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Heterogeneous Learning and the Targeting of Marketing Communication for New Products

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New product launches are often accompanied by extensive marketing communication campaigns. Firms' allocation decisions for these marketing communication expenditures have two dimensions—across consumers and over time. This allocation problem is different relative to the problem of allocation of resources for existing products. In the case of new products, consumers are uncertain about their quality and learn about the products through marketing communication. Furthermore, different consumers may have different rates of learning about product quality; i.e., there may be heterogeneous learning. Thus, consumer responsiveness to marketing communication could vary along two dimensions. For each consumer, this responsiveness would vary over time, as she learns about product quality. Across consumers, there would be differences in responsiveness in each time period. For optimal allocation of marketing communication across both consumers and time, firms would need estimates of how consumer responsiveness varies across consumers and over time. Past studies have typically focused on one of these two dimensions in which responsiveness varies. They have either looked at heterogeneity in responsiveness across agents or the variation in responsiveness over time. In the context of new products, past research has looked at how consumer learning about product quality causes responsiveness to vary over time. In this study, we build a model that allows for heterogeneous learning rates and obtain individual-level learning parameters for each consumer. We use a novel and rich panel data set that allows us to estimate these model parameters.

To obtain individual-level estimates of learning, we add a hierarchical Bayesian structure to the Bayesian learning model. We exploit the natural hierarchy in the Bayesian learning process to incorporate it in the hierarchical Bayesian model. We use data augmentation, coupled with the Metropolis-Hastings algorithm, to make inferences about individual-level parameters of learning. We conduct this analysis on a unique panel data set of physicians where we observe prescription decisions and detailing (i.e., sales-force effort) at the individual physician level for a new prescription drug category.

Our results show that there is significant heterogeneity across physicians in their rates of learning about the quality of new drugs. We also find that there are asymmetries in the temporal evolution of responsiveness of physicians to detailing—physicians who are more responsive to detailing in early periods are less responsive later on and vice versa. These findings have interesting implications for the targeting of detailing across physicians and over time. We find that firms could increase their revenue if they took these temporal and cross-sectional differences in responsiveness into account while deciding on allocations of detailing.

Key words: resource allocation; pharmaceutical markets; learning models; Markov chain Monte Carlo methods History: Received: November 21, 2005; accepted: February 2, 2008; processed by Wagner Kamakura. Published online in Articles in Advance January 12, 2009.

1. Introduction

New products are the lifeblood of firm performance—they account for about a quarter of all sales and revenue growth. Firms spend about half their marketing budgets promoting new products (see Urban and Hauser 1993). A major concern of firms is the allocation of marketing resources during the launch and rollout of a new product. The mechanism that governs this resource allocation over time is the response of the market to this new product. This response is

driven by considerable uncertainty about the quality of the new product. A major role of marketing activity is to disseminate information about new products in a manner that reduces that uncertainty. Consumers typically use this information to reduce their uncertainty about a new product's quality via a learning process. Besides the evolution of this learning process over time, consumers may also differ in their learning behavior; i.e., there may be heterogeneous learning. For example, some consumers may learn faster than

others about the quality of new products. Firms could use their knowledge of this heterogeneous learning to allocate marketing resources both over time and across consumers. If the extent of this heterogeneity is large, then firms stand to gain significantly if they can incorporate this knowledge into their product rollouts.

However, previous literature has typically calibrated this learning process for the market as a whole. In this research, we use unique data on the launch of new (ethical) pharmaceutical drugs, and a novel methodology to calibrate learning processes for each individual (consumer) physician. Our approach is able to estimate the individual rate of learning for each physician after controlling for other behaviors such as risk aversion. We then use these estimated rates to examine firms' marketing resource allocation during the time of product rollout.

The pharmaceutical industry in general is particularly well suited to study this problem for three reasons. First, uncertainty about drug quality is particularly relevant in the case of prescription drugs. In spite of an extensive process of clinical trials before the launch of new drugs, there is still considerable uncertainty about their quality, side effects, and other associated risks. For instance, the Food and Drug Administration (FDA), without whose approval prescription drugs cannot be marketed, has the following to say about new drugs (Center for Drug Evaluation and Research (CDER) 2000, p. 24):

The practical size of pre-marketing clinical trials means that we cannot learn everything about the safety of a drug before we approve it. Therefore, a degree of uncertainty always exists about the risks of drugs.

Furthermore, while the premarketing clinical trials provide information at the mean level (efficacy, incidence of side effects, etc.), physicians with heterogeneous patient bases are often uncertain about the quality of a new drug for their specific bases.

Second, pharmaceutical firms spend a large amount of money on marketing communication directed towards physicians. For example, the industry spent \$8.5 billion on marketing communication directed at physicians in 2000 (Wittink 2002, Neslin 2001). Most of this is spent on detailing, which involves personal sales calls made by salespersons of the pharmaceutical firm to physicians. Third, because detailing is a personal interaction between a physician and the firm's representatives, it is allocated at the individual physician level. Firms need to decide how many calls to make to each individual physician and when to make these calls. These three factors—true uncertainty about a new product, large expenditure, and individual-level allocation—make the pharmaceutical industry an excellent setting for our problem.

In this study, we use a unique panel data set of physicians, which contains prescription and detailing information for all drugs in a category, to estimate individual physician-level effects in the presence of learning. We develop a methodology to estimate individual physician-level effects by specifying a Bayesian learning model for each physician and estimating the model in a hierarchical Bayesian framework using Markov chain Monte Carlo (MCMC) methods. Our results show that there is considerable heterogeneity in learning rates across physicians. This heterogeneity is economically significant for the firm, and accounting for it can lead to considerable revenue gain. Specifically, heterogeneity in learning accounts for about 50%–56% of the revenue gain, with the rest coming from heterogeneity in the other individuallevel effects.

The rest of the paper is organized as follows. In §2, we briefly discuss the extant literature related to this study. In §3, we discuss the data. Section 4 presents the model in detail, and we discuss our estimation strategy. In §5, we discuss identification of our model parameters. Section 6 covers the results of our analysis and our counterfactual simulations. In §7, we discuss the managerial implications of our study. Section 8 concludes with a discussion of the model, our results, and the limitations of the study.

2. Related Literature

This study is broadly related to three streams of research in the literature—pharmaceutical promotions, learning, and targeted marketing. We discuss these briefly and point out the contribution of this study relative to these streams.

Although there have been many research studies on pharmaceutical promotions, none has focused on the targeting of these promotions in the presence of learning. Early studies in the marketing literature (Parsons and Vanden Abeele 1981, Lilien et al. 1981) covered the effect of sales-force effort on sales using aggregate data. Recent research (Kamakura et al. 2004, Manchanda and Chintagunta 2004, Gönül et al. 2001, Manchanda et al. 2004) has used panel data to investigate the effect of detailing on pharmaceutical demand (see Manchanda and Honka 2005 for an extensive review of detailing studies). Other research has also investigated the role of pharmaceutical promotion, classifying it as informative and persuasive (Leffler 1981, Hurwitz and Caves 1988, Rizzo 1999, Narayanan et al. 2005). The broad consensus in this literature is that detailing affects prescriptions by physicians positively and that there is heterogeneity in physician response to detailing.

The second stream of research that is relevant to this study is the literature on Bayesian learning (good examples of early studies are Stoneman 1981, Meyer and Sathi 1985, and Roberts and Urban 1988). Erdem and Keane (1996) produced a pioneering paper in marketing that used a model of Bayesian learning to incorporate informative effects of advertising. Since then, there has been a growing interest in problems involving learning (Crawford and Shum 2005, Coscelli and Shum 2004, Anand and Shachar 2004, Ackerberg 2003, Narayanan et al. 2005, Byzalov and Shachar 2004). These studies find evidence that advertising and product experience generate significant learning and reduce uncertainty. Ackerberg (2003), Byzalov and Shachar (2004), and Narayanan et al. (2005) specifically address the issue of informative and noninformative roles of advertising or promotional activity. Narayanan et al. (2005) find evidence for the presence of both these roles, whereas the other studies find evidence for only the informative effect. Heilman et al. (2000) and Ackerberg (2001) also suggest that the role of marketing communication is different for new versus familiar products.

Targeted promotions have interested marketing researchers in recent years. Targeting promotions to segments of consumers has long been an industry practice as well as a topic for research. Numerous studies have looked at price discrimination (cf. Villas-Boas 1999, Fudenberg and Tirole 2000), targeted coupons (Shaffer and Zhang 1995), and the targeting of advertising (cf. Iyer et al. 2005) to different segments of consumers. Rossi et al. (1996) and Manchanda et al. (2004) show that firms can obtain significant benefits by targeting their promotions.

This paper builds on the literature on pharmaceutical promotion by explicitly modeling the process by which detailing affects individual physician prescription behavior from the launch of a product (drug). This is the first study to document heterogeneity in both the informative and persuasive effects of marketing communication such as detailing. In addition, this study documents, for the first time, the effect of patient influence on physicians' prescription behavior. The paper also adds to the literature on Bayesian learning models by proposing a methodology to estimate learning rates at the individual level, as opposed to the extant literature, which assumes that all agents learn at the same rate. Finally, this paper contributes to the research on targeted marketing by providing insights on targeting in early stages after the launch of a product. Thus, this paper contributes both substantively and methodologically to marketing literature.

