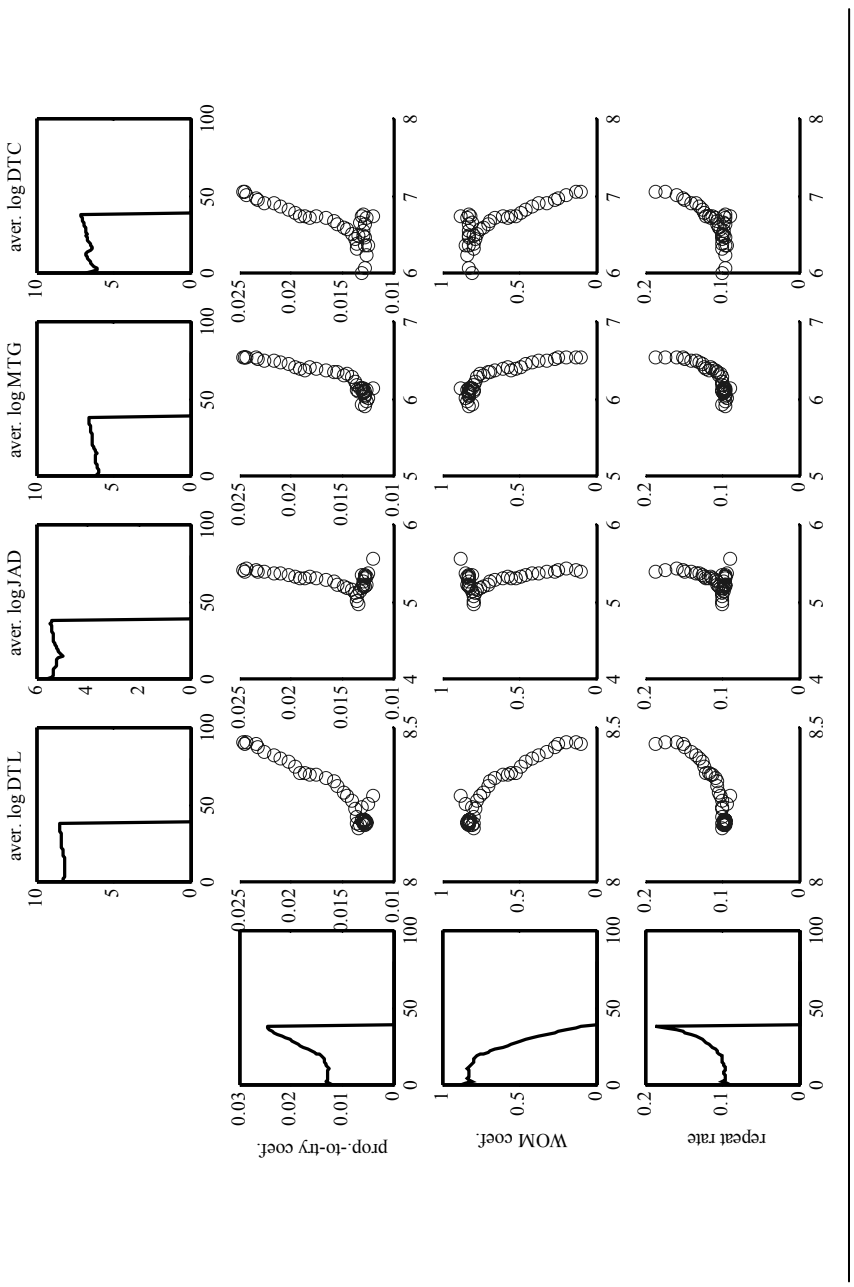


Figure 6B.1. Time-varying diffusion parameters –basic propensity to try ($\beta_{10i,t}$), internal influence ($\beta_{2i,t}$) and repeat rate ($\beta_{3i,t}$)- and the mean of the log of expenditures on detailing (DTL), medical journal advertising (JAD), physician meetings (MTG) and direct-to-consumer advertising (DTC). [Drug No 1 – rhinitis category]

