

# A Dynamic Competitive Forecasting Model Incorporating Dyadic Decision Making

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New products are often launched sequentially, by different firms, and the purchasing decisions are sometimes made by dyads. This paper proposes a new model that explicitly considers dyadic decision making in drug prescription and allows assessment of the relative influence that physicians and patients have in making decisions concerning new as well as existing ethical drugs. Modeling sequentially launched competing products in a category allows for parsing out effects that are hard to differentiate in models designed to capture only a single product's dynamics. The proposed model is applied to prescription drug data sets in the pharmaceutical industry, and it also explicitly captures both physicians' and patients' pretrial and posttrial utilities of each drug in the therapeutic category. Based on the model's fit and out-of-sample forecasting performance, we find that, in many cases, the incorporation of the dyadic decision making leads to better performance vis-à-vis models where such decision making is not explicitly considered. We also find that in many cases the posttrial utility of a drug is greater than its corresponding pretrial utility, lending partial empirical support to the prevailing industry practice of spending on various activities (e.g., sampling to physicians) needed to get potential patients to try a new drug. The proposed model enables managers to predict in advance the sales of sequentially launched new drugs and plan the new product launch and strategy accordingly. The model is also applicable to other product categories involving more than a single decision maker, including business-to-business products (e.g., office equipment) as well as to products targeting children (e.g., toys).

*Key words:* forecasting; pharmaceutical marketing; dyadic decision making; dynamic competitive models; trial-repeat models; new products

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## 1. Introduction

Understanding and forecasting the dynamics of competing products' sales within an evolving category has been a challenge for marketing scientists and practitioners. Consider, for example, a new class of anti-arthritis drugs called COX-2 blockers. Unlike traditional anti-arthritis drugs (nonsteroidal antiinflammatory drugs), COX-2 blockers selectively target the COX-2 enzyme (bad enzyme) without affecting the function of COX-1 enzyme (good enzyme). As a result, COX-2 reduces symptoms of arthritis with minimal side effects. The market outlook for COX-2 blockers was extremely attractive (nonsteroidal antiinflammatory drugs had \$5.5 billion in worldwide sales in 1996). By the end of 1998, Searle, Merck, Johnson & Johnson, GlaxoWellcome, Abbott, and Roche all had COX-2 blocker candidates at various stages in their new-product development pipelines. Each drug candidate had a slightly different profile (mostly with regard to side effects) and was expected to enter

the market at a different date. Searle launched the first COX-2 blocker, Celebrex, in January 1999. Merck followed with Vioxx<sup>1</sup> in June 1999. The third entry, Bextra, was approved by the Food and Drug Administration (FDA) on November 21, 2001, and is marketed by Pharmacia and Pfizer. These sequential entries led managers to revise accordingly their future market shares and sales estimates.

Market shares and competitive market structure have had a long research tradition in marketing/management science and economics (Cooper 1993). Models have been developed aggregately and disaggregately. To date, existing disaggregate (utility-based) market share models (e.g., multinomial logit (MNL)) typically assume individual utility and choice probability (McFadden 1981, Cooper 1993, Sudhir 2001, Ofek and Srinivasan 2002). However, there are

<sup>1</sup> Vioxx was later found to have serious side effects at high dosage and was withdrawn voluntarily by Merck.

situations where two parties are involved in the decision making (e.g., household purchase, business to business). Hence, dyadic decision-making formulation is warranted.

Recent evidence from the pharmaceutical industry suggests that the actual prescription decision is increasingly becoming a result of joint decision making between physicians and patients. An article in the *Wall Street Journal* (O'Connell 2002) reported an FDA study indicating that on average "nearly one quarter of people who saw a doctor recently asked the physician for a specific brand of medicine. Sixty-nine percent of the patients who asked for a specific brand ultimately received a prescription for the brand they requested." In another recent FDA survey (Aikin et al. 2004), 81% of the patients reported exposure to some type of direct-to-consumer advertisement (DTC) in the past three months, and 43% of the patients indicated that such exposure led them to actively seek out information about either the drug or the medical condition. Some of the patients (13%) have gone further and asked their physicians about a specific brand of prescription drug, and half of these patients also reported that the physicians prescribed the drug they had asked about. Patients' active role is corroborated in a parallel FDA survey of 500 physicians (250 primary care physicians and 250 specialists). At least 86% of these physicians recalled patients asking about a specific prescription drug. Primary care physicians, however, were both more likely to receive a request for prescription (65%) than specialists (52%) and more likely to comply (64% versus 46%). This somewhat surprisingly high percentage of compliance by physicians should not be interpreted as physicians being irresponsible. After all, all prescription drugs have undergone rigorous clinical tests and must pass the FDA's evaluation. We do not contend that *all* prescription drug decisions are and should be made jointly. There are, of course, complicated medical problems such as schizophrenia where the decision is, and should, be made solely by the expert—the physician. In less complex situations, however, incorporating the patient's preference may have a desirable impact because of the positive psychological effect on the patient who uses drugs she or he believes in and for which she or he may have a higher rate of compliance. It is also likely that the physician has already had some experience with these drugs with other patients, and, hence, feels comfortable in prescribing them. The degree to which physicians accommodate their patients' preference for a prescription drug may vary, however, not only by the therapeutic area but also by the physician's specialty (e.g., general practitioner, family doctor, and specialist). This issue is of particular interest to our study because of its diagnostic targeting implications. Hence, recognizing

such physicians' (discrete) heterogeneity, the model developed here will be applied separately to various segments of physicians. Despite previous studies that have utilized pharmaceutical data (see Rao and Yamada 1988; Gatignon et al. 1990; Shankar 1999; Shankar et al. 1998, 1999), none of them has looked at the notion of dyadic decision making and the relative influence on prescription behavior. This is a focal issue in our paper.

The objective of this study is to develop a new dynamic model that is capable of addressing the emerging dyadic physician–patient interactions over time for sequentially launched new drugs. The model's development begins with a Markovian framework that classifies patients, differently from traditional models, into a discrete number of usage (or nonusage) states. We model the various conditional transition probabilities further based on the physician/patient dyadic utilities, which may vary depending on the particular state of the patient. We then derive unconditional dynamic probabilities of prescribing (or not prescribing) various competing drugs in a therapeutic category. We also show how managerial statistics such as the drugs' market shares and their corresponding sales volumes can be derived and decomposed into trial and repeat usage components and how they compare to other known models.

In six empirical applications (two drug classes: proton pump inhibitors (PPI) and statins  $\times$  three physician segments: general practitioners/family practitioners/doctors of osteopathic medicine, internal medicine, specialists), we find, with the exception of specialists segments, that the dyadic decision-making version of the model outperforms its nested single decision-making version in out-of-sample forecasting. It also performs considerably better than the state-of-the-art benchmark model (Hahn et al. 1994) in out-of-sample forecasting. The posttrial utility of a drug is found to be greater than its corresponding pretrial utility in most cases, lending partial empirical support to the prevailing industry practice of spending on various activities that encourage trial (e.g., sampling to physicians). We also find substantial difference between the Specialists segment and the other two segments. In particular, patients appear to play a lesser role in the prescription decision when the doctor is a specialist. This insight corroborates the evidence from the FDA survey cited earlier. Finally, it appears that the prescription decision involving specialists treating patients with severe symptoms is qualitatively different from other cases. In this situation, physicians are not influenced by marketing activities, and patients have little influence over the prescription decisions.

This paper is organized as follows: Section 2 reviews the relevant literature, §3 presents the model

development, §4 describes empirical applications of the model, and §5 summarizes the paper and provides suggestions for further research.

## 2. Relevant Literature

In developing our new model we rely on, and synthesize, insights and analytics from pharmaceutical marketing research and models of joint decision making.

There is a large body of literature on diffusion that explicitly models trial (e.g., Parker and Gatignon 1994, 1996) or trial and repeat (e.g., Shankar et al. 1998, 1999). The literature that includes trial and repeat usage of a new prescription drug is particularly relevant to this paper. Because the forecasting model proposed here uses aggregate data, we refer to the only three existing (to our knowledge) trial and repeat models that have also been calibrated on aggregate data from the pharmaceutical industry: Lilien, Rao, and Kalish 1981 (LRK), Mahajan, Wind, and Sharma 1983 (MWS), and Hahn et al. 1994 (HPKZ). These three models are noncompetitive, focusing on a single product of interest. The LRK model is based on a four-segment trial and repeat framework, but in its empirical application the model was simplified to two segments: prescribers and nonprescribers. The MWS model is based on a two-segment (prescribers and nonprescribers) framework. The HPKZ model is a four-segment trial-repeat model. The four segments considered in this model are nontriers, triers, posttrial nonrepeaters, and posttrial repeaters. In its empirical application with pharmaceutical data, however, the four-segment model was simplified through the use of an average stationary repeat usage rate (thus no longer differentiating among triers, nonrepeaters, and repeaters). Moreover, the HPKZ model focuses on analyzing a single (focal) prescription drug. Hence, it is not a truly competitive model. In Online Appendix 2 (provided in the e-companion)<sup>2</sup> we provide a detailed discussion concerning differences in mathematical structures and models' properties between our new model (A2.17 and A2.18) vis-à-vis the LRK, MWS (A2.25), and HPKZ (A2.26) models, as well as the competitive Lanchester model (Kimball 1957, A2.23 and A2.24). Also related to our model is the family of macro-flow models exemplified by Hauser and Wisniewski (1982) and models with one late entrant (e.g., Krishnan et al. 2000).