3. Data

The data used for the empirical analysis in this study are from the category of prescription drugs known as erectile dysfunction (ED) drugs. The drugs in this category are prescribed to treat ED among adult men—a condition that affects between 15 and 30 million men in the United States. There is only one category of oral drugs that can treat this condition, and currently there are three drugs that have been approved by the FDA. The three drugs are Viagra (marketed by Pfizer and approved by FDA in March 1998), Levitra (marketed jointly by GSK Pharmaceuticals and Bayer, and approved in August 2003), and Cialis (marketed by Eli Lilly and approved in November 2003). To date, no further drugs have been approved.

The data are at the physician level from a panel of 900 physicians in the United States. These data were obtained from a New Jersey-based data-vending firm—ImpactRx—that set up this panel and sells the data to pharmaceutical firms. The panel is a representative sample of physicians, balanced across geographic regions, specialties, and prescription volume. For these physicians, we have observations of prescriptions written for their patients and of detailing calls made by representatives of pharmaceutical firms (detailers). These data are collected directly from the physician using a Personal Digital Assistant. There are 15,320 prescription observations and 16,700 observations of detailing calls in the data set.

We have specialty information (e.g., general practitioner, urologist) for each physician. In each prescription observation, we observe the drug that was prescribed. Unlike most existing data sources, which collect prescription data from pharmacies or insurance companies, our data set captures the physician's decision. This is because pharmacy data, for instance, may be unable to capture the fact that the prescription filled in the pharmacy may sometimes be different from that prescribed by the physician. Our data set captures the physician's intended prescription and thus is not susceptible to slippage or missing data. A unique feature of the prescription data in our set is that we observe whether a patient requested a drug. This is recorded by the physician at the time of the consultation. In terms of detailing, the physician records the drug that is detailed in that call on every detail visit.

In terms of temporal patterns, about 90% of physicians are detailed at least once for the new drugs within three months of launch. The mean number of calls after which Levitra and Cialis are adopted is 2.8 and 2.4, respectively, and the correlation in adoption calls across physicians for the two drugs is 0.48. By the end of the data, 490 (55%) of physicians have prescribed all three drugs at least once, while 159 (18%) of physicians prescribed only one drug. The mean number (standard deviation) of total new prescriptions written by a physician was 8.95 (18.22),

¹ See Lilien (1974a, b) for a model that does not impose a specific functional form on the learning process.

12.26 (19.60), and 21.39 (30.10) for Cialis, Levitra, and Viagra, respectively, resulting in overall market shares of 21.0%, 28.8%, and 50.2%. By the last month of the data, however, the share pattern had changed significantly: 38.1%, 33.4%, and 28.5% for Cialis, Levitra, and Viagra, respectively. The mean number (standard deviation) of total details received by a physician was 5.55 (6.14), 7.65 (7.64), and 5.68 (6.12) for Cialis, Levitra, and Viagra, respectively. The average correlation between total prescriptions and total details across physicians was 0.38 (Cialis), 0.42 (Levitra), and 0.32 (Viagra). The number of patient requests (i.e., when a patient specifically asks for a drug to be prescribed) was relatively small; on average, patients requested any one of the three drugs only 15.4% of the time. The largest number of requests in aggregate is for Viagra (51.7% of the total requests), followed by Levitra (25.5%) and Cialis (22.8%). However, in the last month in which data are available, these proportions were 32.6% (Viagra), 21.1% (Levitra), and 46.3% (Cialis).

4. Model

4.1. Model Development

The paper that is closest to ours, both in terms of the empirical setting and the model itself, is Crawford and Shum (2005) (henceforth CS). We base our model on the CS model, which in turn is an extension of learning models previously proposed in the literature (Erdem and Keane 1996 and others). We first describe the CS model and then point out the similarities and differences of our approach with respect to that model.

CS was one of the early attempts to understand decisions made under uncertainty in the context of prescription drugs. They observe a sequence of prescription decisions for a panel of patients, for the category of anti-ulcer drugs. These drugs provide both symptomatic and curative benefits, i.e., they mitigate the symptoms of the conditions for which they are prescribed and also cure the condition, such that the patients can discontinue treatment. CS model learning about the specific patient-drug match, both with respect to the symptomatic benefit of the drug, as well as the curative benefit. They assume that patients are uncertain about these match values, but learn about them in a Bayesian manner through use. Furthermore, patients are assumed to be forward looking; i.e., they maximize their expected stream of future utilities in choosing a drug. In the CS setup, risk-averse patients compare their expected utilities across different drugs and choose the drug that provides them the highest expected utility. Patients are also assumed to start out with rational expectations; i.e., they know the population distribution of match values but do not know their specific match value.

First, we describe our model assumptions that are similar to those in CS. Like CS, we assume that at every prescription occasion, physicians maximize a utility function to decide which drug to prescribe. We similarly abstract away from any agency issues involved in this decision and assume that physicians represent the physician-patient pair perfectly. The physicians in our model are risk-averse agents who are uncertain about the qualities of the new drugs and maximize expected utilities to make the drug choice at every prescription occasion. Physicians are assumed to be Bayesian updaters, i.e., they are assumed to have some prior beliefs about the drugs qualities, and they are assumed to update these prior beliefs with any new information they receive using Bayes rule to obtain posterior beliefs. They are assumed to use the most recent posterior beliefs when evaluating their expected utilities for the choice alternatives. These posterior beliefs are in turn the prior beliefs for the subsequent update. Another common assumption between the two studies is that physicians know the overall quality distribution for a drug across all patients at the initial time period, although they do not know the quality of the drug for their specific patient bases. Finally, like CS, we also have a conditional-choice situation; i.e., we observe a patient in our data set only if he decides to take a drug, and hence, we do not model the patient's decision of whether to seek drug therapy.

Next, we describe some significant differences in our model assumptions relative to those in CS. First, the focus of CS is learning by individual patients about their match values for drugs. By contrast, our focus is on learning by individual physicians about the mean quality of the drugs for their patients. Our data are at the physician level, whereas those of CS are at the patient level. Thus, we observe the sequence of prescription decisions by a specific physician but cannot track specific patients. CS, on the other hand, have data on the sequence of visits by a particular patient but do not observe the sequence of prescriptions by a physician. This suggests that our data are well suited to study learning about the overall quality of a drug, whereas theirs are well suited to study learning about patient-specific match values. A second difference in our approach is that in the CS case, the drug can cure the disease as well as provide symptomatic benefits, while in our case, the drug can only provide symptomatic benefits. Thus, while they model learning about curative as well as symptomatic match values, we model learning only about an overall symptomatic quality of the drug. A third difference is that CS model learning only through the patient's own experience and feedback to the physician, while we model learning through feedback as well as marketing communication directed at the physician. Thus, we address the problem of optimal resource allocation for physician-directed marketing communication by pharmaceutical firms, especially in the context of new drugs. Another difference is that while the CS model assumes forward-looking agents, we assume that agents are myopic.² The most important difference in our approach relative to CS is that they assume that all physicians learn at the same rate. In the Bayesian learning model used in both CS and this paper, the rate at which physicians learn is summarized by the variances of the signals through which they update their beliefs about the drugs. We allow the signal variances to be different for different physicians, thus allowing for different rates of learning. In the CS approach, all physicians have the same signal variance and thus learn at the same rate, all else remaining equal. A key contribution of this paper is that we model heterogeneous learning and develop a general Bayesian hierarchical approach (using MCMC methods) to estimate learning rates at the lowest level of aggregation (individual physicians in our case).

4.2. Model Specification

When physician i has to make a decision on which drug to prescribe at occasion t, she chooses the alternative j that provides the greatest utility, with the utility function defined as

$$\widetilde{U}_{ijt} = -\exp(-r_i\widetilde{Q}_{ijt}) + X_{ijt}\beta_i + \varepsilon_{ijt}, \qquad (1)$$

where

 \widetilde{Q}_{ijt} is physician i's belief about the true quality of drug j at time t and is stochastic from the point of view of the physician;

 r_i is the coefficient of absolute risk aversion;

 X_{ijt} is a row vector $(1 \times K)$ of physician, drug, and time (patient)-specific variables;³

 β_i is a column vector ($K \times 1$) of physician-specific sensitivity to these variables; and

 ε_{ijt} is an i.i.d. physician, drug, and time-specific shock.

This utility function, which resembles the one in CS, has a subutility function in drug quality that has the constant absolute risk aversion (CARA) form. Thus, the underlying assumption is that physicians have a CARA with respect to uncertainty in drug quality. The utility function is linear in drug and patient-specific variables and in an additive shock that includes the match value between a drug and a specific patient.

As we describe later, our data set includes only the new prescriptions to patients, and hence, the i.i.d. assumption for this shock is reasonable. Note that because we have an observation every time a patient walks into the office and because we do not have repeated observations for the same patient, the prescription occasion t is identical to patient t for the physician.

This utility is stochastic from the physician's perspective because the belief about the quality \widetilde{Q}_{ijt} is stochastic. Remember, however, that ε_{ijt} is not stochastic from the point of view of the physician (i.e., the physician observes the ε_{ijt} on each prescription occasion). The physician is assumed to be an expected utility maximizer. This expected utility is

$$U_{iit} = E[\widetilde{U}_{iit}] = E[-\exp(-r_i\widetilde{Q}_{iit})] + X_{iit}\beta_i + \varepsilon_{iit}.$$
 (2)

We allow the true mean qualities of the drugs to be heterogeneous across physicians. This is analogous to the heterogeneous true match values in the CS model. In our case, these mean qualities represent the average qualities of the drugs for the specific patient base of each physician and are heterogeneous because patient bases are different across physicians.