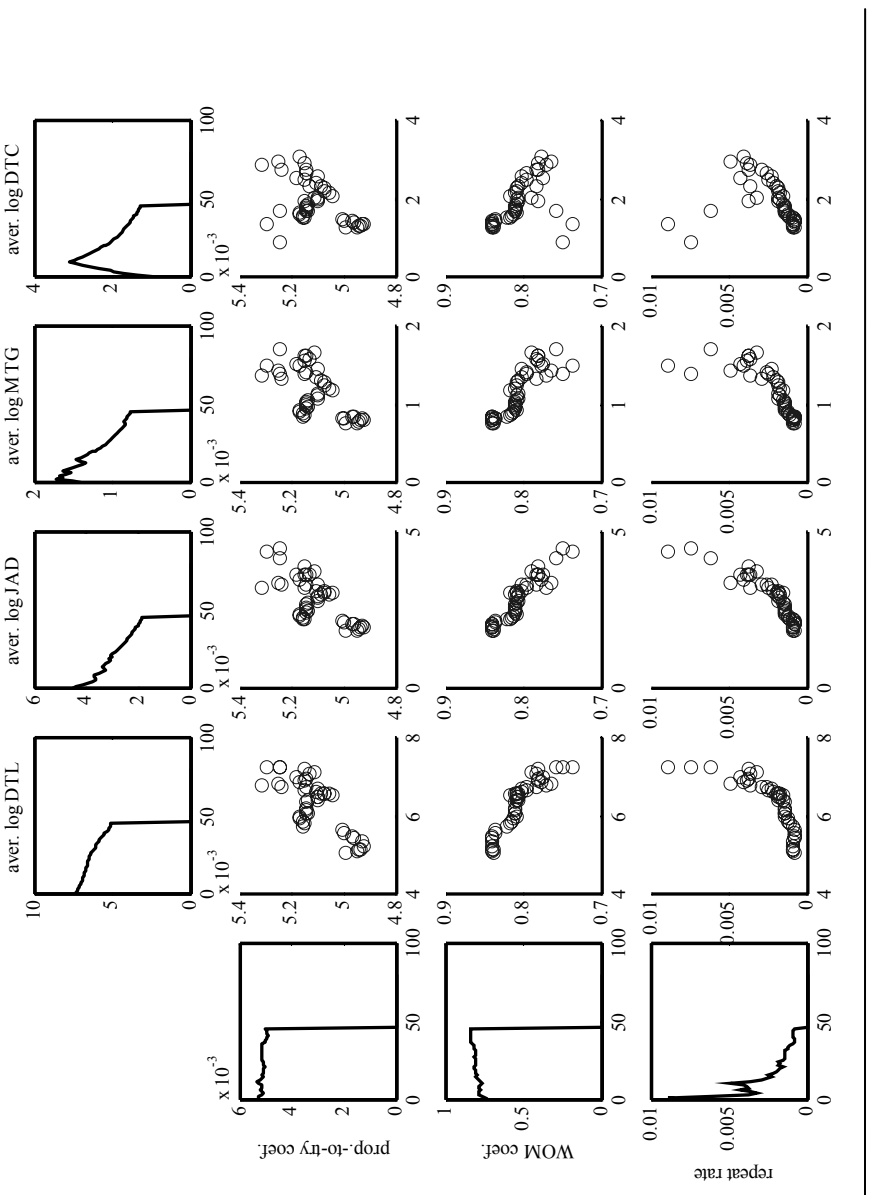


Figure 6B.2. Time-varying diffusion parameters –basic propensity to try ($\beta_{10i,t}$), internal influence ($\beta_{2i,t}$) and repeat rate ($\beta_{3i,t}$)- and the mean of the log of expenditures on detailing (DTL), medical journal advertising (JAD), physician meetings (MTG) and direct-to-consumer advertising (DTC). [Drug No 4 – rhinitis category]