Another important research stream that inspires our work addresses models of joint decision making. While most, if not all, marketing choice models have been based on individual preference and decision-making processes, many purchasing/usage decisions

are made by more than a single individual. Indiscriminant applications of individual choice models to products purchased by more than one person may lead to erroneous conclusions. One such example is repeat buying in households (see Corfman and Gupta 1993 for a detailed discussion). In this context, differences in variety-seeking and reinforcement behavior are not accounted for when the household is treated as a single decision-making unit (Givon 1984; Kahn et al. 1986a, 1986b). To capture the joint decision making at the utility (preference) level, various mechanisms have been proposed, axiomatized, and reported in the literature. The formulation of a group utility function that has been applied most extensively is the additive. Under this formulation, the joint (group) utility function is specified as a weighted sum of the individual members' utilities where the weights represent the relative influence of each member on the group's collective decision (Keeney and Raiffa 1976, Eliashberg and Winkler 1981). Applications of the group utility in modeling, econometrically, the group choice probability are limited, however. One notable exception is Gliebe and Koppelman (2002, 2005), who have applied the framework in the context of household members' joint activity participation.

In sum, the product category on which this paper focuses, prescription drugs, covers three important modeling challenges. First, prescription drugs are frequently purchased and used (especially for patients with chronic medical conditions). Second, the prescription decision may be heavily influenced by the patients' preference for, and experiences with, various drugs. Third, there are usually several sequentially launched drugs in a given class of drugs, and they compete closely against each other.

## 3. Model Development

To illustrate the modeling process, we begin with an expository discussion of a two-drug case, drugs A and B; both drugs belong to the same therapeutic class (e.g., statins for cholesterol reduction). Next we discuss how the model can be expanded to a  $K$ -drug category.

### 3.1. The Basic Model: Two-Drug Case

We formulate the basic model in three steps: (1) Whenever the patient is either prescribed a drug from the therapeutic class of interest (i.e., drugs A and B) or prescribed neither, his or her next-period state's membership is determined by a first-order Markovian process. (2) The unconditional future prescription probabilities and the resulting market shares are formulated. (3) The joint (dyadic) decision making made by the physician and the patient and the effect of the marketing variables are modeled in greater detail.

<sup>2</sup> An electronic companion to this paper is available as part of the online version that can be found at <http://mansci.journal.informs.org/>.

**Table 1** Transition Matrix  $\mathbf{P}_t$

Period $t - 1$	Period $t$			
	Nonuser (0, 0): $\pi_{(0,0)}(t)$	A-user (1, 0): $\pi_{(1,0)}(t)$	B-user (0, 1): $\pi_{(0,1)}(t)$	AB-user (1, 1): $\pi_{(1,1)}(t)$
Nonuser (0, 0): $\pi_{(0,0)}(t - 1)$	$p_{(0,0),t}^{(0,0)}$	$p_{(0,0),t}^{(1,0)}$	$p_{(0,0),t}^{(0,1)}$	0
A-user (1, 0): $\pi_{(1,0)}(t - 1)$	0	$p_{(1,0),t}^{(0,0)} + p_{(1,0),t}^{(1,0)}$	0	$p_{(1,0),t}^{(0,1)}$
B-user (0, 1): $\pi_{(0,1)}(t - 1)$	0	0	$p_{(0,1),t}^{(0,0)} + p_{(0,1),t}^{(0,1)}$	$p_{(0,1),t}^{(1,0)}$
AB-user (1, 1): $\pi_{(1,1)}(t - 1)$	0	0	0	$p_{(1,1),t}^{(0,0)} + p_{(1,1),t}^{(1,0)} + p_{(1,1),t}^{(0,1)} = 1$

**3.1.1. Modeling the Prescriptions of Drugs A and B Based on Transition Probabilities.** We define  $\{X_1, X_2, \dots\}$  as a sequence of a first-order Markovian random variable (Grimmett and Stirzaker 1992) that takes value in the states space  $S$ . Here,  $S$  is defined based on the patient’s prior experience with the drugs. That is, based on whether the patients have used the drug at all *up until* the end of the previous period. Such states definition is appropriate for products such as prescription drugs, which require test periods and where experience with the drug is the overwhelming driver in future treatments. The transition probabilities matrix that governs the Markovian process can expand as more new products enter the market. Our definition of the states allows us to separate clearly the trial from the repeat purchase.

We use the notation  $(I^A, I^B)$  to denote individual state  $(s)$ , where  $I^j$  ( $j = A, B$ ) is a dummy variable that takes the value of 0 or 1.  $I^j$  equals to 1 if a patient has used drug  $j$  before, otherwise 0. There are a total of four states. They are nonuser (has not used either drug A or B), A-user (has used A at least once but not B), B-user (has used B at least once but not A), and AB-user (has used both A and B), and they are denoted as (0, 0), (1, 0), (0, 1), and (1, 1), respectively.

The (nonstationary) transition matrix  $\mathbf{P}_t$  for the first-order Markov process is defined in Table 1. Note that the conditional probabilities represent the patients’ various one-period transitions based on the *dyadic* prescription decision making. At each period there are three options that the dyad (patient/physician) has to choose from: no drug from the class of interest, drug A, or drug B. We use the same notation to denote the prescription decision in period  $t$ , where  $P_t^{(0,0)}$ ,  $P_t^{(1,0)}$ , and  $P_t^{(0,1)}$  denote the unconditional probability of prescribing, in period  $t$ , no drug from the class, drug A, or drug B, respectively. To clarify, the binary notation (0, 0), (1, 0), (0, 1) denotes neither drug, drug A, drug B, respectively, in the context of prescribing one of the three options as well as in describing the patient’s state: nonuser, A-user, and

B-user, respectively.<sup>3</sup> The  $p_{s,t}^n$  in the transition matrix  $\mathbf{P}_t$  denotes the (dyadic) conditional probability that a patient in state  $s$  ((0, 0), (1, 0), (0, 1), (1, 1)) in period  $t - 1$  is prescribed option  $n$  ( $n =$  neither, A, or B) in period  $t$ .

Let  $\pi_s(t)$  denote the unconditional probability of a patient being in state  $s$  in time period  $t$ , where  $t = 0$  is the time period immediately before the first drug in the class was launched. That is,

$$\begin{aligned} \pi_{(0,0)}(0) &= 1 \quad \text{and} \\ \pi_{(1,0)}(0) &= \pi_{(0,1)}(0) = \pi_{(1,1)}(0) = 0. \end{aligned} \tag{1}$$

The unconditional state membership probabilities can be written as

$$\begin{aligned} &[\pi_{(0,0)}(t) \quad \pi_{(1,0)}(t) \quad \pi_{(0,1)}(t) \quad \pi_{(1,1)}(t)] \\ &= [1 \quad 0 \quad 0 \quad 0] \left[ \prod_{i=1}^t \mathbf{P}_i \right]. \end{aligned} \tag{2}$$

One of the challenges in empirical analyses is when the researchers do not have the data starting from the first in the class launching date. To apply our model in such situations, the patients’ proportion of memberships in the first period when the data become available,  $t_0$ , must be estimated. Explicit expressions for the states membership probabilities ( $\pi_s(t)$ ) at time  $t$  as functions of their initial conditions can be obtained (see Online Appendix 1, provided in the e-companion). The unconditional prescription probabilities,  $P_t^{(0,0)}$ ,  $P_t^{(1,0)}$ , and  $P_t^{(0,1)}$ , are needed for the model estimation. (See derivation in Online Appendix 1.) They can be rewritten to highlight relevant managerial metrics. Specifically, for the two drugs of interest (A and B),

$$\begin{aligned} P_t^{(1,0)} &= \underbrace{\pi_{(0,0)}(t-1)p_{(0,0),t}^{(1,0)} + \pi_{(0,1)}(t-1)p_{(0,1),t}^{(1,0)}}_{\text{trial}} \\ &+ \underbrace{\pi_{(1,0)}(t-1)p_{(1,0),t}^{(1,0)} + \pi_{(1,1)}(t-1)p_{(1,1),t}^{(1,0)}}_{\text{repeat}}, \end{aligned} \tag{3}$$

<sup>3</sup> We thank an anonymous reviewer for suggesting this notation.

$$P_t^{(0,1)} = \underbrace{\pi_{(0,0)}(t-1)p_{(0,0),t}^{(0,1)} + \pi_{(1,0)}(t-1)p_{(1,0),t}^{(0,1)}}_{\text{trial}} + \underbrace{\pi_{(0,1)}(t-1)p_{(0,1),t}^{(0,1)} + \pi_{(1,1)}(t-1)p_{(1,1),t}^{(0,1)}}_{\text{repeat}}, \quad (4)$$

$$P_t^{(0,0)} = 1 - P_t^{(1,0)} - P_t^{(0,1)}. \quad (5)$$

The market shares of drugs A and B are given, respectively, by

$$MS_t^{(1,0)} = \frac{P_t^{(1,0)}}{P_t^{(1,0)} + P_t^{(0,1)}} \quad \text{and} \quad (6)$$

$$MS_t^{(0,1)} = \frac{P_t^{(0,1)}}{P_t^{(1,0)} + P_t^{(0,1)}}.$$

**3.1.2. Modeling Further the Transition Probabilities Based on Dyadic Decision Making and Random Utility Theory.** Note that, up to this point, the unconditional prescription probabilities, used later to construct the likelihood function for parameters estimation, and their implied managerial metrics are all functions of the transition probabilities, which in turn are functions of the conditional prescription probabilities. The next step is therefore to model further these probabilities. We follow previous research (e.g., Zufryden 1986) in which the first-order Markovian transition probabilities were modeled via utilities and multinomial logit formulation. We depart from this literature by formulating dyadic utilities as opposed to individual utilities on which the previous literature has been based. This is accomplished in a three-step process, similar to that employed, for instance, by Gliebe and Koppelman (2002, 2005): (1) specification of the individual (physician and patient) utilities as functions of covariates; (2) specification of the systematic component of the dyadic utility functions; and (3) specification of the error term in the dyadic utility and derivation of random utility-based transition (choice) probabilities.