Physicians are assumed to update their quality belief in each period based on signals they receive through detailing and through patient feedback (via past prescriptions). We assume that prescriptions written by a physician generate feedback from the patient after a period of 30 days. This is based on the fact that about 98% of all prescriptions in the ED category are for 30 days or fewer. We obtained this number from the Pharmtrends database maintained by Ipsos North America. Detailing and feedback signals are assumed to be normally distributed around the physician-specific true mean quality of the drug.

Assume that there are nd_{ijt} detailing signals at time t and the mth signal is assumed to be given as

$$\widetilde{D}_{ijtm} \sim N(Q_{ij}, \sigma_{D_i}^2),$$
 (3)

and that there are nf_{jt} patient feedback signals, and the mth signal is given by

$$\tilde{F}_{ijtm} \sim N(Q_{ij}, \sigma_{F_i}^2).$$
 (4)

A series of unobserved signals that are normally distributed can be summarized by their sample mean, which is also normally distributed. We define these sample means as

$$\widetilde{D}_{ijt} = \frac{\sum_{m} \widetilde{D}_{ijtm}}{nd_{iit}} \sim N\left(Q_{ij}, \frac{\sigma_{D_i}^2}{nd_{iit}}\right)$$
 (5)

$$\widetilde{F}_{ijt} = \frac{\sum_{m} \widetilde{F}_{ijtm}}{n f_{ijt}} \sim N\left(Q_{ij}, \frac{\sigma_{F_i}^2}{n f_{ijt}}\right). \tag{6}$$

² Without this assumption, our model is analytically intractable because the state-space for a model without this assumption is very large. For our problem, it will be 7,200—the product of 900 physicians and eight state variables.

³ The set of variables included in this vector are described in Equation (12).

The quality belief at time t=0 is assumed to be a normal distribution whose mean is the mean of the population distribution of the true quality and whose variance is the variance of this population distribution. Thus, the assumption is that physicians know the distribution of the true quality across all physicians but are uncertain about the true quality for their own patient base.

As described earlier, and similar to CS, the physician is assumed to update her beliefs in a Bayesian manner; i.e., at any given period of time, she combines her prior belief about the quality of the drug with the information obtained through detailing and feedback signals and applies Bayes rule to form her posterior belief. Because the prior belief at time t=0 and all signals are assumed to be normally distributed, the posterior belief at every time period is also a normal distribution. This posterior belief is given by

$$\widetilde{Q}_{ijt} \sim N(Q_{ijt}, \sigma_{Q_{iii}}^2),$$
 (7)

where

$$Q_{ijt} = \frac{\sigma_{Q_{ijt}}^2}{\sigma_{Q_{ij(t-1)}}^2} Q_{ij(t-1)} + n f_{ijt} \frac{\sigma_{Q_{ijt}}^2}{\sigma_{F_i}^2} \tilde{F}_{ijt} + n d_{ijt} \frac{\sigma_{Q_{ijt}}^2}{\sigma_{D_i}^2} \tilde{D}_{ijt}, \quad (8)$$

and

$$\sigma_{Q_{ijt}}^{2} = \frac{1}{1/\sigma_{Q_{ii(t-1)}}^{2} + nd_{ijt}/\sigma_{D_{i}}^{2} + nf_{jt}/\sigma_{F_{i}}^{2}}.$$
 (9)

It is important at this time to point out how heterogeneity in learning manifests itself in the model. Note also that the variances of the detailing and feedback signals ($\sigma_{D_i}^2$ and $\sigma_{E_i}^2$, respectively), are physician specific. It can be seen from Equation (8) that for a physician for whom $\sigma_{D_i}^2$ is a large value, relatively lower weight is placed on the detailing signal \bar{D}_{ijt} than for a physician with a low value of σ_D^2 , all else remaining the same. The former physician learns more slowly from detailing than the latter. Similarly, a physician with a higher value of σ_E^2 would learn more slowly from feedback than a physician with a lower value of this parameter. Thus, the variances of the detailing and feedback signals summarize the heterogeneity in learning across physicians. By contrast, in traditional learning models, these variances are assumed to be homogenous across agents. Allowing for these variances to be individual specific is a key contribution of our approach.

Given that the quality belief in any period is a normal distribution with mean Q_{ijt} and variance $\sigma^2_{Q_{ijt}}$ and r_i is the coefficient of absolute risk aversion, the expected utility of the physician is

$$U_{ijt} = E[\widetilde{U}_{ijt}] = -\exp\left(-r_i Q_{ijt} + \frac{r_i^2 \sigma_{Q_{ijt}}^2}{2}\right) + X_{iit} \beta_i + \varepsilon_{iit}.$$
(10)

Next, we describe the variables included in the linear subutility function $(X_{iit}\beta_i)$.

In our model, we have already described how detailing affects the prescription decision of the physician, through its effect on learning about drug quality. This effect is referred to as the informative effect of detailing or sometimes as its indirect effect (Ackerberg 2004, Narayanan et al. 2005). It can be seen from Equation (8) that the effect of detailing on the physician's quality belief is highest initially and reduces with every subsequent updation. This can be seen from the fact that the variance of the detailing signal $\sigma_{O_{iii}}^2$ converges towards zero with every updation and thus, the coefficient of the detailing signal in Equation (8) also converges to zero. Thus, the informative effect of detailing is highest at the introductory phase of a drug and after the physician has learnt about the drug and reduced her uncertainty, this effect is negligible.

However, it is well documented that detailing has an effect on physicians' prescription behavior even in the case of mature products (cf. Gönül et al. 2001, Manchanda and Chintagunta 2004). It has been suggested that this effect in the case of mature products may be because of an image or prestige role of detailing, or perhaps stems from reminder effects. We refer to all effects of detailing, except the informative effect defined in the previous section, as the persuasive effect. As noted earlier, these terms—informative effect and persuasive effect—are labels to differentiate between the two and are not meant to convey that one effect exclusively involves information and the other persuasion. The inclusion of these detailing effects is a significant difference from the CS study, in which marketing communication is not part of the model.

The persuasive effect of detailing is captured in a reduced form manner by including a stock of detailing counts for the 30-day period preceding the prescription occasion in the linear X_{ijt} variable in the utility function (Equation (1)). The coefficient of this variable measures the persuasive effect of detailing. This effect would capture any role of detailing that remains unchanged over the product life cycle of the drug. This approach is similar to that adopted in the literature (Ackerberg 2003, Anand and Shachar 2004, Narayanan et al. 2005).

Our data set allows us to account for patient influence on the prescription decision of the physician. We observe in the data whether the patient requested a specific drug. A dummy variable indicates whether the patient requested the drug in the linear X_{ijt} variable in the utility function in Equation (1). The coefficient of this variable captures the influence of the patient's request on the prescription decision of the physician. Indirectly, this also captures the effect of direct-to-consumer (DTC) advertising because DTC

often asks a patient to talk to the doctor about the drug. In summary, X_{iit} is thus given by

$$X_{ijt} = (DetailingStock_{ijt} PatientRequest_{ijt}).$$
 (11)

Our empirical analysis suggests revenue-enhancing allocation plans for detailing. Because detailing has to be allocated at the individual physician level, estimates of the effect of detailing must also be obtained at that level. Past studies using Bayesian learning models, including CS, have used frequentist methods for estimation, making the estimation of individual physician level parameters infeasible. A Bayesian approach is a natural way to estimate these parameters. We provide a new modeling approach by estimating a Bayesian learning model under a hierarchical Bayesian framework. We use MCMC methods to estimate the individual-level parameters.

However, the challenge in specifying our model as a hierarchical Bayesian model is that the quality beliefs are unobserved. In a standard frequentist estimation of learning models (Erdem and Keane 1996), one could integrate out these unobserved quality beliefs by simulation methods. However, for MCMC methods, we use a simple observation: From Equation (8), it is clear that not only is the quality belief, Q_{ijt} , a stochastic variable, but its mean, Q_{ijt} , is also stochastic. This is because, from Equation (8), Qijt is a function of two stochastic variables—the realizations of the detailing and feedback signals, D_{iit} and \vec{F}_{ijt} , respectively. Furthermore, because these two variables are assumed to have normal distributions, Q_{iit} is a linear combination of normal variables and therefore is also a normal variable.

In particular, we can derive the distribution of Q_{ijt} , conditional on $Q_{ii(t-1)}$ as

$$Q_{ijt} \mid Q_{ij(t-1)} \sim N(\overline{Q}_{ijt}, \nu_{ijt}^2), \tag{12}$$

where

$$\overline{Q}_{ijt} = \frac{\sigma_{Q_{ijt}}^2}{\sigma_{Q_{ii(t-1)}}^2} Q_{ij(t-1)} + \left(n f_{ijt} \frac{\sigma_{Q_{ijt}}^2}{\sigma_{F_i}^2} + n d_{ijt} \frac{\sigma_{Q_{ijt}}^2}{\sigma_{D_i}^2} \right) Q_{ij}, \quad (13)$$

and

$$\nu_{ijt}^{2} = n f_{ijt} \frac{\sigma_{Q_{ijt}}^{4}}{\sigma_{F_{i}}^{2}} + n d_{ijt} \frac{\sigma_{Q_{ijt}}^{4}}{\sigma_{D_{i}}^{2}}.$$
 (14)

Note that from Equation (9), the variance of the quality belief $\sigma_{Q_{ijt}}^2$ is not a stochastic variable, conditional on the parameters of the model. Given those parameters, it is known deterministically. The main difference between the mean Q_{ijt} and variance of the quality belief $\sigma_{Q_{ijt}}^2$ is that while the former depends on the (unobserved) *realizations* of the detailing signal \widetilde{D}_{ijt} and the feedback signal \widetilde{F}_{ijt} , the latter does not. Hence, in a frequentist estimation of a learning model,

we integrate out the mean but not the variance of the quality belief. Analogously, in a hierarchical model, we specify the mean of the quality belief as a level of the hierarchy, but not the variance.