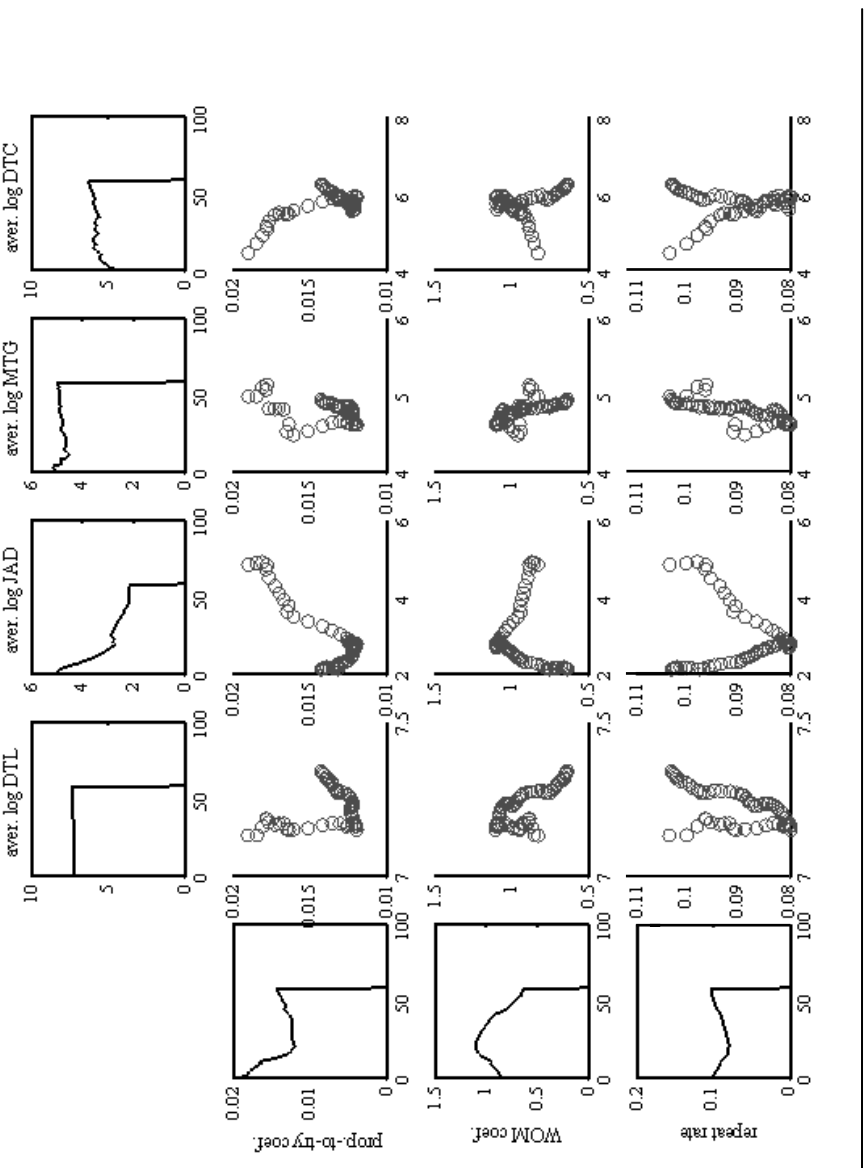


Figure 6B.3. Time-varying diffusion parameters –basic propensity to try ($\beta_{0i,t}$), internal influence ($\beta_{2i,t}$) and repeat rate ($\beta_{3i,t}$)- and the mean of the log of expenditures on detailing (DTL), medical journal advertising (JAD), physician meetings (MTG) and direct-to-consumer advertising (DTC). [Drug No 9 – rhinitis category]