We adopt an exponential utility formulation, where the individual utility functions for the physician and patient for option  $n$  are specified as follows:

$$V_{m,s,t}^n(x_{m,t}^n) = e^{\alpha_{m,s,t}^n + \varphi_m^n x_{m,t}^n}, \quad (7)$$

$$V_{p,s,t}^n(x_{p,t}^n) = e^{\alpha_{p,s,t}^n}. \quad (8)$$

$V_{m,s,t}^n$  and  $V_{p,s,t}^n$  denote the physician and patient's individual utilities in period  $t$ , respectively, for option  $n$  ( $n \in \{0, A, B\}$ ), given that the patient is in state  $s$  in period  $t-1$ .  $x_{m,t}^k$  denotes the marketing activities for drug  $k$  (A or B) directed toward the physician at time  $t$ , and  $x_{m,t}^0 = 0$ . The utilities have intercepts  $a_{m,s,t}^n$  and  $a_{p,s,t}^n$ .

We next invoke an additive formulation for the systematic part of the dyadic utility function (see p. 260

in Curry et al. 1991; see also Harsanyi 1955, Keeney and Raiffa 1976, Dyer and Sarin 1979, Kirkwood and Sarin 1980, Eliashberg and Winkler 1981) but with a relative influence parameter ( $\lambda_s$ ) that depends on the state the patient is in. The specification is

$$V_{s,t}^n(x_t^n) = V_{m,s,t}^n(x_{m,t}^n) + \lambda_s V_{p,s,t}^n(x_{p,t}^n) \\ = e^{\alpha_{m,s,t}^n + \varphi_m^n x_{m,t}^n} + \lambda_s e^{\alpha_{p,s,t}^n}. \quad (9)$$

The dyadic utility ( $U_{s,t}^n(x_t^n)$ ) for a given option  $n$  is assumed to have a systematic component ( $V_{s,t}^n(x_t^n)$ ) and a multiplicative error term,  $e^{\varepsilon_t^n}$ , where  $\varepsilon_t^n$  is assumed to be independently and identically distributed according to type I extreme value distribution. That is,

$$U_{s,t}^n(x_t^n) = V_{s,t}^n(x_t^n) e^{\varepsilon_t^n}. \quad (10)$$

The multiplicative utility can then be converted to additive format by taking log on both sides:

$$\ln[U_{s,t}^n(x_t^n)] = \ln[V_{s,t}^n(x_t^n)] + \varepsilon_t^n. \quad (11)$$

A standard MNL choice probability model can now be derived accordingly for the choice set ( $N = \{0, A, B\}$ ):

$$p_{s,t}^n = e^{\ln V_{s,t}^n(x_t^n)} / \sum_{d=0}^N (e^{\ln V_{s,t}^d(x_t^d)}). \quad (12)$$

Substituting in (12) the systematic dyad utility component from (9) and simplifying, the following choice probability expression is obtained:

$$p_{s,t}^n = (e^{\alpha_{m,s,t}^n + \varphi_m^n x_{m,t}^n} + \lambda_s e^{\alpha_{p,s,t}^n}) / \sum_{d=0}^N (e^{\alpha_{m,s,t}^d + \varphi_m^d x_{m,t}^d} + \lambda_s e^{\alpha_{p,s,t}^d}). \quad (13)$$

Note that, without the dyadic decision making, the probability of a drug being prescribed can be simply obtained from the physician's utility (i.e., setting  $\lambda_s = 0$ ). This scenario is called Model 1. We refer to the scenario with dyadic decision making as Model 2. Table 2 summarizes the model's notations for the two-drug case.

### 3.2. The General Model: $K$ -Drug Case

The general model is conceptually similar to the basic two-drug case, and the market share equations can be obtained accordingly, but computations are much more involving. Because a patient can either have tried or not tried a drug by a given time period, the number of states for a class of  $K$  drugs is  $2^K$ . The structure is the same as in the two-drug case except that the transition matrix  $\mathbf{P}_t$  is now  $2^K \times 2^K$ . The operationalization of prescription probabilities is similar to the two-drug case, except that now the choice set is  $K$  drugs plus the option of prescribing no drug from the

**Table 2** Summary of Notations Used in the Two-Drug Case

Notation	Interpretation
$k$	A given drug in the class. In the two-drug case, $k$ could be (1, 0) or (0, 1), representing drug A or B, respectively.
$n$	A decision option. In the two-drug case, $n$ could be (0, 0), (1, 0), or (0, 1), representing prescribing neither A nor B, prescribing drug A, and prescribing drug B, respectively.
$t$	Time period. $t = 1$ is the period the first drug in the class was launched.
$t_0$	The first time period when the data become available.
$s$ , denoted by $(I^A, I^B)$	An individual state, where $I^j$ ( $j = A, B$ ) is a dummy variable that takes the value of 0 (if drug $j$ has not been used before) or 1 (if used before).
$p_{s,t}^n$	The (dyadic) probability that a patient, in state $s$ in period $t - 1$ , will be prescribed option $n$ ( $n = 0, A$ , or $B$ ) in the class of interest in period $t$ .
$P_t^n$	The unconditional (dyadic) probability of taking option $n$ in time period $t$ .
$\pi_s(t)$	The unconditional probability of a patient being in state $s$ in a future time period $t$ .
$P_t$	Transition matrix.
$MS_t^{(1,0)}, MS_t^{(0,1)}$	Market share of drug A or B, respectively, in time period $t$ .
$V_{m,s,t}^n, V_{p,s,t}^n, V_{s,t}^n$	Physician, patient, and dyadic <i>systematic</i> utilities for choosing option $n$ for a patient in state $s$ in time period $t - 1$ , respectively.
$U_{m,s,t}^n, U_{p,s,t}^n, U_{s,t}^n$	Physician, patient, and dyadic utilities for choosing option $n$ for a patient in state $s$ in time period $t - 1$ , respectively.
$a_{m,s,t}^n$	Physician's utility parameter (intercept) in time period $t$ , for prescribing $n$ for a patient in state $s$ .
$a_{p,s,t}^n$	Patient's utility parameter (intercept) in time period $t$ , for prescribing $n$ when the patient is in state $s$ .
$x_{m,t}^k$	Marketing activities toward physicians in time period $t$ related to drug $k$ ( $k = A, B$ ).
$\phi^k$	Coefficient of the marketing activities $x_t^k$ .
$\lambda_s$	Decision weight for patients in state $s$ , used to construct the dyadic utility of the patient and physician. $\lambda_s = 0$ for all $s$ represent an individual decision-making model (Model 1); otherwise they represent a dyadic decision-making model (Model 2).

focal therapeutical class. The dyadic decision-making formulation remains the same as in the basic model. As a result, the prescription probability of selecting option  $n$  ( $n$  denotes either prescribing a drug or not prescribing any from the class of interest) in time period  $t$  when a patient is in state  $s$  in time period  $t - 1$  is identical to that specified for the two-drug case, except now  $N$  is the entire choice set for  $K$  drugs. Again, it can be reduced to the single decision-maker model (Model 1) by setting  $\lambda_s$  to 0.

## 4. Empirical Applications

### 4.1. Likelihood Function

Similar to Pakes (1986) and Song and Chintagunta (2003), the log likelihood function of the observed aggregate data is given by

$$\ln(L) = \sum_{t=t_0}^{t_c} \sum_{n=0}^N [S_t^n \ln(P_t^n)]. \quad (14)$$

$N$  is total number of drugs in a class plus 1 (nonprescription outcome),  $t_c$  is the last time period of data used for calibration, and  $S_t^n$  is total number of patients who were given option  $n$  in period  $t$ .  $P_t^n$  is given in Equations (3)–(5) for the two-drug case, and expressions for  $K$ -drug case are similar.

### 4.2. Parameters Constraints

Theory-based constraints (restrictions) have been imposed on the parameters of models for identification and parsimony (e.g., McGuire and Staelin 1983).

A few additional constraints are imposed here either because of data limitation or because empirical applications suggest that more elaborate specifications do not lead to better predictive performance. In total, there are six key parameter constraints, discussed in detail below, using the two-drug case for illustration.

The first constraint is imposed on the state-specific utilities. Unique state-specific utilities cannot be reliably estimated because of the limited nature of our data. For example, a three-drug class will require a total of 48 state-specific parameters.<sup>4</sup> We thus trade off the richness of the model to accommodate the limitations of the data. The major difference among patients in different states is their prior (or lack of) experience with drugs in the class. Accordingly, each member of the dyad (physician or patient) is characterized by two intercepts in the utility function for each drug  $k$  (A or B): pretrial intercept (if the patient in question has not used previously the drug) and posttrial intercept (if the patient in question has used previously the drug). These intercepts will be designated as  $a_{m,pre}^k, a_{m,post}^k, a_{p,pre}^k$ , and  $a_{p,post}^k$ , respectively. Empirically, a simpler model where the pretrial and posttrial utilities were set to be the same was tested and rejected on the basis of poor fit and prediction.