Given that we can write the unobserved mean of the quality belief in any period as a random variable, conditional on the mean of the quality in the previous period, we thus have a natural hierarchy of quality beliefs:

$$Q_{ijt} \mid Q_{ij(t-1)} \sim N(\overline{Q}_{ijt}, \nu_{ijt}^{2}),$$

$$Q_{ij(t-1)} \mid Q_{ij(t-2)} \sim N(\overline{Q}_{ij(t-1)}, \nu_{ij(t-1)}^{2}),$$

$$...$$

$$Q_{ij1} \mid Q_{j0} \sim N(\overline{Q}_{ij1}, \nu_{ij1}^{2}).$$
(15)

We can then specify the rest of the hierarchical model as follows. We assume that the prescription choice follows a probit process. Thus, the random errors ε_{ijt} of the utility function in Equation (1) follow a multivariate normal distribution:

$$\begin{bmatrix} \varepsilon_{i1t} \\ \cdot \\ \varepsilon_{ijt} \end{bmatrix} \sim MVN(0, \Sigma). \tag{16}$$

The alternative that provides the greatest utility is chosen. Hence, U_{ijt} follows a truncated multivariate distribution, conditional on choice. If choice is given by the indicator variable I_{ijt} (which is 1 if brand j is chosen and 0 otherwise), the truncation is such that

$$U_{ijt} > U_{ikt}$$
, $I_{ijt} = 1$, $I_{ikt} = 0 \quad \forall k \neq j$. (17)

Let the vector γ_i (dimension $K \times 1$) denote the individual level parameters of the model. These parameters are specified as a function of physician-level characteristics, as follows:

$$\gamma_i = [\beta_i' \quad \ln(\sigma_{D_i}^2) \quad \ln(\sigma_{F_i}^2) \quad \ln(r_i) \quad (Q_{i1}, \dots, Q_{iJ})]'$$

$$\sim MVN(\Lambda Z_i, V_{\gamma}), \tag{18}$$

where Z_i is a $M \times 1$ column vector of physician characteristics, including a first element that has the value 1; and $\Lambda(K \times M \text{ matrix})$ and $V_{\gamma}(K \times K \text{ matrix})$ are parameters.

Thus, the hierarchical model can be specified as

$$\begin{aligned} U_{ijt} \mid I_{ijt}, X_{ijt}, Q_{ijt}, \sigma_{Q_{ijt}}^{2}, r_{i}, \beta_{i}, \Sigma \\ Q_{ijt} \mid Q_{ij(t-1)}, nd_{ijt}, I_{ij(t-1)}, \sigma_{D_{i}}^{2}, \sigma_{F_{i}}^{2}, Q_{ij}, r_{i} \\ \gamma_{i} \mid \Lambda, Z_{i}, V_{\gamma}. \end{aligned}$$

To complete the model, the priors for the parameters are specified as

$$\begin{split} Q_{j0} \sim N(\overline{Q}_0, \theta_0^2), \qquad \Sigma = \begin{pmatrix} \sigma_1^2 & \cdot & \cdot & 0 \\ \cdot & \sigma_2^2 & \cdot & \cdot \\ \cdot & \cdot & \cdot & \cdot \\ 0 & \cdot & \cdot & 1 \end{pmatrix}, \\ \sigma_j^2 \sim \text{Inverse Gamma}(s_{1j}, s_{2j}), \\ \lambda = \text{vec}(\Lambda') \sim N(\overline{\lambda}, V_{\lambda}), \quad V_{\gamma} \sim \text{Wishart}(g, G). \end{split}$$

To make draws from the joint posterior distribution of parameters, we use Gibbs sampling, making draws from the full conditional distributions of subvectors of the parameter vector. Furthermore, we use data augmentation to draw utilities and qualities. Finally, we use the Metropolis-Hastings algorithm to make draws for parameters for which the full conditional distributions are not easily and directly drawn. The full details of the likelihood, the full conditional distributions, and the sampling algorithm are given in the appendix.

5. Identification

Given the structure of our model, it is important that we provide some intuition for the identification of the individual-specific parameters of learning and the separation of the informative and persuasive effects of detailing. In addition, our simulation results (available from the authors on request) showed that our model could recover the parameters with a reasonable degree of accuracy.

The individual-level parameters in the model are the variances of the detailing and feedback signals $(\sigma_{D_i}^2)$ and $\sigma_{F_i}^2$, respectively), the risk aversion parameter r_i , the true mean quality of each drug Q_{ij} , and the linear coefficients β_i . Note that we need observations for multiple new drugs to infer that the learning rates are systematic to the physician. Otherwise, it would be hard to separate from random events like specific realizations of the random error ε_{ijt} , which would give us similar prescription patterns. However, if we observe the patterns of evolution of prescription behavior for the same physician for multiple drugs, we can make inferences about the learning rate and thus about the parameters that summarize this learning rate $(\sigma_{D_i}^2)$ and $\sigma_{E_i}^2$.

The true quality, Q_{ij} , of the drug is identified by the physician's steady-state prescription behavior. We have already seen that as the physician learns, the quality belief, Q_{ijt} , evolves to the true quality of the drug Q_{ij} . At the extreme, i.e., at steady state, the quality belief is the true quality. Thus, we need to observe a time series of prescriptions long enough for the

physicians to be able to correctly identify the true mean quality. For our empirical analysis, the data are observed for about nine months after launch. Hence, the prescription behavior of the physician towards the end of the data set tells us about the true mean quality of the drug. One caveat: Because we cannot test for whether the steady state has been reached, it is an assumption that nine months is adequate to reach steady state. To see the extent to which results are affected, we estimated our model after dropping one observation per physician (a loss of 6% of data per physician on average). We find that our results (available from the authors on request) are largely unchanged.

For the identification of the risk aversion parameter, we first note from Equation (11) that the expected utility of the physician depends not only on the mean of the quality belief, Q_{ijt} , in any time period, but also on its variance, $\sigma_{Q_{iit}}^2$. We have seen earlier that this variance declines monotonically as the physician learns about the drug. It is easy to see from Equation (11) that the expected utility of the physician is inversely related to this variance. In addition, this variance interacted with the risk aversion parameter—the higher the risk aversion, the lower the expected utility for a given value of this variance. The lower the expected utility, the lower the probability of prescribing a drug. Because all physicians are assumed to start with the same quality belief, and because this variance is the same for all physicians, systematic differences in shares of prescribing the new drug in early periods after the introduction would be explained only by differences in risk aversion, all else being equal. Because we observe two new drugs being introduced in our data, the identification is even stronger. Physicians who are more risk averse would have lower initial probability of prescription for every new drug compared to the average physician. Thus, correlation in such behavior across initial periods after the introduction of at least two new drugs is helpful in identifying an individual-specific risk aversion coefficient. Although it can, in principle, be identified without such observations, in our data set, we observe two new introductions, thus strengthening the identification of this parameter.

The variances of the detailing and feedback signals are identified from the evolution patterns of physician prescription behavior as they relate to detailing and feedback signals. We have seen that with every signal, the physician's quality belief is updated. As a result, the mean of this quality belief evolves from the mean of the initial quality belief towards the mean of the final quality belief. Equation (9) summarizes the process of updating of the mean of the quality belief. As the quality belief for a drug evolves, the probability of prescribing that drug also evolves over time. In the

discussion on model specification, we explained how a greater variance of the detailing signal implies a slower learning rate. Thus, the rate of learning helps identify the variance parameter for the detailing signal. A similar argument holds for the variance of the feedback signal.

The linear coefficients β_i are identified from the covariance of the prescription behavior of the physician with the linear variables (detailing stock variable and dummy variable for patient requests). An important concern could be the separate identification of the informative and persuasive effects of detailing. Identification of the persuasive effect, which is the coefficient of the detailing stock variable in the utility (Equation (1)), is aided by the fact that in our data set the incumbent drug-Viagra-existed for about five years before the first observation in the data. Thus, we make the a priori assumption that physicians were fully knowledgable about Viagra, making the informative effect for this drug zero. Any detailing effect of Viagra on the prescription behavior of physicians stems entirely from the persuasive effect. For the new entrants—Levitra and Cialis—both the informative and persuasive effects are present and can be identified.

Another potential concern is the endogeneity in the detailing allocation. If firms optimally decide on individual detailing levels based on a complete knowledge of physicians' behavior, then the detailing would be endogenous and our parameter estimates would be biased. However, because of an institutional feature of the pharmaceutical industry, this may not be a significant concern for the purpose of our study. Generally, firms use a volumetric decile-based rule to decide on their detailing levels to physicians. Physicians are typically classified into deciles based on their total category volumes, and detailing allocations are made at the decile levels (see Manchanda et al. 2004 for a discussion of these allocation rules). We find that this is consistent with the patterns of detailing in our data. Manchanda et al. (2008) also find similar decilebased detailing patterns by firms during launch of a new product.