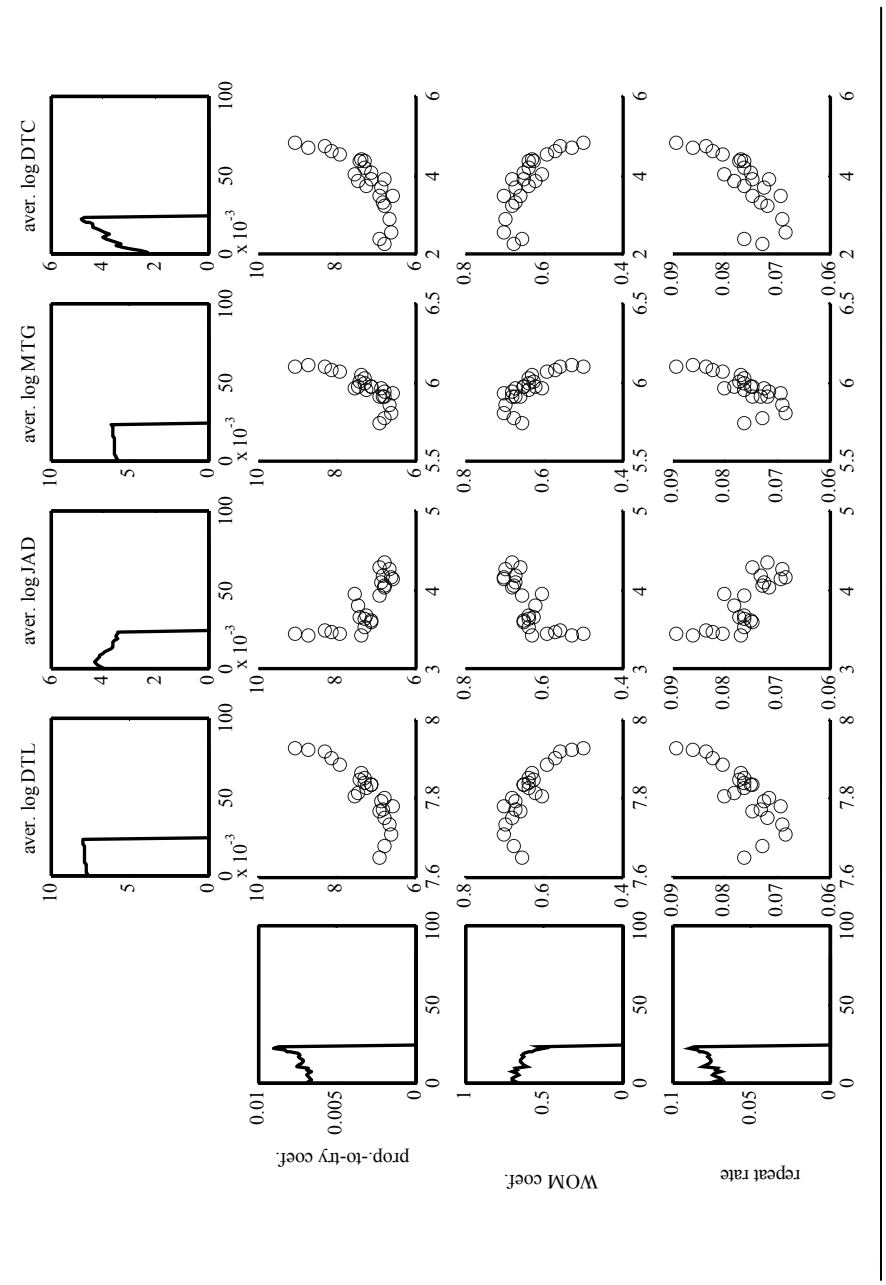


Figure 6B.4. Time-varying diffusion parameters –basic propensity to try ($\beta_{10i,t}$), internal influence ($\beta_{2i,t}$) and repeat rate ($\beta_{3i,t}$)- and the mean of the log of expenditures on detailing (DTL), medical journal advertising (JAD), physician meetings (MTG) and direct-to-consumer advertising (DTC). [Drug No 12 – rhinitis category]