<sup>4</sup> State-specific utilities can theoretically be estimated with a richer data set with a limited number of states. However, a richer data set usually implies a larger number of drugs in the data set (a plus), which also means a larger number of states (a minus). It is conceivable that a data set with many more periods of observations in a dynamic market (varying marketing activities, etc.) will help.

**Table 3** Operationalizing the Systematic Utilities for the Two-Drug Case

Physician	Utility for prescribing no drug, $V_{m,s,t}^{(0,0)}$	Utility for prescribing drug A, $V_{m,s,t}^{(1,0)}$	Utility for prescribing drug B, $V_{m,s,t}^{(0,1)}$
Nonusers, (0, 0)	$V_{m,s,t}^{(0,0)} = 1$	$V_{m,(0,0),t}^{(1,0)} = e^{b_{m,pre}^{(1,0)} + \varphi} x_t^{(1,0)}$	$V_{m,(0,0),t}^{(0,1)} = e^{b_{m,pre}^{(0,1)} + \varphi} x_t^{(0,1)}$
A-users, (1, 0)	$V_{m,s,t}^{(0,0)} = 1$	$V_{m,(1,0),t}^{(1,0)} = e^{b_{m,post}^{(1,0)}}$	$V_{m,(1,0),t}^{(0,1)} = e^{b_{m,pre}^{(0,1)} + \varphi} x_t^{(0,1)}$
B-users, (0, 1)	$V_{m,s,t}^{(0,0)} = 1$	$V_{m,(0,1),t}^{(1,0)} = e^{b_{m,pre}^{(1,0)} + \varphi} x_t^{(1,0)}$	$V_{m,(0,1),t}^{(0,1)} = e^{b_{m,post}^{(0,1)}}$
AB-users, (1, 1)	$V_{m,s,t}^{(0,0)} = 1$	$V_{m,(1,1),t}^{(1,0)} = e^{b_{m,post}^{(1,0)}}$	$V_{m,(1,1),t}^{(0,1)} = e^{b_{m,post}^{(0,1)}}$
Patient	Utility for prescribing no drug, $V_{p,s,t}^{(0,0)}$	Utility for prescribing drug A, $V_{p,s,t}^{(1,0)}$	Utility for prescribing drug B, $V_{p,s,t}^{(0,1)}$
Nonusers, (0, 0)	$V_{p,s,t}^{(0,0)} = 1$	$V_{p,(0,0),t}^{(1,0)} = e^{b_{p,pre}^{(1,0)}}$	$V_{p,(0,0),t}^{(0,1)} = e^{b_{p,pre}^{(0,1)}}$
A-users, (1, 0)	$V_{p,s,t}^{(0,0)} = 1$	$V_{p,(1,0),t}^{(1,0)} = e^{b_{p,post}^{(1,0)}}$	$V_{p,(1,0),t}^{(0,1)} = e^{b_{p,pre}^{(0,1)}}$
B-users, (0, 1)	$V_{p,s,t}^{(0,0)} = 1$	$V_{p,(0,1),t}^{(1,0)} = e^{b_{p,pre}^{(1,0)}}$	$V_{p,(0,1),t}^{(0,1)} = e^{b_{p,post}^{(0,1)}}$
AB-users, (1, 1)	$V_{p,s,t}^{(0,0)} = 1$	$V_{p,(1,1),t}^{(1,0)} = e^{b_{p,post}^{(1,0)}}$	$V_{p,(1,1),t}^{(0,1)} = e^{b_{p,post}^{(0,1)}}$

As a result, for the two-drug case, the twelve theoretically possible intercepts (three options in four states) associated with the physician's utility are reduced to five intercepts; and similarly twelve theoretically possible intercepts associated with the patient's utility are reduced to five intercepts.

The second constraint, normalization, imposed for identification purposes, is consistent with the extant literature and is operationalized by setting the intercepts of both physician's and patient's utility associated with the option of receiving no drug,  $V_{m,s,t}^{(0,0)}$  and  $V_{p,s,t}^{(0,0)}$ , equal to 0. This is equivalent to normalizing all utilities relative to the utility of the no-drug prescription option (e.g., Chintagunta et al. 1991). To differentiate the normalized intercepts from those in the nonnormalized utilities, we will use the notation  $b_{m,pre}^k$ ,  $b_{m,post}^k$ ,  $b_{p,pre}^k$ , and  $b_{p,post}^k$  to represent the normalized pretrial and posttrial intercepts in the utilities of the physician and patient, respectively. The various parameters for each drug in each state are shown in Table 3.

The four utility parameters for a given drug (patient's pretrial and posttrial, physician's pretrial and posttrial) can be conceptually linked to relevant temporal covariates. For example, marketing activities directed at physicians, such as samples,<sup>5</sup> are likely to impact the physician's utility whereas DTC is likely

to affect the patient's utility. Following others (e.g., HPKZ 1994 discussed below in more detail), and employing data sets during time periods when DTC was not a prevalent activity, only the physician's pretrial utility parameters for each drug are modeled further. The marketing activities included in our data set (professional ad, detailing, and sampling) are aimed at inducing trial (of a new drug), not at repeat usage. It is widely accepted in the medical profession that, once a patient has been prescribed a drug, his or her utility for the drug will depend only on the specific reactions (efficacy, side effects) that this patient had to this drug. As a result, another restriction is imposed consistent with this practice.

The next two constraints are taking the parameter  $\varphi$  to be class specific rather than drug specific, and consistent with Hahn et al. (1994) we employ a composite covariate (total marketing expenditure) instead of three separate covariates (ad, detailing, and sampling). These constraints are imposed for parsimony purposes. Both constraints were tested against non-constrained specifications, and it was found that they did not lead to deterioration in terms of fit or in terms of predictions (see Table 3).

Finally, different versions of models with different degrees of constraints imposed on the relative decision influence parameters were empirically estimated. The results are quite comparable (in parameter estimates, fit, and forecasting performance), and only one specification is reported here (see Online Appendix 4, provided in the e-companion, for results of an alternative specification). Theoretically, the dyadic decision making can be contingent on the patient's prior experience with drugs in a class or, equivalently, on

<sup>5</sup> Unlike many other product categories, in prescription drugs samples are designed for trial only (each sample has limited supply and is much smaller than that contained in a regular prescription) and constrained by the gatekeeper (physicians are trained and morally bound to use sample for patients who have not used that drug before).

**Table 4** Parameters that Need to be Estimated in the Two-Drug Model

Parameter	Appears in	Interpretation
$b_{m,pre}^k$	Models 1 and 2	Normalized pretrial utility parameter of physician for drug $k$
$b_{m,inc}^k$	Models 1 and 2	Normalized incremental posttrial utility parameter of physician for drug $k$
$\varphi$	Models 1 and 2	Coefficient for class-level total marketing expenditure
$\pi_{(0,0)}(t_0)$	Models 1 and 2	Proportion of patients who are in state (0, 0) at the first period when the data become available
$b_{p,pre}^k$	Model 2 only	Normalized pretrial utility parameter of patient for drug $k$
$b_{p,inc}^k$	Model 2 only	Normalized incremental posttrial utility parameter of patient for drug $k$
$\lambda_1$	Model 2 only	Relative influence parameter, when patient has no experience (i.e., used no drug in the class)
$\lambda_2$	Model 2 only	Relative influence parameter, when patient has partial experience (i.e., used either one of the two drugs in the class)
$\lambda_3$	Model 2 only	Relative influence parameter, when patient has complete experience (i.e., used both drugs in the class)

any particular state the patient is in. In the specification presented here, three decision-making dyads are explicitly recognized: those with patients who have not experienced any drugs in the class (and thus have no information), those with patients who have experienced some (but not all) drugs in the class (and thus have partial information), and those with patients who have already experienced all of the drugs in the class (and thus have complete information about the class).

Mathematically,

$$\lambda_s = \begin{cases} \lambda_1 & \text{if } s = (0, 0), \\ \lambda_2 & \text{if } s = (1, 0) \text{ or } (0, 1), \\ \lambda_3 & \text{if } s = (1, 1). \end{cases} \quad (15)$$

The corresponding equation for Model 1 can be obtained by setting  $\lambda_1 = \lambda_2 = \lambda_3 = 0$ .