6. Results

We first present the parameter estimates of the model. In Table 1, we report the estimates for the individual-level model parameters. These parameters are the detailing signal variance $(\sigma_{D_i}^2)$, the feedback signal variance $(\sigma_{F_i}^2)$, the absolute risk aversion (r_i) , the coefficients for the detailing stock (β_{1i}) and patient requests (β_{2i}) , and the true mean qualities for Cialis (Q_{1i}) and Levitra (Q_{2i}) . In our Bayesian inference, we obtain a distribution for each individual-level parameter for each physician. We compute the mean parameter value for each physician and then report the

Table 1 Individual-Level Parameter Estimates

Parameter Mea	
Detailing signal variance $\sigma_{D_i}^2$ 1.05	67 0.1519
Feedback signal variance $\sigma_{F_i}^2$ 1.09 Absolute risk aversion r_i 0.00 Coefficient—detailing stock β_{1i} 0.61 Coefficient—patient request β_{2i} 1.56 True mean quality—Cialis Q_{i1} 1.00 True mean quality—Levitra Q_{i2} 0.99	45 0.0347 13 0.1040 95 0.0611 99 0.0901

Notes. Because these parameters are at the individual level, for each physician, the parameter has a mean and a standard deviation. The reported parameters are the mean and standard deviation of the physician-specific parameter means.

mean and standard deviation across physicians of this individual-level mean parameter value in Table 1.

The estimate for detailing signal variance is 1.0567. This parameter value implies that it takes about 9.5103 detailing calls for the uncertainty of a physician to reduce to one-tenth of its initial value. This is an estimate of the informative effect of detailing. Similarly, the parameter estimate of the feedback signal variance is 1.0986, which corresponds to 9.8874 feedback signals to reduce the uncertainty to onetenth of its initial value. This also suggests that an average detailing call is somewhat more informative than an average past prescription because it requires a smaller number of detailing calls than feedback signals to reduce the physician's uncertainty about drug quality.4 This is consistent with the findings of prior research (cf. Narayanan et al. 2005). The standard deviations for these estimates suggest that there is considerable heterogeneity across physicians in these parameters. We return to a discussion of heterogeneity in these parameters later in this section.

The parameter estimates for the coefficient of the linear detailing stock (β_{1i}) suggest that there is a positive persuasive effect of detailing. Thus, even after a physician's uncertainty about the drug is reduced, there is still a positive effect of detailing. There is also a strong positive effect of patient feedback, as indicated by the parameter estimates for the coefficient for patient requests (β_{2i}). To the best of our knowledge, this is the first time that the effect of patient requests has been quantified. Given the data description earlier, it is clear that the incidence of patient requests is very low at about 15%, but if a patient makes a request, it has a strong effect on the physician's prescription behavior.

The parameter estimates for the mean true qualities for Cialis and Levitra are both close to one (the true

⁴ Note that this conclusion is not invariant to the number of patient feedback signals per past prescription. Hence we compare the degree of information in a detailing call and in a past prescription, as opposed to that in a detailing call and in a patient's feedback.

Table 2 Pooled Parameter Estimates

Parameter	Mean	Std. dev.
Utility error variance	5.7057	0.9012
Utility error variance	5.4010	0.5695

Notes. Other pooled parameters include the elements of the matrix V_{γ} and the vector λ . These are not reported here for the sake of brevity.

quality of Viagra, which we fix for identification purposes), suggesting that the qualities of the three drugs are similar, on average, although they may differ significantly at the individual physician level. Cialis is, on average, marginally of higher quality than Viagra; Levitra is of marginally lower quality than Viagra. This is consistent with industry reports that Cialis is a higher quality drug. Note that in steady state, the share of prescriptions of the drug with the highest true quality would be largest, all else being equal.

In Table 2, we report the parameter estimates for the pooled parameters. The pooled parameters in this model are the variances of the normal errors in the utility function (σ_1^2 and σ_2^2). The additional pooled parameters include those that relate the individual-level parameters to physician demographics, i.e., λ , and these are reported in Table 3. The main conclusion from Table 3 is that while observed demographics have significant effects on the detailing stock coefficient, they are mostly not significant in the case of the other parameters.

Next, we move to the heterogeneity in the individual-level parameter estimates. Figure 1 depicts the histograms of the individual-level parameters across physicians. Figures 1(a) and 1(b), respectively, show the histograms of the detailing and feedback signal variances. These figures suggest that there is considerable heterogeneity in these parameters across

physicians. There is much greater heterogeneity in the detailing signal variance than in the feedback signal variance. Figure 1(c) shows the distribution of the risk aversion parameter. Physicians are fairly heterogeneous in their risk aversion levels as well. In the next section we assess the economic significance of heterogeneity in learning and risk aversion.

We find from Figure 1(d) that for all physicians the mean of the detailing stock coefficient is positive. Furthermore, for the majority of these physicians, the 95% credible intervals of the individual level parameter do not include zero. Thus, we show that the detailing stock has a positive effect on their choice probabilities of the detailed brand. Figure 1(e) shows the distribution across physicians of the patient request coefficient. It is positive for all physicians and the 95% credible interval does not cross zero for any physician.

In Figures 1(f) and 1(g), we show the distributions across physicians of the true mean qualities of Cialis and Levitra, respectively. Once again, on average, Cialis has a higher quality than Viagra and Levitra a lower quality than Viagra. However, because there is considerable heterogeneity in the true quality levels, for a particular physician, it is not necessary that this rank ordering of drugs be maintained.

Regarding heterogeneity, a valid concern could be whether the individual-level parameters are significantly different from each other. One could compare these parameters for each pair of physicians to see if they differ in statistical terms. We present a more informal analysis of the heterogeneity of these parameters across physicians. In Table 4, we compare the across-physician and within-physician standard deviations of these parameters. If the across-physician standard deviation for a parameter is smaller than or similar to the within-physician standard deviation, it would

Table 3 Demographic Heterogeneity Parameters (λ)

Parameter		ailing /ariance		ck signal ance	Diek a	version		quality alis		quality vitra		ng stock ficient		request icient
- arailleter	Signal	/allallut	Vall	ance	nisk a	VE121011	Ui	2115	LG/	rilla	COGII	ICICIIL	COGII	ICIGIII
Intercept	1.0492	(0.0268)	1.1062	(0.2399)	0.0532	(0.0061)	1.0220	(0.0160)	0.9944	(0.0179)	0.5887	(0.0183)	1.5630	(0.0108)
Specialty—GP	-0.0050	(0.0217)	-0.0172	(0.0194)	-0.0050	(0.0050)	0.0003	(0.0129)	0.0007	(0.0145)	-0.0174	(0.0148)	-0.0009	(0.0087)
Specialty—urologist	-0.0393	(0.0306)	-0.0384	(0.0274)	-0.0018	(0.0070)	-0.0152	(0.0182)	-0.0030	(0.0205)	-0.0145	(0.0209)	-0.0180	(0.0123)
Decile 1	0.0120	(0.0252)	0.0129	(0.0226)	-0.0027	(0.0058)	-0.0091	(0.0150)	0.0044	(0.0169)	0.0524	(0.0172)	0.0110	(0.0101)
Decile 2	-0.0212	(0.0242)	-0.0203	(0.0217)	-0.0038	(0.0055)	-0.0098	(0.0145)	0.0062	(0.0162)	0.0562	(0.0165)	0.0032	(0.0098)
Decile 3	-0.0068	(0.0252)	-0.0075	(0.0226)	-0.0032	(0.0058)	-0.0337	(0.0150)	-0.0082	(0.0169)	0.0470	(0.0172)	0.0031	(0.0101)
Decile 4	0.0179	(0.0214)	0.0112	(0.0192)	-0.0072	(0.0049)	-0.0090	(0.0128)	0.0045	(0.0144)	0.0538	(0.0146)	0.0052	(0.0086)
Decile 5	0.0102	(0.0255)	0.0080	(0.0229)	-0.0116	(0.0058)	-0.0082	(0.0152)	0.0030	(0.0171)	0.0535	(0.0174)	0.0181	(0.0103)
Decile 6	0.0165	(0.0229)	0.0059	(0.0205)	-0.0009	(0.0052)	-0.0093	(0.0137)	0.0039	(0.0154)	0.0421	(0.0156)	0.0064	(0.0092)
Decile 7	0.0137	(0.0213)	0.0090	(0.0191)	-0.0054	(0.0049)	-0.0235	(0.0127)	0.0120	(0.0143)	0.0429	(0.0145)	0.0082	(0.0086)
Decile 8	0.0318	(0.0243)	0.0179	(0.0218)	-0.0135	(0.0056)	-0.0152	(0.0145)	-0.0006	(0.0163)	0.0315	(0.0166)	0.0091	(0.0098)
Decile 9	0.0536	(0.0210)	0.0474	(0.0188)	0.0026	(0.0048)	-0.0009	(0.0125)	0.0162	(0.0140)	0.0272	(0.0143)	0.0225	(0.0084)

Note. The numbers within the parentheses are the posterior standard deviations.

suggest that the 95% credible intervals for the physicians overlap, and thus they are not significantly different from each other in terms of that parameter. On the other hand, if the across-physician standard deviation is larger than the within-physician standard deviation, it would provide greater support for the claim that individual-level parameters are significantly different for different physicians.

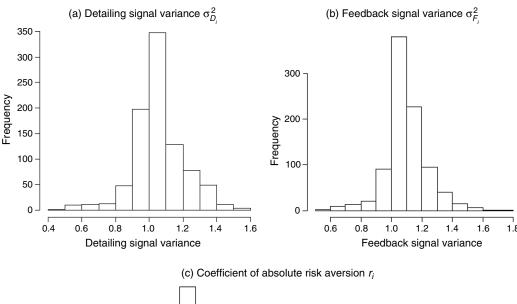
We find that the across-physician variation is much higher than the within-physician variance in the case of the two signal variances. Because the heterogeneity in the detailing signal variance summarizes the heterogeneity in learning as discussed in §4.2, we conclude that there is a significant amount of heterogeneity in learning across physicians.