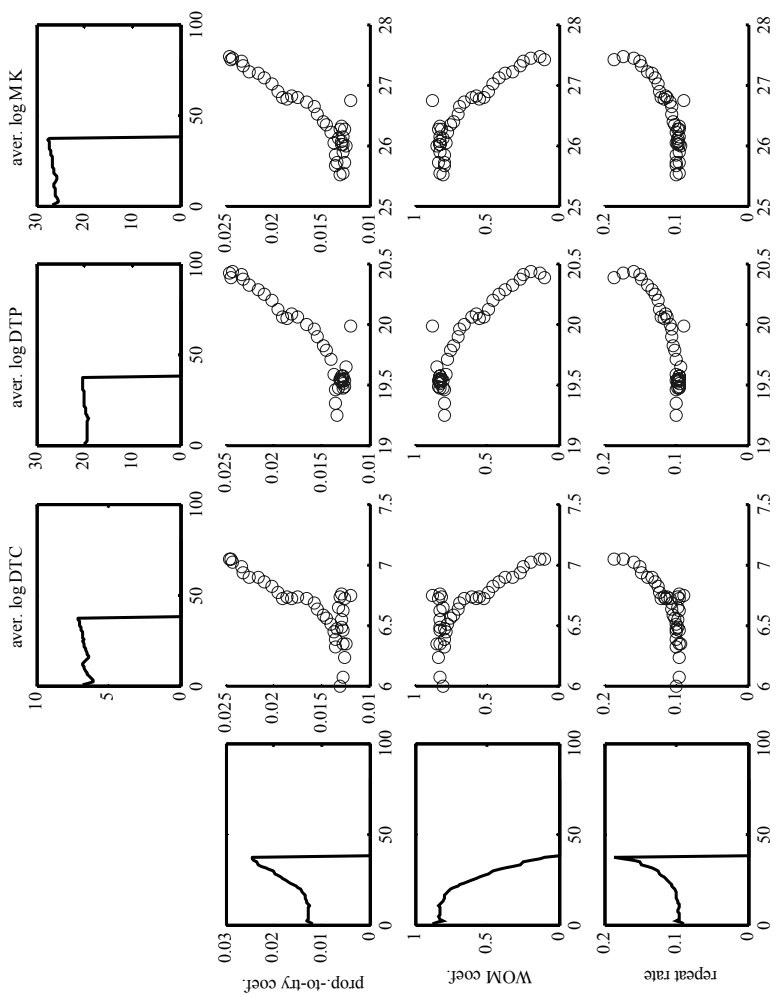


Figure 6B.5. Time-varying diffusion parameters –basic propensity to try ($\beta_{0i,t}$), internal influence ($\beta_{1i,t}$) and repeat rate ($\beta_{3i,t}$)- and the mean of the log of expenditures on direct-to-consumer advertising (DTC), direct-to-physician marketing (DTP) and aggregated marketing (MK). [Drug No 1 – rhinitis category]