Given the constraints discussed above, we can now demonstrate the structure of the dyadic probability of prescribing drug A in time period  $t$ , given that the patient is in state (0, 1). (Other probabilities can be obtained in a similar fashion).<sup>6</sup>

$$P_{(0,1),t}^{(1,0)} = \frac{e^{b_{m,pre}^{(1,0)} + \varphi x_t^{(1,0)}} + \lambda_2 e^{b_{p,pre}^{(1,0)}}}{1 + e^{b_{m,pre}^{(1,0)} + \varphi x_t^{(1,0)}} + e^{b_{m,post}^{(0,1)}} + \lambda_2 (1 + e^{b_{p,pre}^{(1,0)}} + e^{b_{p,post}^{(0,1)}})}. \quad (16)$$

In the empirical implementation, instead of estimating the posttrial utility directly, we estimate the incremental posttrial utility (taking the pretrial utility as base). While theoretically equivalent, this new formulation facilitates the estimation process by providing a

meaningful initial value for the incremental posttrial utility (0). That is,

$$b_{m,post}^k = b_{m,pre}^k + b_{m,inc}^k \quad \text{and} \quad (17)$$

$$b_{p,post}^k = b_{p,pre}^k + b_{p,inc}^k. \quad (18)$$

Table 4 summarizes the parameters (and their interpretation) that need to be estimated for the two-drug model. Note that if the data become available after the launch of the first in the class drug, but prior to the launch of the second drug (as in our case), patients could be in only one of two states, (0, 0) or (1, 0). As a result, one needs to estimate only the initial proportion of patients in (0, 0),  $\pi_{(0,0)}(t_0)$  (where  $\pi_{(1,0)}(t_0) = 1 - \pi_{(0,0)}(t_0)$ ).

For a class with  $K$  drugs, for which the data are available from the launch of the first drug in the class (as in HPKZ), the total number of parameters that needs to be estimated is  $2K + 1$  for Model 1 and  $4K + 4$  for Model 2. For a left censored data set (e.g., launch of the first-in-class drug is not available), one needs to estimate the patients' states memberships at the first period when the data become available.<sup>7</sup>

### 4.3. Estimation Procedure and Identification

To estimate the model, the concentrated likelihood method (Seber and Wild 1989), which divides parameters set into two subsets and estimates them in a two-step procedure, has been employed. The key advantage of the concentrated likelihood method is its efficiency in reducing the number of parameters that need to be estimated simultaneously. Initial points and convergence are examined following the recommendation by Bates and Watts (1988).

Our model belongs to the stream of research where models are specified at the disaggregate level but

<sup>6</sup> Similarly, we can specify, for the general case of  $K$ -drug class, the probability of selecting option  $n$  ( $n$  denotes either one drug in the class or not using any drug from the class) in time period  $t$  when a patient is in state  $s$ .

<sup>7</sup> The current model can be potentially extended to accommodate continuous heterogeneity and additional trends if richer data are available. Although constrained by the data, we believe that this will be a fruitful direction for future research with appropriate data.

**Table 5** Description of the Data Sets

Drug class (names of competing drugs)	First and last date in data set	Number of data points for each drug <sup>a</sup>	Number of new entrants covered by the data window	Physician's specialty		
				GP/FP/DO <sup>b</sup>	IM <sup>c</sup>	Specialists <sup>d</sup>
Class 1, PPI (Prilosec, Prevacid)	December 1994–July 1999	50, 56	1	Data set 1	Data set 2	Data set 3
Class 2, statin (Lipitor, Baycol, Combo-4) <sup>e</sup>	January 1995–June 1999	18, 30, 54	2	Data set 4	Data set 5	Data set 6

<sup>a</sup>Because the model uses one period lag marketing activities, the first data point is not used in the estimation (the actual numbers are thus 50 and 55 for PPI and 18, 30, and 53 for statin).

<sup>b</sup>GP denotes general practitioners, FP denotes family practitioners, and DO stands for doctors of osteopathic medicine.

<sup>c</sup>IM denotes internal medicine practitioners.

<sup>d</sup>Specialists denote the specialists for a particular disease; they, in general, are different in different classes.

<sup>e</sup>There are a total of six drugs in the original statin data set. Four of the six drugs (Mevacor, Zocor, Prevacol, and Lescol) were already on the market prior to the first data point (January 1995) in the data set. As a result, we would have to estimate fifteen additional parameters (initial membership). This is certainly unrealistic. Instead, we chose to combine all four existing drugs into one single drug (Combo-4) and transform class 2 into a class with three drugs (one existing combination drug and two new drugs).

parameters are estimated using aggregate market-level data (e.g., Petrin 2002, Song and Chintagunta 2003). In our dyadic decision-making model, because only aggregate data are available, if the two decision participants always made decisions together and never with other partners, it would have been impossible to recover the individual decision influence from such data. It is possible, however, to recover the individual decision influence if the physician prescribes to different patients, while a patient could potentially see more than one physician, a fairly realistic assumption. Moreover, to ensure that the model can be identified, a simulation study was conducted (see also, e.g., Song and Chintagunta 2003). This is available in Online Appendix 3 (provided in the e-companion). The empirically estimated parameters (PPI, IM segment) were used as the “true” parameters. Data sets were generated by aggregating individual prescription choices over 100,000 simulated patients. A total of 50 such data sets were generated and estimated. Based on the mean and standard deviation of the estimates, the estimation approach appears to have recovered the individual-level parameters from the aggregate-level data fairly well.

#### 4.4. Data Sets

The data used for the empirical analyses are total number of prescriptions (TRx) for each drug in two different drug classes (proton pump inhibitors (PPI) and statin) prescribed by three different physician specialties (see Table 5). In total, there are six class  $\times$  specialty data sets. The data were provided by National Data Corporation (NDC) from its retail pharmacy audit database.

Both classes of drugs employed in our empirical analyses are used to treat chronic diseases, and they are commonly prescribed once per month for a one-month supply of the drug—a practice reinforced by

insurance companies. Previous research has also used a monthly time period as the time interval unit of analysis (e.g., HPKZ 1994).

Marketing activities data were collected from a major pharmaceutical firm that requested confidentiality. Consistent with previous research (Hahn et al. 1994), we employed a composite measure for the marketing activities: total marketing effort. It is the total spending on professional advertisement, contact, and sampling. We also invoke insights from previous literature (Lilien et al. 1981, Rao and Yamada 1988, Hahn et al. 1994) and employ the marketing activities lagged by one month.

The model was formulated in terms of shares of nonusers and users of various drugs. To use it with the TRx data, however, we need to know the total number of patients who have a particular disease and thus may potentially benefit from a certain drug treatment. This was obtained by using the total U.S. population as baseline (adjusted for population expansion in our data window) and the commonly accepted percentage of population afflicted with a certain disease. The true U.S. populations on May 1, 1990, and April 1, 2000, were obtained from U.S. Census data. The U.S. populations in each month between May 1, 1990, and April 1, 2000, were estimated, assuming that the population increases at a constant rate (similar to the official estimated growth rate by the U.S. government). Because ulcer is estimated to affect 5%–10% of the population (<http://www.astrazeneca.ie>), the median (7.5%) was taken to obtain the total patient population. The breakdown of the patients' population by physician type is based on the total prescriptions written for ulcer, which is 41%, 26%, and 28% for patients seeing general practitioners/family physicians, internists, and specialists, respectively (the remainder is prescribed by other health care

**Table 6** Class 1 (PPI) Drugs: Estimated Parameters

Parameters estimated	Notation	GP/FP/DO (GFD)		IM		SPE	
		M1	M2	M1	M2	M1	M2
<b>Drug A (Prilosec)</b>							
Physician							
1 Utility intercept before trial	$b_{m,pre}^A$	<b>-6.6972</b>	<b>-7.5249</b>	<b>-6.1632</b>	<b>-8.1724</b>	<b>-3.6644</b>	<b>-3.6389</b>
2 Incremental utility intercept	$b_{m,inc}^A$	<b>7.0455</b>	<b>8.1440</b>	<b>5.5923</b>	<b>9.4239</b>	<b>0.9880</b>	<b>0.9633</b>
Patients							
3 Utility intercept before trial	$b_{p,pre}^A$		<b>-6.2056</b>		<b>-4.9009</b>		0.0013
4 Incremental utility intercept	$b_{p,inc}^A$		<b>7.8732</b>		<b>7.4718</b>		0.0014
<b>Drug B (Prevacid)</b>							
Physician							
5 Utility intercept before trial	$b_{m,pre}^B$	<b>-7.2860</b>	<b>-6.9337</b>	<b>-7.1700</b>	<b>-7.0790</b>	<b>-7.0219</b>	<b>-7.0996</b>
6 Incremental utility intercept	$b_{m,inc}^B$	<b>9.9616</b>	<b>9.9976</b>	<b>10.0588</b>	<b>10.1681</b>	<b>9.9994</b>	<b>9.7799</b>
Patients							
7 Utility intercept before trial	$b_{p,pre}^B$		<b>-9.0678</b>		<b>-8.3512</b>		<i>-0.8563</i>
8 Incremental utility intercept	$b_{p,inc}^B$		<i>9.9942</i>		10.1144		<i>-0.0001</i>
Covariate							
9 Coefficient for marketing activities	$\varphi$	<b>2.8855</b>	<b>4.4484</b>	<b>4.6797</b>	<b>5.4232</b>	0.1926	0.0000
Initial membership (only nonusers estimated)							
10 Nonusers	$\pi_{(0,0)}(t_0)$	<b>0.9617</b>	<b>0.9717</b>	<b>0.8996</b>	<b>0.9555</b>	<b>0.8765</b>	<b>0.8765</b>
A-users	$\pi_{(1,0)}(t_0)$	<b>0.0383</b>	<b>0.0283</b>	<b>0.1004</b>	<b>0.0445</b>	<b>0.1235</b>	<b>0.1235</b>
Patient's relative decision influence (conditional on his or her experience with the category)							
11 No experience	$\lambda_1$		<b>0.8510</b>		<b>0.2623</b>		0.0000
12 Partial experience	$\lambda_2$		<b>0.9238</b>		<b>0.2847</b>		<b>0.0005</b>
13 Full experience	$\lambda_3$		<b>0.9958</b>		<b>0.8840</b>		<b>0.0077</b>
Total data points used for estimation (including nonuser)		142	142	142	142	142	142
Log likelihood		-115,788	-115,774	-99,921	-99,909	-72,358	-72,358
Akaike's information criterion (AIC)		-115,794	-115,787	-99,927	-99,922	-72,364	-72,371
Bayesian information criterion (BIC)		-115,802	-115,807	-99,936	-99,941	-72,373	-72,390

Notes. Bold indicates significant at 0.01, and italic indicates significant at 0.05. M1 represents Model 1, no dyadic decision, and M2 represents Model 2, with dyadic decision. The model estimates the incremental utility, for example,  $b_{m,inc}^A = b_{m,post}^A - b_{m,pre}^A$ .

providers). For the high cholesterol class it is estimated that 20% of the U.S. population could benefit from drug treatment (<http://www.lipitor.com>); similarly, 35.3%, 42.1%, and 19.9% of prescriptions are written by the three physician segments of interest and are used to represent the number of patients seeing each physician segment.