The coefficient of the detailing stock also has a higher across-physician standard deviation than within-physician standard deviation. Thus, there is a significant amount of heterogeneity in this parameter as well. The same is the case for the true mean qualities of Cialis and Levitra. Regarding the risk aversion parameter, however, and for the coefficient for patient requests, the within-physician standard deviation is larger than the across-physician standard deviation, suggesting that physicians do not significantly differ in these parameters.

To explore the heterogeneity in learning across physicians even further, we plot a histogram of the number of detailing calls required to reduce the physician's uncertainty about a new drug to one-tenth its initial value. This is computed using the parameter estimates of the detailing signal variance for each physician. Figure 2, which shows this plot, suggests that heterogeneity in the detailing signal variance parameter indeed manifests itself in significant heterogeneity in the number of calls required to reduce the physicians' uncertainty.

An interesting pattern in the parameter estimates is the negative correlation between the informative and persuasive effects of detailing for physicians. Physicians who have a high informative effect of detailing are likely to have a low persuasive effect and vice





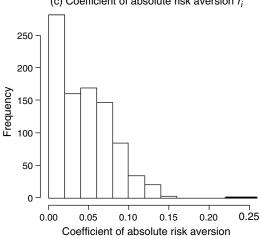
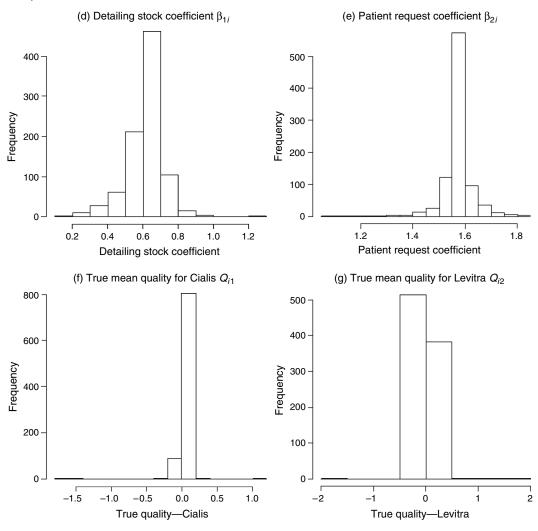


Figure 1 (Cont'd.)



versa. The informative effect of detailing is summarized by the detailing signal variance. A high informative effect is equivalent to saying that the learning rate for the physician is high. In the parameter estimates,

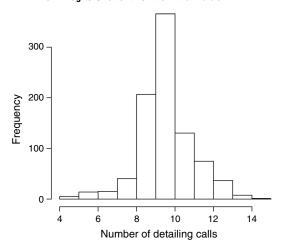
Table 4 Across-Physician vs. Within-Physician Standard Deviations of the Individual-Level Parameters

		Across-physician	Within-physician
Parameter		standard deviation	standard deviation
Detailing signal variance	$\sigma_{D_i}^2$	0.1519	0.1044
Feedback signal variance	$\sigma_{F_i}^2$	0.1358	0.1286
Absolute risk aversion	r_i	0.0347	0.1284
Coefficient—detailing stock	$\dot{\beta}_{1i}$	0.1040	0.0313
Coefficient—patient request	β_{2i}	0.0611	0.0839
True mean quality—Cialis	Q_{i1}	0.0901	0.0529
True mean quality—Levitra	Q_{i2}	0.1009	0.0432

Notes. Across-physician standard deviation: The mean parameter value for each physician is first computed, and then the standard deviation of these mean values is reported. Within-physician standard deviation: The within-physician standard deviation of the parameter is computed for each physician, and then the mean of these standard deviations is reported.

this manifests itself as a low value of the detailing signal variance. Similarly, a low informative effect manifests itself in a high detailing variance. The persuasive effect is summarized by the coefficient of the detailing stock variable. Thus, a physician for whom the value of this coefficient is low would have a low persuasive effect and vice versa. Therefore, a negative correlation between the informative and persuasive effects of detailing implies a positive correlation between the detailing signal variance and the detailing stock coefficient. In Figure 3, we plot the detailing signal variance for each physician against the detailing stock coefficient to show this positive correlation between the parameters and, consequently, the negative correlation between the informative and persuasive effects. In Figure 4, we plot the detailing signal variance against the risk aversion parameter to assess whether these parameters are correlated. As can be seen in Figure 4 (and verified by a regression), the risk aversion coefficient is not significantly correlated with the detailing signal variance.

Figure 2 Histogram of the Number of Detailing Calls Required to Reduce Uncertainty (Variance of Quality Belief) About a New Drug to One-tenth of the Initial Value



Finally, we compare our model with a series of null models: (1) a random-coefficient logit model with state dependence; (2) the full model with the persuasive effect, but no learning; (3) the full model with learning, but no persuasive effect; (4) the full model with a common learning rate across physicians for detailing and feedback; (5) the full model with heterogeneous learning rates on feedback but a common learning rate for detailing; and (6) the full model with heterogeneous learning rates on detailing but a common learning rate for feedback. As can be seen from Table 5, our model performs better on all five criteria—likelihood, Bayes factor, out-of-sample likelihood, within-sample hit rates, and out-of-sample hit rates. Because learning plays a particularly significant role in early periods after the introduction of a new product, we should expect to see even better hit rates for our model in such early periods, relative to models that do not have learning. We report the hit rates for the first

Figure 3 Plot of Informative Effect vs. Persuasive Effect

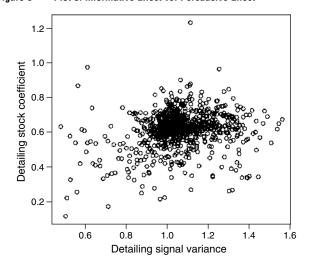
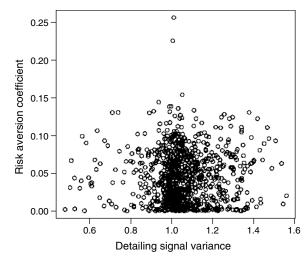


Figure 4 Plot of Informative Effect vs. Risk Aversion



three months of the data and find that our model performs considerably better than the null models without learning.

We can also gain some insights by comparing the fit statistics of the various null models. We see that the in-sample hit rates for the first three months jump significantly between null model 2 and null model 4. This is intuitive because model 4 has learning while model 2 does not, and learning helps explain the early evolution of prescription patterns. However, the fit statistics of null models 4 through 6 are comparable, while those for the full model are much higher. The main insight is that heterogeneity in learning, both through detailing and feedback, results in a big improvement in fit. Note, however, this is not achieved through adding heterogeneity in learning through either detailing or feedback. To understand this better, we look at the correlation between detailing and feedback signal variances at the individual level and find that they are negatively correlated, with a correlation coefficient of -0.63. This suggests that gains in fit are likely to be achieved only when both individual level parameters are allowed to be heterogeneous. Otherwise, the model is trying to summarize two cross-sectional heterogeneity distributions that are negatively correlated via just one distribution.5

7. Managerial Implications

The negative correlation between the informative and persuasive effects has implications for the allocation of detailing efforts across physicians and over time. To illustrate this, let us consider a situation with two physicians, one with a high informative effect and a

⁵ We thank an anonymous reviewer for bringing this to our attention.

Table 5 Comparison with Null Models

				Model			
Measure	Full model	Null model 1	Null model 2	Null model 3	Null model 4	Null model 5	Null model 6
Log marginal likelihood	-5,186.7	_	-5,807.8	-5,792.1	-5,517.2	-5,370.0	-5,382.1
Bayes factor (wrt. full model)*	_	_	447.1	547.6	79.9	120.6	132.7
Log marginal likelihood—out of sample	-398.7		-476.12	-421.8	-418.4	-408.1	-409.8
Hit rate—in sample (%)	46	29	37	39	40	40	41
Hit rate—out of sample (%)**	38	_	35	36	36	36	37
Hit rate—in sample (3 months) (%)	52	31	31	42	42	43	44

Notes. Null model 1: Random Coefficient Logit model with state dependence.

Null model 2: The full model with the persuasive effect of detailing, but the learning process removed.

Null model 3: The full model with learning, but with no persuasive effect of detailing.

Null model 4: The full model with homogenous learning, i.e., no heterogeneity on either the detailing signal variance or the feedback signal variance

Null model 5: The full model with homogenous detailing signal variance, but heterogeneous feedback signal variance.

Null model 6: The full model with homogenous feedback signal variance, but heterogeneous detailing signal variance.

low persuasive effect (a fast learner), and the other with the opposite (a slow learner). For both these physicians, the informative and persuasive effects are present in the introductory phases of the new drug's life cycle. In later stages, only the persuasive effect plays a role. For the fast learner, the total effect of detailing is initially very high, but reduces rapidly until it converges to the persuasive effect, where it then remains constant. For the slow learner, all else being the same, the total effect again starts at a high level (although perhaps not as high as for the fast learner), but falls more slowly. It then converges to a persuasive effect that is higher than for the fast learner. Because this total effect denotes the responsiveness of the physician to detailing and the optimal detailing level depends on responsiveness, it would be optimal for firms to allocate higher amounts of detailing initially to the fast learner, but then to rapidly reduce the allocation. By contrast, for the slow learner, we might expect to see a different rate at which optimal detailing allocation is reduced.