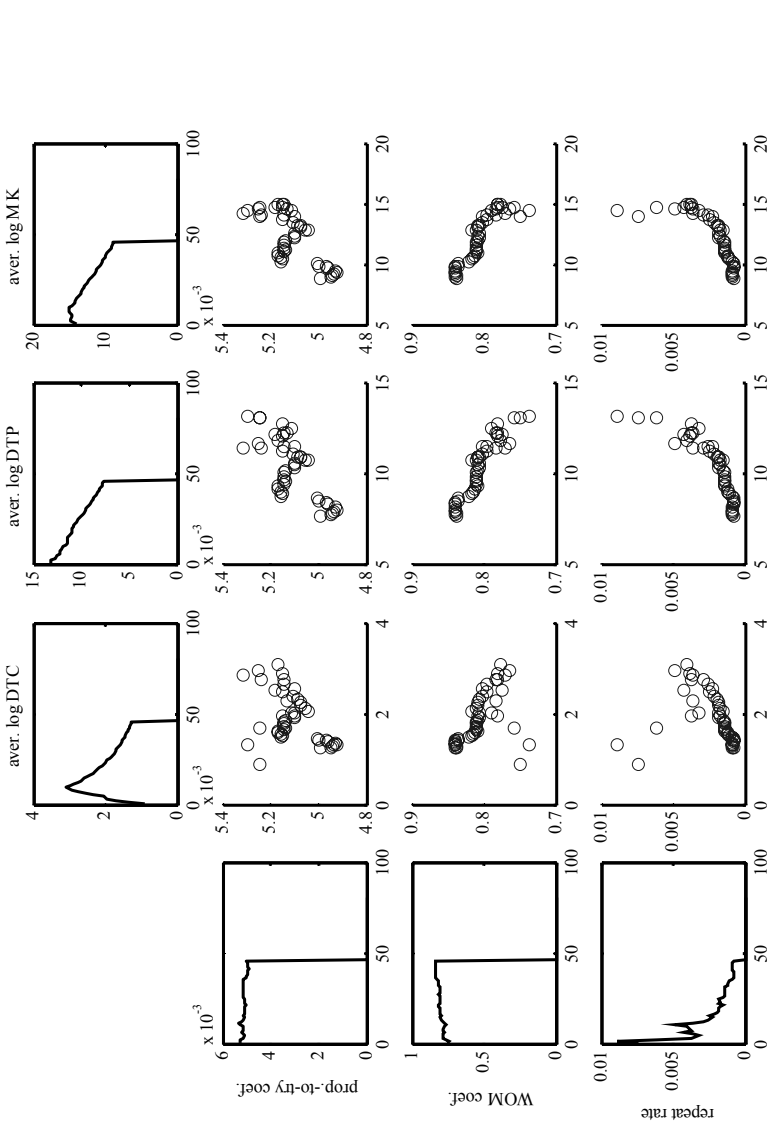


Figure 6B.6. Time-varying diffusion parameters –basic propensity to try ($\beta_{10i,t}$), internal influence ($\beta_{2i,t}$) and repeat rate ($\beta_{3i,t}$)- and the mean of the log of expenditures on direct-to-consumer advertising (DTC), direct-to-physician marketing (DTP) and aggregated marketing (MK). [Drug No 4 – rhinitis category]