#### 4.5. Results

Both Models 1 and 2 were estimated based on all available data, except for the last six months in each data set, which were used as hold-out samples for the purpose of testing the models' forecasting performance. The results are presented in Tables 6 and 7. The MAD (mean absolute deviation) and APE (absolute percentage of errors) metrics for the fit and out-of-sample predictions are summarized in Table 8. We discuss below parameter estimates, fit, and out-of-sample forecasting performance.

**4.5.1. Parameters Estimates and Their Managerial Implications.** The empirical analyses led to a number of interesting diagnostic insights. We discuss and

interpret those related to utilities, marketing activities, and patients' decision-making influence. Finally, we highlight the differences across physician segments and therapeutic classes, and we make some conjectures concerning the generalizability of the empirical results.

*Utility Parameters.* First, note that the posttrial utility intercepts for a drug are, in general, higher than the pretrial. Out of a total of 45 incremental utility intercepts (fifteen pairs of physician's utilities estimated by Model 1; fifteen pairs of physician's utilities estimated by Model 2; and fifteen pairs of patient's utilities estimated by Model 2), 34 pairs exhibited this pattern (e.g.,  $b_{m,inc}^A = b_{m,post}^A - b_{m,pre}^A = 0.9633 > 0$  in Class 1, Prilosec, specialists segment, for Model 2 in Table 6). All differences for the remaining 11 cases are not significantly different from zero.

A related observation worth noting is that the incremental intercepts are all significantly different from zero (and positive) for physicians in the statin class, for all three drugs, and across all three physician segments. The incremental intercepts are, in general, not

**Table 7** Class 2 (Statin) Drugs: Estimated Parameters

Parameters estimated	Notation	GP/FP/DO (GFD)		IM		SPE	
		M1	M2	M1	M2	M1	M2
<b>Drug A (Combo-4)</b>							
Physician							
1 Utility intercept before trial	$b_{m,pre}^A$	<b>-3.8754</b>	<b>-3.9213</b>	<b>-3.8628</b>	<b>-3.9535</b>	<b>-4.0817</b>	<b>-4.1631</b>
2 Incremental utility intercept	$b_{m,inc}^A$	<b>1.2733</b>	<b>1.5890</b>	<b>1.1922</b>	<b>1.4319</b>	<b>1.1408</b>	<b>0.7598</b>
Patients							
3 Utility intercept before trial	$b_{p,pre}^A$		<b>-2.9446</b>		<b>-2.7764</b>		-0.1444
4 Incremental utility intercept	$b_{p,inc}^A$		-6.3617		-8.2630		<b>0.9853</b>
<b>Drug B (Lipitor)</b>							
Physician							
5 Utility intercept before trial	$b_{m,pre}^B$	<b>-6.4660</b>	<b>-6.2368</b>	<b>-6.5821</b>	<b>-6.4273</b>	<b>-6.6053</b>	<b>-6.5775</b>
6 Incremental utility intercept	$b_{m,inc}^B$	<b>10.8354</b>	<b>13.1888</b>	<b>13.8491</b>	<b>12.5002</b>	<b>9.7678</b>	<b>11.5289</b>
Patients							
7 Utility intercept before trial	$b_{p,pre}^B$		<b>-8.5012</b>		<i>-11.0023</i>		<b>-6.4750</b>
8 Incremental utility intercept	$b_{p,inc}^B$		-0.0220		0.0050		0.0000
<b>Drug C (Baycol)</b>							
Physician							
9 Utility intercept before trial	$b_{m,pre}^C$	<b>-8.7606</b>	<b>-8.7880</b>	<b>-9.2746</b>	<b>-9.1281</b>	<b>-9.7100</b>	<b>-9.5794</b>
10 Incremental utility intercept	$b_{m,inc}^C$	<b>10.6939</b>	<b>13.7324</b>	<b>13.1436</b>	<b>13.1503</b>	<b>11.6545</b>	<b>10.9178</b>
Patients							
11 Utility intercept before trial	$b_{p,pre}^C$		<b>-8.9298</b>		<b>-10.9949</b>		<b>-7.4910</b>
12 Incremental utility intercept	$b_{p,inc}^C$		-0.1630		0.0000		0.0000
Covariate							
13 Coefficient for marketing activities	$\varphi$	<b>0.5766</b>	<b>0.5799</b>	<b>0.4695</b>	<b>0.5010</b>	<b>1.5752</b>	<b>1.1676</b>
Initial membership (only nonusers estimated)							
14 Non-ABC-users	$\pi_{(0,0)}(t_0)$	<b>0.8829</b>	<b>0.9650</b>	<b>0.8714</b>	<b>0.9668</b>	<b>0.7998</b>	<b>0.8589</b>
A-users	$\pi_{(1,0)}(t_0)$	<b>0.1171</b>	<b>0.0350</b>	<b>0.1286</b>	<b>0.0332</b>	<b>0.2002</b>	<b>0.1411</b>
Patient's relative decision influence (conditional on his or her experience with the category)							
15 No experience	$\lambda_1$		<b>0.2543</b>		<b>0.1798</b>		<b>0.0085</b>
16 Partial experience	$\lambda_2$		<b>0.3337</b>		<b>0.1832</b>		<b>0.0081</b>
17 Full experience	$\lambda_3$		<b>0.9151</b>		<b>0.4048</b>		<b>0.1940</b>
Total data points used for estimation (including nonuser)		130	130	130	130	130	130
Log likelihood		-229,026	-229,018	-257,204	-257,197	-107,675	-107,673
AIC		-229,034	-229,035	-257,212	-257,214	-107,683	-107,690
BIC		-229,045	-229,059	-257,224	-257,238	-107,695	-107,714

Notes. Bold indicates significant at 0.01, and italic indicates significant at 0.05. M1 represents Model 1, no dyadic decision, and M2 represents Model 2, with dyadic decision. The model estimates the incremental utility, for example,  $b_{m,inc}^A = b_{m,post}^A - b_{m,pre}^A$ .

statistically different from zero for patients. This result is most likely due to the fact that high cholesterol (the disease that statin treats) is an asymptomatic disease, and a patient does not experience any reduction in

physical suffering (e.g., pain) even if a drug (statin) is highly effective. On the other hand, the physicians do observe the change in the measurement of cholesterol (patients also observe such measurement, but patients care more about what they experience). In the PPI class, which treats peptic ulcer, a medical condition with serious symptoms, patients in the PPI-SPE combination appear to have close to zero incremental intercepts for the two drugs. This could simply mean that these patients completely rely on the specialists for the decision. Even if they may actually have different utilities, these utilities cannot be recovered if the patients do not assert themselves in the prescription decision.

**Marketing Activities.** Marketing activities have a positive and significant impact on total prescriptions by influencing the physician's preference, as one

**Table 8** Model Fit and Six-Period Forecasting

	Statistics**	Average reduction in statistics*** (%)		
		Model 1	Model 2	
Fit	Average MAD	28.37	26.67	8.33
statistics* (%)	Average APE	9.24	8.78	6.53
Six-period forecast	Average MAD	64.88	56.88	15.65
statistics* (%)	Average APE	7.31	6.29	18.59

\*Analysis is based on results from the four class-specialty data sets where the dyadic model performs better than the nondyadic model in terms of LL.

\*\*MAD and APE are calculated based on the actual and predicted TRx.

\*\*\*The reduction in statistics is calculated for each class-specialty data set, and then the average is computed.

expects, with the only exception being specialists who prescribe PPI. This is somewhat surprising because marketing activities clearly have strong effects on specialists for statin. Upon closer examination, though, this does not appear to be due to the difference in specialists (gastroenterologist for PPI and cardiologist for statin). Instead, this appears to reflect the difference in the two diseases that two classes of drugs treat. Peptic ulcer (treated by PPI) has many serious symptoms, and those who are seeing a specialist most likely experience the most severe form of such symptoms (e.g., vomiting blood, severe abdominal pain, bloody stool). On the other hand, high cholesterol (treated by statin) is asymptomatic, and patients seeing a specialist still do not experience any symptoms even though their cholesterol levels may be very high. We thus conjecture that marketing activities, although useful in general, may not be effective for physicians treating patients with severe symptoms.