We have also seen that the informative and persuasive effects are related to the decile of the physician. Specifically, physicians in higher deciles have a lower persuasive effect, while those in lower deciles have a higher persuasive effect. Optimally, then, firms would allocate a high proportion of resources to physicians in high deciles in the early stages after the introduction of the drug and then reduce this proportion over time. However, discussions with industry

practitioners reveal that firms most commonly use a decile-based rule, which remains constant over time. We find the same patterns in our data as well, with detailing for every decile remaining approximately constant over the entire period in our data set.

We conduct three counterfactual simulations to see how firms could increase their revenues if they took into account learning by physicians and heterogeneity in this learning. We conduct separate simulations for each of the two new drugs—Levitra and Cialis one by one. We keep the total amount of detailing by firms in the first three months after the launch of the respective drug as fixed. We only alter the allocation of detailing across physicians or over time and then compute the predicted expected revenues. In all these simulations, detailing calls are rounded off; i.e., a physician can only get an integer number of calls. All calls are assumed to be made in the beginning of the month so as to abstract away from the problem of timing of detailing calls within a month. Competitors' detailing is kept unchanged. Optimal allocations are obtained using a numerical optimization routine. To keep the optimization feasible, we conduct all simulations for a subset of 100 physicians randomly chosen from the sample. We then compare the predicted revenues in the counterfactual case with those from the actual allocation plans.

In simulation 1, we change only the temporal allocation of detailing, but keep the cross-sectional allocation unchanged; i.e., we vary the allocation of

^{*}A Bayes factor greater than 1 favors the full model. Bayes factor is relative to the recomputed full model with one observation per physician dropped.

^{**}The out-of-sample hit rate for the random coefficients model with state dependence is not computed as it involves the loss of two observations per physician, making it hard to compare with other models. Also, we have not reported the likelihood and Bayes factors for the same reason.

detailing to each of the three months in the launch quarter, but do not vary the proportions of detailing within that month to individual physicians. Thus, this is a two-dimensional optimization exercise with the unknown variables being the allocation for the first two months; the allocation for the third month is automatically known because the proportions add up to one. We find that by varying the month-to-month allocations of detailing, Levitra achieves revenue gains of 5.9%, on average, compared to the current allocation plan, while Cialis achieves an even higher gain of 8.3%, on average. These revenue gains arise from frontloading detailing to the period immediately after launch during which time all physicians have both an informative effect and a persuasive effect and are hence most responsive to detailing. As physicians learn about the drug, the informative effect asymptotes down to zero, leaving only the persuasive effect. For all physicians, the responsiveness to detailing reduces over time. This result is similar to that reported in Narayanan et al. (2005). Note that the structure imposed by Bayesian learning causes the informative effect to decrease. Although it may seem as if the frontloading of detailing is driven by structure alone, the empirical question that our estimates help to answer is the rate at which the informative effect reduces over time and therefore the extent of frontloading.

In simulation 2, we change the cross-sectional allocation of detailing, but keep the temporal allocation unchanged. Thus, we keep the total amount of detailing within each month fixed, but vary how much of that is allocated to each of the physicians. In each month, we have an independent optimization, with the dimension of the unknown vector being one fewer than the number of physicians (i.e., 99 because the simulation was conducted for 100 physicians). We find that relatively modest revenue increases can result through this exercise, 4.7% on average for Levitra and 5.8% on average for Cialis. The lower revenue increases could mean that firms are using some decile-based rule for allocation. Although this is not optimal, it does inherently take into account heterogeneity across physicians because the informative and persuasive effects of detailing are explained to some extent by decile. Finally, in simulation 3, we allow both the temporal and cross-sectional allocation of detailing to change; i.e., we find the detailing level for each physician for each month that maximizes revenues. This gives us a revenue increase of 10.6%, on average, for Levitra and 14.1%, on average, for Cialis.

The results of these three counterfactual simulations are summarized in Table 6. To summarize, it appears that firms could significantly increase their revenues during the launch period by reallocating existing expenditure on detailing.

Table 6 Counterfactual Simulations: Revenue Gains Through Reallocation of Detailing

Cross-sectional	Temporal allocation				
allocation	No change	Change			
No change	Current situation	Simulation 1 Cialis: 8.3% (3.20) Levitra: 5.9% (2.74)			
Change	Simulation 2 Cialis: 5.8% (1.36) Levitra: 4.7% (2.19)	Simulation 3 Cialis: 14.1% (2.78) Levitra: 10.6% (2.51)			

Notes. The reported estimates are the posterior mean revenue gains, and the figures in parentheses are the posterior standard deviations of these revenue estimates, obtained by conducting these simulations for 30 draws from the joint posterior distribution of all parameters.

Although these counterfactual simulations give us a sense of the revenue gains that firms could achieve by taking heterogeneity into account, the extent to which these gains arise because of heterogeneity in learning, in risk aversion, and in the persuasive effect is not yet obvious. To assess the contribution of heterogeneity in these three parameters on revenue gains, we sequentially repeat simulation 3 taking into account heterogeneity for each parameter while fixing the other two at the population means. We then compute the revenue gains in the three simulations described earlier. Results of this exercise (Table 7) suggest that 50%–56% of the revenue gains in simulation 3 (where detailing is reallocated both temporally and cross-sectionally) come from accounting for heterogeneity in learning. About 9%–10% of the gains come from accounting for heterogeneity in the risk aversion parameter, while the remaining 35%-41% comes from accounting for heterogeneity in the persuasive effect.

8. Conclusion

We began with a problem of optimal allocation of marketing communication for new products. Our

Table 7 Revenue Gains from Reallocation of Detailing: Relative Contributions of Heterogeneity in Learning, Risk Aversion and Persuasive Effect

	Percent contribut	Percent contribution of revenue gain by heterogeneity in						
Drug	Learning	Risk aversion	Persuasive effect					
Cialis Levitra	49.78% (11.36) 55.93% (13.11)	9.60% (7.74) 9.27% (7.61)	40.62% (8.49) 34.80% (8.37)					

Notes. The reported numbers are the posterior mean relative contributions, and the figures in parentheses are the posterior standard deviations of these relative contribution. They are obtained by taking 30 draws from the joint posterior distribution of all parameters, fixing the nonfocal parameters (e.g., for the contribution from heterogeneity in learning, the nonfocal parameters are risk aversion and the persuasive effect) at their posterior means, and conducting the simulations for each draw. The simulation estimates are reported as a percentage of the revenue gains for the simulations without any constraints on the parameters.

focus was on allocation of these resources over time and across consumers. Heterogeneous learning was recognized as a factor that would affect the temporal as well as cross-sectional allocation of these resources. We specified a structural model of heterogeneous learning and developed a methodology to estimate such a model at the individual physician level. We estimated this model using a unique panel data set consisting of physician prescriptions and detailing calls. We allowed for detailing to have both an informative and a persuasive effect and estimated these effects at the individual physician level. We then conducted a set of counterfactual simulations to find the implications of heterogeneous learning for optimal allocations of detailing over time and across agents (physicians, in this case).

Our parameter estimates indicate that there is considerable heterogeneity across physicians in terms of learning rates. Some physicians require only a few detailing calls to substantially reduce their uncertainty about a new drug. Others require many repeated detailing calls to reduce their uncertainty to the same extent. Physicians also differ significantly in the persuasive effect of detailing, which is the only effect present once they are fully knowledgeable about the drug. Because of both these effects, however, there is a significant amount of variation across physicians in terms of how their responsiveness to detailing varies over time.

We also find that volume based deciles explain the variation in the persuasive effects of detailing to some extent. Specifically, physicians who are heavy prescribers in the category of erectile dysfunction are more likely to have a low persuasive effect. Thus, their responsiveness to detailing reduces rapidly to a low level. The responsiveness of light prescribers reduces more slowly and settles at a relatively high responsiveness after they have learned about the drug.

We conducted three counterfactual simulations to see if firms could increase their revenues in this category by changing their detailing allocation patterns. By changing just their temporal allocation without changing the cross-sectional allocation of detailing, firms obtain a 5.9 to 8.3% increase in revenues in the first three months after launch. This reflects gains from front-loading their detailing to early periods after the launch of the drug. By changing their cross-sectional allocation without altering the temporal allocation, firms obtain a more modest 4.7% to 5.8% increase in revenues. Changing both temporal and cross-sectional allocations yields a substantial 10.6% to 14.1% increase in revenues. Accounting for heterogeneity in learning accounts for 50%–56% of these gains, while accounting for heterogeneity in risk aversion accounts for about 9%-10%; the remaining 35%–41% comes from accounting for heterogeneity in the persuasive effect.

Finally, we list some of the limitations of this study. In specifying this model of learning about new drugs, we have assumed away other potentially important sources of learning, for instance, learning from other physicians. This assumption of no learning through other sources is based on the absence of appropriate data and might overstate the degree of learning through detailing that we infer. We do not directly include the effect of free samples, which could be another source of learning. However, our learning through patient feedback includes the effect of samples because we include occasions when the physician gives only a free sample to the patient (and does not write a prescription) when we count the number of feedback signals.

In ignoring the effect of other marketing instruments, we might be overstating the effect of detailing if these other instruments are positively correlated with detailing. However, it must be kept in mind that detailing is by far the primary form of marketing in prescription drug categories. This is especially true for the category we study during the period of the data. Furthermore, it is the primary marketing instrument that can be allocated differentially across physicians (unlike other prominent marketing instruments like DTC and journal advertising).