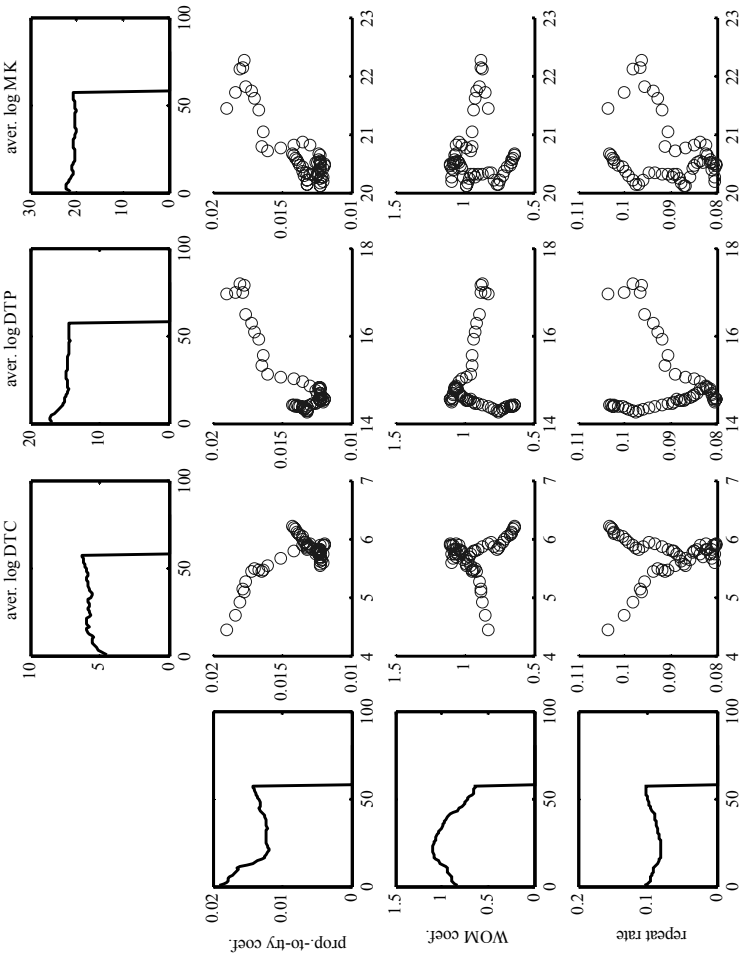


Figure 6B.7. Time-varying diffusion parameters –basic propensity to try ($\beta_{10i,t}$), internal influence ($\beta_{2i,t}$) and repeat rate ($\beta_{3i,t}$)- and the mean of the log of expenditures on direct-to-consumer advertising (DTC), direct-to-physician marketing (DTP) and aggregated marketing (MK). [Drug No 9 – rhinitis category]

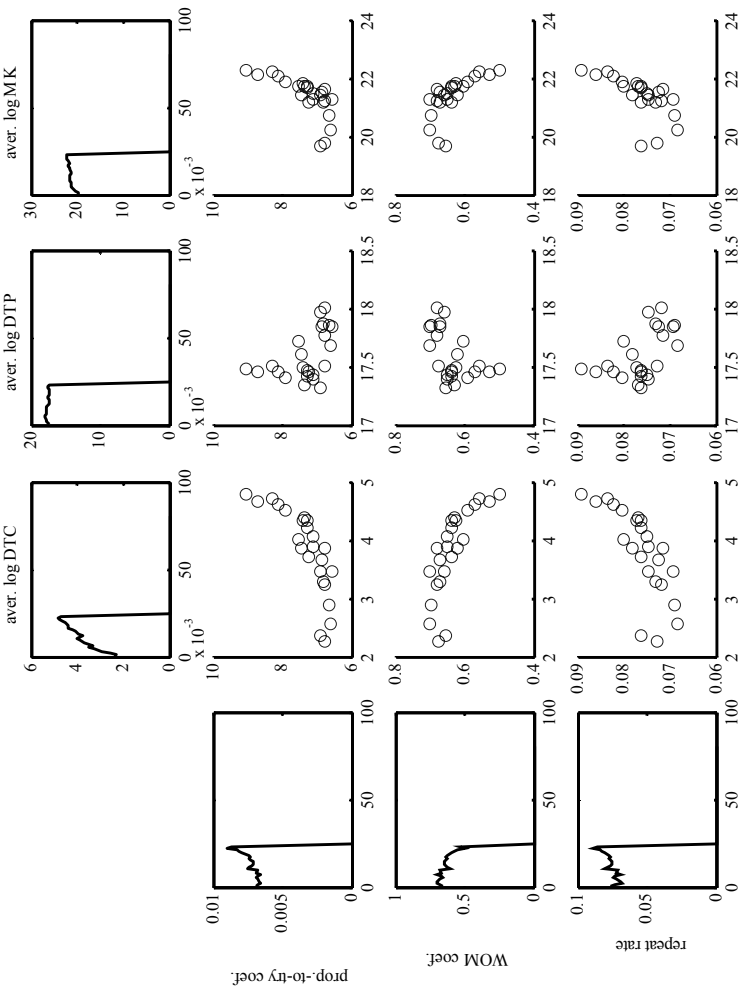


Figure 6B.8. Time-varying diffusion parameters –basic propensity to try ($\beta_{0i,t}$), internal influence ($\beta_{1,t}$) and repeat rate ($\beta_{3i,t}$)- and the mean of the log of expenditures on direct-to-consumer advertising (DTC), direct-to-physician marketing (DTP) and aggregated marketing (MK). [Drug No 12 – rhinitis category]