*Patients' Decision-Making Influence.* The decision-making relative influence is clearly dependent on the class of drugs as well as on the physician specialty. Note that a value of  $\lambda$  close to 1 indicates that a patient has influence in the final prescription choice as almost equal to that of a physician. (It does not mean that the patient makes prescription decisions.) A value close to 0 means that the physician makes the prescription decisions. In estimating it, we constrained  $\lambda$  to be bounded from above by 1, because physicians, by training and obligation, will not and should not let patients have more power over them (note that this is different from a situation where a physician may appear to accommodate a patient's request for a specific drug, because it could simply mean that the physician has a preference similar to that of the patient or is indifferent among several drugs).

One clear insight from the empirical analyses is that specialists tend to yield little decision-making power to their patients. Patients' preferences have essentially zero input in the final prescription decision unless the patients have had experience with all drugs in the class, and, even then, the relative influence of patients is rather small ( $\lambda_3 = 0.0077$  and  $\lambda_3 = 0.1940$  for the PPI and statin classes, respectively). Interviews with executives in the pharmaceutical industry corroborate this finding.<sup>8</sup>

Another insight obtained from the analyses is that patients tend to have more influence over the prescription decision as her/his experience with the drug

class increases. In all six data sets, we found that  $\lambda_3$  is the highest among the three influence parameters.  $\lambda_1$  and  $\lambda_2$  are more similar, compared to  $\lambda_3$ , and in general  $\lambda_2 > \lambda_1$  (the only exception is specialists for statins, but the difference is extremely small (0.0004)). This result is also consistent with anecdotal evidence.

Finally, overall it appears that patients' decision influence in the specialist segments is qualitatively different from that in the GFD and IM segments. This empirical result supports the conventional wisdom because the GFDs and internists are more similar in the training they receive and the type of patients they see, compared to specialists. A somewhat surprising result is the zero input of patients interacting with specialists for PPI. This may be due to the unique nature of the disease. Peptic ulcer (which PPI treats) is a serious disease with serious symptoms, including, but not limited to, severe pain, black stool, and vomiting (blood). Someone who is referred to see a specialist is likely to have the most serious symptoms of ulcer. These patients are not likely to be in a position to discuss with the specialist the specific treatment they will receive. In contrast, high cholesterol (the disease that statin treats) is asymptomatic (no symptoms). Patients who see specialists do not feel differently from those who see GFDs or IMs, even though the actual count of cholesterol may be much higher.

*Segment, Disease, and Generalizability.* Based on the observed parameter estimates and the discussion above, it appears that the patterns of utilities, effectiveness of marketing activities, and patients' decision-making powers all depend on the nature of disease, specifically, whether a disease is asymptomatic or not. Patients appear to play a minimum role in prescription decisions when they have serious symptoms. Furthermore, marketing activities, although effective in general, are not very effective for physicians seeing patients with serious symptoms.

There are also notable differences between behavior associated with primary care physicians (either GFDs or IMs) or specialists, as discussed above. It should be noted, however, that patients who see specialists are likely in more severe stages of the disease (ulcer or high cholesterol). The data sets do not control for this difference. This is a key reason why we have estimated the model separately for the three segments. Out of the six data sets we estimated, the specialists segment for PPI particularly stands out as unique. We conjecture that this is due to the combination of unique nature of the disease, physicians segment (specialists), and characteristics of those patients who see the specialists (severe symptoms).

**4.5.2. Models Fit.** To evaluate the fit of the two models, we examined the maximum log likelihood (LL), AIC, and BIC. Not surprisingly, the LL indicates,

<sup>8</sup> In the words of a senior vice president at one of the top ten pharmaceutical companies: patients are likely to have little influence with specialists because it might take six months to get an appointment with a specialist, patients are less likely to assert his or her opinion, and the specialist is less likely to listen.

with the exceptions of specialists in both drug classes, that the dyadic decision-making model (Model 2) outperforms the nondyadic model (Model 1) in fit. Taking into consideration the additional number of parameters used in Model 2, the AIC indicates that Model 2 is a better model in fit for GFD and IM in the PPI class, but Model 1 is a marginally better model in terms of fit for the GFD and IM in the statin class. Taking into consideration both the number of parameters and observations, the BIC suggests that Model 2 does not provide any improvement in fit in all cases. We examined additional fit statistics MAD (mean absolute deviation) and APE (absolute percentage of errors) for the four data sets (GFD and IM for PPI and statin) where Model 2 fits better in terms of LL. The results suggest that Model 2 performs better on both metrics (see Table 8).

#### 4.5.3. Out-of-Sample Predictive Performance.

The key criterion in assessing the capability of different models is whether one model outperforms the other in forecasting, not fit. We thus examine both performance metrics (MAD and APE) for six-period out-of-sample forecasting. Model 2 provides substantial improvement in forecasting performance in both metrics for all four data sets (see averages in Table 8). The reduction in either MAD or APE for each data set when Model 2 is used, as compared to the case when Model 1 is used, was also calculated. The averages across the four data sets are also presented in Table 8. Note that the reduction is upper-bounded (maximum reduction cannot exceed 100%), but it is not lower-bounded. Thus, the average reduction is a conservative measure of the relative performance of Model 2 vis-à-vis Model 1. Judging by LL, Model 2 outperforms Model 1 in three of the four data sets (except for IM in statins:  $-53,791.03$  for Model 1 and  $-53,791.04$  for Model 2).

#### 4.6. Comparison Against a Benchmark Model

While the dyadic model (Model 2) performs better than its individual decision-maker counterpart (Model 1), it is also important to evaluate its performance relative to a state-of-the-art existing model. It has been compared against the HPKZ (1994) model in terms of out-of-sample forecasting performance. Before the results of this comparison are presented, a few comments are in order.

Because the HPKZ single-drug model can be used only when the first in the class launching data are available (see Equation 2.15 in HPKZ 1994), we focus on the three drugs (Prevacid in the PPI class and Lipitor and Baycol in the statin class) for which we have such initial launching data points. Because we have three physicians segments for each of these three drugs, this provides us with a total of nine cases for comparison.

**Table 9** Predictive Comparison of the New Models to the HPKZ2 Model

Case	HPKZ2		New Model 1		New Model 2	
	MAD	APE (%)	MAD	APE (%)	MAD	APE (%)
Prevacid in the GFD physicians segment	12.46	2.52	3.95	0.87	5.11	1.13
Prevacid in the IM physicians segment	4.93	1.22	7.64	1.93	3.25	0.83
Prevacid in the specialists physicians segment	5.33	2.25	1.16	0.50	1.09	0.45
Baycol in the GFD physicians segment	3.79	6.61	3.20	5.54	3.19	5.54
Baycol in the IM physicians segment	1.30	3.23	3.25	7.03	1.02	2.49
Baycol in the specialists physicians segment	2.25	19.63	0.30	2.98	0.26	2.42
Lipitor in the GFD physicians segment	13.70	1.46	3.20	0.34	3.20	0.34
Lipitor in the IM physicians segment	11.52	1.15	3.31	0.34	1.01	0.11
Lipitor in the specialists physicians segment	2.34	0.59	0.30	0.08	0.26	0.07
Average	6.40	4.29	2.92	2.18	2.04	1.49
Reduction in statistics			30.98%	33.53	61.72%	61.09

Our model is more comprehensive and incorporates several aspects that the HPKZ model does not. To have a meaningful comparison, we need to make some adjustments. Recall that our model is a class level that simultaneously fits and forecasts the sales of all drugs competing in the class as well as the number of nonusers, considered as those patients who potentially should, but do not, use any drug in the class in a given period. This is different from the HPKZ model where nonusers are considered as those who do not use the focal drug. To make our model comparable to the HPKZ model we thus exclude people who do not use any drug in the class. Our model uses the original TRx data. Because the HPKZ model does not use the original TRx data but deseasonalizes data before using them, we also deseasonalize the data prior to employing our model in this comparison. Finally, there are two versions of HPKZ models. HPKZ1 employs the marketing variable as a ratio between a firm's expenditure and total expenditure by all competing firms. HPKZ2 employs the marketing variables as the total expenditures attributed to the focal product. Because HPKZ2 performs better than HPKZ1 (Hahn et al. 1994), the six-period forecasting performance of our new Model 2 is compared against the HPKZ2 model. The results are presented in Table 9.

Our new model clearly outperforms HPKZ2 in the out-of-sample forecasting testing. Both statistics (MAD and APE) are lower in Model 2 for all nine cases. Furthermore, the reductions in these statistics are substantial: both are more than 60% (note that reduction is upper-bounded by 100%). For completeness, we have also conducted estimation and prediction using Model 1 and included the results in Table 9. Model 1 also outperforms HPKZ2 in general,

although not to the same extent as Model 2. It thus appears that modeling the dynamics at the class level (Model 1 or Model 2) offers additional benefits over a modeling approach that focuses on an individual product level (HPKZ). The additional incorporation of dyadic utility further enhances the value of modeling the data sets we employed in this paper.

## 5. Conclusions and Future Research

The new dynamic forecasting model developed and described in this paper fills an important research gap. By modeling all competitors (current and future), we are able to parse out important effects not previously known. The dyadic decision-making mechanism incorporated in the model, an aspect that has not been considered before, enables making various inferences concerning the relative decision influence of the parties involved. It has implications in guiding management in how to target effectively patients with DTC. For example, the empirical analyses suggest that managers in the PPI and statin categories should target mostly those patients who visit GM/FP/DO physicians and, to a lesser extent, those who visit IM, but not those who visit specialists. Such targeting has a high likelihood of physicians complying with their patients' requests.