As mentioned earlier, another potential concern could be endogeneity in detailing allocation. Although there are reasons to believe that this may not be a big concern for the specific category we study, it could be addressed by including a detailing supply equation in the model and jointly estimating parameters of demand and supply. A complication that would arise is that the detailing supply equation cannot be static because of the presence of learning. Learning causes persistent effects in detailing over time. Thus, firms are likely to take into account the effect of detailing on the future prescription behavior of physicians. This would complicate the problem substantially. We also assume away any forward-looking behavior of physicians. If they are aware that they learn through detailing, physicians may be more willing to see detailers early and thus learn about the drug more quickly. Finally, firms may also be interested in determining the optimal level and the allocation of detailing. Our counterfactual analysis focuses only on allocation keeping the amount of detailing fixed. These are challenging problems, and the methodology to account for these phenomena is not yet fully developed. Future research could address these questions.

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Appendix: Full Conditional Distributions

Given the model described, the joint posterior distribution of all the parameters conditional on the data is given by the following expression:

$$L \propto \prod_{i=1}^{N} \left\{ \prod_{t=1}^{T_{i}} \left[f(U_{it} \mid \{I_{ijt}\}, \{X_{ijt}\}, \{Q_{ijt}\}, r_{i}, \beta_{i}, \Sigma) \right. \right. \\ \left. \cdot \prod_{j=1}^{J} \left[f(Q_{ijt} \mid Q_{ij(t-1)}, nd_{ijt}, I_{ij(t-1)}, \sigma_{D_{i}}^{2}, \sigma_{F_{i}}^{2}, Q_{ij}, Q_{j0}) \right] \right] \\ \left. \cdot f(\gamma_{i} \mid \Lambda, Z_{i}, V_{\gamma}) \right\} \cdot f(\lambda \mid \bar{\lambda}, V_{\lambda}) f(V_{\gamma} \mid g, G) \prod_{i=1}^{J} \left[f(\sigma_{j}^{2} \mid s_{1j}, s_{2j}) \right].$$

We next derive the full conditional distributions of all the parameters (and augmented parameters like U_{ijt} and Q_{iit}), so that we can use a Gibbs sampling method. For those parameters for which the full conditional distributions are not from known distribution families, we use the Metropolis-Hastings algorithm to draw from the respective full conditional distributions.

Suppose we define

$$U_{it} = \begin{pmatrix} U_{i1t} \\ \cdot \\ U_{iJt} \end{pmatrix}, \qquad \Omega_{it} = \begin{pmatrix} -\exp\left(-r_i Q_{i1t} + \frac{r_i^2 \sigma_{Q_{i1t}}^2}{2}\right) \\ \cdot \\ -\exp\left(-r_i Q_{iJt} + \frac{r_i^2 \sigma_{Q_{iJt}}^2}{2}\right) \end{pmatrix},$$

$$X_{it} = \begin{pmatrix} X_{i1t} \\ \cdot \\ X_{iJt} \end{pmatrix}.$$

The full expression for the joint posterior distribution can then be written as

$$L \propto \prod_{i=1}^{N} \left\{ \prod_{t=1}^{T_i} \left[|\Sigma|^{-1/2} \exp\left(-\frac{1}{2} (U_{it} - \Omega_{it} - X_{it}\beta_i)' \Sigma^{-1} (U_{it} - \Omega_{it} - X_{it}\beta_i) \right) \right. \right. \\ \left. \cdot \prod_{i=1}^{J} \left[1(U_{ijt} > \max(U_{ikt}), k \neq j) \right]^{I_{ijt}} \right.$$

$$\begin{split} \cdot \prod_{j=1}^{J} \left[\frac{1}{\sigma_{\mathrm{Q}ijt}^{2} \sqrt{\frac{nf_{ijt}}{\sigma_{F_{i}}^{2}} + \frac{nd_{ijt}}{\sigma_{D_{i}}^{2}}}} \\ \cdot \exp \left(-\frac{1}{2} \frac{\left[Q_{ijt} - \frac{\sigma_{\mathrm{Q}ijt}^{2}}{\sigma_{\mathrm{Q}ij(t-1)}^{2}} Q_{ij(t-1)} - \sigma_{\mathrm{Q}ijt}^{2} Q_{ij} \left(\frac{nf_{ijt}}{\sigma_{F_{i}}^{2}} + \frac{nd_{ijt}}{\sigma_{D_{i}}^{2}} \right) \right]^{2}}{\left(\frac{nf_{ijt}}{\sigma_{F_{i}}^{2}} + \frac{nd_{ijt}}{\sigma_{D_{i}}^{2}} \right) \sigma_{\mathrm{Q}ijt}^{4}} \right] \\ \cdot |V_{\gamma}|^{-1/2} \exp \left(-\frac{1}{2} (\gamma_{i} - \Lambda Z_{i})' V_{\gamma}^{-1} (\gamma_{i} - \Lambda Z_{i}) \right) \right\} \\ \cdot |V_{\lambda}|^{-1/2} \exp \left(-\frac{1}{2} (\lambda - \bar{\lambda})' V_{\lambda}^{-1} (\lambda - \bar{\lambda}) \right) \\ \cdot \frac{V_{\gamma}^{(g-k-1)/2}}{|G|^{g/2}} \exp \left(-\frac{1}{2} tr(G^{-1} V_{\gamma}) \right) \cdot \prod_{j=1}^{J-1} \left[\frac{\exp(-1/s_{2j}\sigma_{j}^{2})}{\sigma_{i}^{2(s_{1j}+1)}} \right]. \end{split}$$

From this joint posterior, we can derive the full conditional distributions as follows:

$$\begin{array}{l} 1. \ \ \sigma_{j}^{2} \ | \ \{U_{ijt}\}, \{X_{ijt}\}, \{Q_{ijt}\}, \{\beta_{i}\}, \{r_{i}\} \sim IG(\sum_{i=1}^{N} T_{i}/2 + s_{1j}, \\ 2s_{2j}/(2 + s_{2j} \sum_{i=1}^{I} \sum_{t=1}^{T_{i}} (U_{ijt} + \exp(-r_{i}Q_{ijt} + r_{i}^{2}\sigma_{Q_{ijt}}^{2}/2) - X_{ijt}\beta_{i})^{2})) \\ 2. \ \ V_{\gamma} \ | \ \ \gamma_{i}, \Lambda, Z_{i}, g, G \sim \text{ Inverse Wishart } (g + N, \\ [\sum_{i=1}^{N} [(\gamma_{i} - \Lambda Z_{i})(\gamma_{i} - \Lambda Z_{i})'] + G^{-1}]^{-1}) \\ 3. \ \ \lambda \ | \ \{\gamma_{i}\}, V_{\gamma}, Z, V_{\lambda}, \bar{\lambda} \sim N([V_{\gamma}^{-1} \otimes Z'Z + V_{\lambda}^{-1}]^{-1}[V_{\gamma}^{-1} \otimes Z'Z + V_{\lambda}^{-1}]^{-1}), \\ Z'Z\hat{\lambda} + V_{\lambda}^{-1}\bar{\lambda}], [V_{\gamma}^{-1} \otimes Z'Z + V_{\lambda}^{-1}]^{-1}), \\ \text{where} \end{array}$$

$$\hat{\lambda} = \operatorname{vec}\left[\left(Z'Z \right)^{-1} Z'\Gamma \right], \qquad Z = \begin{pmatrix} Z'_1 \\ \cdot \\ Z'_N \end{pmatrix}, \qquad \Gamma = \begin{pmatrix} \gamma'_1 \\ \cdot \\ \gamma'_N \end{pmatrix}.$$

4. Each physician is independent conditional on the X and Z matrices. Each observation for the physician is independent conditional on the vector of Q_{iit} . Thus, we can draw the latent utilities for a particular physician and a particular observation separately from the other observations. This involves sequentially drawing from a truncated multivariate normal distribution for each time period.

$$U_{it} = \begin{pmatrix} U_{i1t} \\ \vdots \\ U_{iJt} \end{pmatrix}, \qquad \Omega_{it} = \begin{pmatrix} -\exp\left(-r_{i}Q_{i1t} + \frac{r_{i}^{2}\sigma_{Q_{i1t}}^{2}}{2}\right) \\ \vdots \\ -\exp\left(-r_{i}Q_{iJt} + \frac{r_{i}^{2}\sigma_{Q_{iJt}}^{2}}{2}\right) \end{pmatrix}, \qquad \begin{bmatrix} U_{i1t} \\ \vdots \\ U_{iJt} \end{bmatrix} \sim Truncated \ MVN \begin{pmatrix} \left(-\exp\left(-r_{i}Q_{i1t} + \frac{r_{i}^{2}\sigma_{Q_{i1t}}^{2}}{2}\right) + X_{i1T}\beta_{i} \\ \vdots \\ -\exp\left(-r_{i}Q_{iJt} + \frac{r_{i}^{2}\sigma_{Q_{iJt}}^{2}}{2}\right) + X_{iJT}\beta_{i} \end{pmatrix}, \Sigma \end{pmatrix},$$

with the truncation such that $U_{ijt} > U_{ikt}$, $\forall k \neq j$, $I_{ijt} = 1$.

5. The full conditional distributions for the individual level parameters, γ_i and the quality means Q_{i1t} are not from known families of distributions. Hence, draws from the distribution of these parameters for each individual physician are obtained using the Metropolis-Hastings algorithm. We use a random walk Metropolis-Hastings algorithm (Chib and Greenberg 1995) with a normal candidate density to make these draws. The variances of these densities were obtained from the hessian of the pooled maximum likelihood estimates for these parameters, which was scaled up to obtain the best numerical efficiency.

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