Judging by both fit and six-months-forward forecasting, we found that in many cases our model performs better when the dyadic decision-making structure is explicitly recognized and modeled, and it outperforms the benchmark HPKZ model. In most cases, the posttrial utility is larger than pretrial utility, lending partial empirical support to the common practice of spending on marketing activities (e.g., sampling to physicians) needed to get potential users to try a new drug. This empirical evidence requires, however, further cost-benefit analysis.

Several aspects, not captured in the current model, represent promising directions for future research. One of the most interesting works, we believe, is to obtain DTC promotional data and understand its impact under various conditions. This could be done by formulating the patient's pretrial utility's parameter as a function of the DTC expenditures. Such an analysis will generate new relevant empirical insights of great interest to researchers and practitioners. Likewise, incorporating brand equity and other relevant covariates that may impact the physician's utility might be of interest.

The model proposed here is based on decision-making unit choice that is next aggregated to the population level to utilize the aggregate data. It will certainly be interesting to obtain individual-level data, test the current model, and, if necessary, adapt it to obtain more refined empirical insights. Important issues that could be elucidated by such data include

other dimensions of heterogeneity across populations and dyadic decision-making mechanisms, not currently captured in our model. The model can also be extended to reflect that physicians will adopt a new drug (or include it in their consideration set) at different time periods. While individual-level data will address the heterogeneity issue, they will also impose a major burden on the estimation. It is possible, however, that a discrete segment approach (of adoption sequence) could provide a compromise.

Finally, it would be interesting and important to explore the potential of the model reported in this paper as a prelaunch forecasting tool. We sketch out four steps that will be required to achieve this goal: (1) a large number of empirical applications of the model that will estimate parameters in many different product categories; (2) a meta-analysis (e.g., Sultan et al. 1990) that regress these parameters against non-product-specific covariates (e.g., efficacy, side effect, delivery method); (3) constructing parameters for a new class of drugs (or a new drug) based on their non-product-specific covariates (e.g., efficacy and side effect of a drug) and results from step (2); (4) running the model using parameters constructed from step (3), launch information (time of sequential entry), and marketing activities to obtain the desired prelaunch forecast over time in competitive markets. Equally important, the model should be able to generate useful diagnostic information, including the number of trials and repeats in any future period. The prelaunch forecasting could be done either for "first in the class" drugs or for new drugs entering an existing class.

## 6. Electronic Companion

An electronic companion to this paper is available as part of the online version that can be found at <http://mansci.journal.informs.org/>.

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### References

- Aikin, K. J., J. L. Swasy, A. C. Braman. 2004. Patient and physician attitudes and behaviors associated with DTC promotion of prescription drugs—Summary of FDA survey research results. Report, U.S. Department of Health and Human Services, FDA, Center for Drug Evaluation and Research, Washington, D.C.
- Bates, D. M., D. G. Watts. 1988. *Nonlinear Regression Analysis and Its Applications*. John Wiley & Sons, New York.

- Chintagunta, P. K., D. C. Jain, N. J. Vilcassim. 1991. Investigating heterogeneity in brand preferences in logit models for panel data. *J. Marketing Res.* **28**(4) 417–428.
- Cooper, L. G. 1993. Market share models. J. Eliashberg, G. L. Lilien, eds. *Handbooks in Operations Research and Management Science*. Elsevier, New York, 259–314.
- Corfman, K. P., S. Gupta. 1993. Mathematical models of group choice and negotiations. J. Eliashberg, G. L. Lilien, eds. *Handbooks in Operations Research and Management Science*. Elsevier, New York, 83–142.
- Curry, D. J., M. B. Menasco, J. W. Van Ark. 1991. Multiattribute dyadic choice: Models and tests. *J. Marketing Res.* **28**(3) 259–267.
- Dyer, J. S., R. K. Sarin. 1979. Measurable multiattribute value functions. *Oper. Res.* **27**(4) 810–822.
- Eliashberg, J., R. L. Winkler. 1981. Risk sharing and group decision-making. *Management Sci.* **27**(11) 1221–1235.
- Gatignon, H., B. Weitz, P. Bansal. 1990. Brand introduction strategies and competitive environments. *J. Marketing Res.* **27**(4) 390–401.
- Givon, M. U. 1984. Variety seeking through brand switching. *Marketing Sci.* **3**(1) 1–22.
- Gliebe, J. P., F. S. Koppelman. 2002. A model of joint activity participation between household members. *Transportation* **29**(1) 49–72.
- Gliebe, J. P., F. S. Koppelman. 2005. Modeling household activity-travel interactions as parallel constrained choices. *Transportation* **32**(5) 449–471.
- Grimmett, G. R., D. R. Stirzaker. 1992. *Probability and Random Processes*. Oxford University Press, New York.
- Hahn, M., S. Park, L. Krishnamurthi, A. A. Zoltners. 1994. Analysis of new product diffusion using a four-segment trial-repeat model. *Marketing Sci.* **13**(3) 224–247.
- Harsanyi, J. C. 1955. Cardinal welfare, individual ethics, and interpersonal comparisons of utility theory. *J. Political Econom.* **63** 309–321.
- Hauser, R. J., K. J. Wisniewski. 1982. Dynamic analysis of consumer response to marketing strategies. *Management Sci.* **28**(5) 455–486.
- Kahn, B. E., M. U. Kalwani, D. G. Morrison. 1986a. Measuring variety-seeking and reinforcement behavior using panel data. *J. Marketing Res.* **23**(2) 89–100.
- Kahn, B. E., D. G. Morrison, G. P. Wright. 1986b. Aggregating individual purchases to the household level. *Marketing Sci.* **5**(3) 260–268.
- Keeney, R. L., H. Raiffa. 1976. *Decisions with Multiple Objectives*. John Wiley & Sons, New York.
- Kimball, G. E. 1957. Some industrial applications of military operations research methods. *Oper. Res.* **5**(2) 201–204.
- Kirkwood, C. W., R. K. Sarin. 1980. Preference conditions for multiattribute value functions. *Oper. Res.* **28**(1) 225–232.
- Krishnan, T. V., F. M. Bass, V. Kumar. 2000. Impact of a late entrant on the diffusion of a new product/service. *J. Marketing Res.* **37**(2) 269–278.
- Lilien, G. L., A. G. Rao, S. Kalish. 1981. Bayesian estimation and control of detailing effort in a repeat purchase diffusion environment. *Management Sci.* **27**(5) 493–506.
- Mahajan, V., Y. Wind, S. Sharma. 1983. An approach to repeat purchase diffusion models. *Proc. Amer. Marketing Educator's Conf.*, Amer. Marketing Association, Chicago, 442–446.
- McFadden, D. 1981. Econometric models of probabilistic choice. C. F. Manski, D. McFadden, eds. *Structural Analysis of Discrete Data with Econometric Applications*. MIT, Cambridge, MA, 198–272.
- McGuire, T. W., R. Staelin. 1983. An industry equilibrium analysis of downstream vertical integration. *Marketing Sci.* **2**(2) 161–191.
- O'Connell, V. 2002. Patients' success in drug requests shows the power of medication ads. *Wall Street Journal* (April 15).
- Ofek, E., V. Srinivasan. 2002. How much does the market value an improvement in a product attribute? *Marketing Sci.* **21**(4) 398–411.
- Pakes, A. 1986. Patents as options: Some estimates of the value of holding European patent stocks. *Econometrica* **54**(4) 755–784.
- Parker, P., H. Gatignon. 1994. Specifying competitive effects in diffusion models: An empirical analysis. *Internat. J. Res. Marketing* **11**(1) 17–39.
- Parker, P., H. Gatignon. 1996. Order of entry, trial diffusion, and elasticity dynamics: An empirical case. *Marketing Lett.* **7**(1) 95–109.
- Petrin, A. 2002. Quantifying the benefits of new products: The case of the minivan. *J. Political Econom.* **110**(4) 705–729.
- Rao, A. G., M. Yamada. 1988. Forecasting with a repeat purchase diffusion model. *Management Sci.* **34**(6) 734–752.
- Seber, G. A. F., C. J. Wild. 1989. *Nonlinear Regression*. Wiley, New York.
- Shankar, V. 1999. New product introduction and incumbent response strategies: Their interrelationship and the role of multimarket contact. *J. Marketing Res.* **36**(3) 327–344.
- Shankar, V., G. S. Carpenter, L. Krishnamurthi. 1998. Late mover advantage: How innovative late entrants outsell pioneers. *J. Marketing Res.* **35**(1) 54–70.
- Shankar, V., G. Carpenter, L. Krishnamurthi. 1999. The advantages of entering in the growth stage of the product life cycle: An empirical analysis. *J. Marketing Res.* **36**(2) 269–276.
- Song, I., P. K. Chintagunta. 2003. A micromodel of new product adoption with heterogeneous and forward-looking consumers: Application to the digital camera category. *Quant. Marketing Econom.* **1** 371–407.
- Sudhir, K. 2001. Competitive pricing behavior in the auto market. *Marketing Sci.* **20**(1) 42–60.
- Sultan, F., J. U. Farley, D. R. Lehmann. 1990. A meta-analysis of applications of diffusion models. *J. Marketing Res.* **27**(1) 70–77.
- Zufryden, F. 1986. Multibrand transition probabilities as a function of explanatory variables: Estimation by a least-squares-based approach. *J. Marketing Res.* **23**(2) 177–183.