A Structural Model of Correlated Learning and Late-Mover Advantages: The Case of Statins

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Abstract

When Lipitor entered the statin (a class of anti-cholesterol drugs) market in 1997, some incumbent drugs had already obtained strong clinical evidence to show their efficacy in preventing heart diseases. However, despite its lack of such important evidence, Lipitor quickly became the most commonly used statin among new patients. To explain this puzzle, we propose a theory of correlated learning and indirect inference by physicians. We introduce a concept of “efficiency ratio”, which measures how efficiently a drug can convert reduction in cholesterol levels to reduction in heart disease risks. We assume physicians are uncertain about drugs’ efficiency ratios, and allow the physicians’ initial prior belief to be correlated across drugs. With correlated prior perceptions, a new clinical trial’s information on a drug’s efficiency ratio can update physicians’ belief on other statins’ efficiency ratios. Physicians then infer each statin’s ability in reducing heart disease risks based on its perceived efficiency ratio and its ability in reducing cholesterol. Consequently, correlated learning may allow late entrants to gain late-mover advantages by free-riding on the clinical evidence and informative marketing activities of the incumbents.

To estimate our model, we use a data set on market shares, patients’ switching rates and discontinuing rates, as well as detailing and media coverage from 1993 to 2004. Our estimation results shows that correlated learning about statins’ efficiency ratios is strong. This, together with the fact that two late entrants, Lipitor and Crestor, are more effective in lowering cholesterol levels, allow them to gain late-mover advantages. Moreover, we find that intensive detailing efforts (via its informative and persuasive roles) also contribute to their successes.

Keywords: Correlated Learning, Late-mover Advantages, Switching Costs, Clinical Trials, Detailing
1 Introduction

Even a few years after a drug is introduced to the market, uncertainty for the drug often remains and deter physicians from prescribing it (Lasser et al., 2002). To reduce this uncertainty, it is quite common for pharmaceutical firms to invest in post-marketing clinical studies. Since pharmaceutical firms are only allowed to make claims which are supported by scientific evidence, these post-marketing clinical trial results can be very important to firms’ marketing strategies. For example, statin is the most popular class of anti-cholesterol drugs and most patients take statins to lower their cholesterol levels, hoping that it will reduce their heart disease risks. However, before a clinical study on reducing heart disease risks becomes available, a drug company can make a direct claim only on the efficacy in lowering cholesterol levels. Although a positive correlation between high cholesterol levels and coronary heart disease risks has been found in medical research, a drug that can lower cholesterol levels effectively does not necessarily reduce heart disease risks. This is because it might have some unknown side-effects that could raise the heart disease risks and counter its benefits of lowering cholesterol levels. To make a claim that their drugs are effective in reducing heart disease risks, statin manufacturers have invested in post-marketing clinical trials to provide such direct evidence. Very often, however, getting post-marketing clinical trial results on reducing heart disease risks takes several years and requires large financial costs.

When Lipitor (atorvastatin) entered the market in 1997, it did not have clinical evidence to show its efficacy in reducing heart disease risks. However, it had expanded its market volume steadily and rapidly since its entry. Lipitor’s success is puzzling. This is because before the entry of Lipitor, three incumbent statins had already established clinical evidence on their ability in reducing heart disease risks. Assuming that physicians’ ultimate goal is to lower patients’ chances of having heart attacks


2For instance, a recent clinical trial shows that a new anti-cholesterol combination drug, Vytorin, does not reduce heart disease risks even though it is very effective in lowering the cholesterol levels (Park, 2008).
or strokes, one would expect they should prefer the older statins with direct clinical evidence on such efficacy.

In this paper, we propose the following explanation to rationalize this puzzle. Since statins use the same chemical mechanism to lower cholesterol levels (Zhou et al., 2006), it is plausible that physicians believe that all statins share similar ability of translating *reduction in cholesterol* to *reduction in heart disease risks* (i.e., the efficiency ratio). Therefore, when encountering clinical evidence on the efficiency ratios from older statins, they then update their beliefs about Lipitor’s efficiency ratio. Since Lipitor is also more effective in lowering cholesterol level, physicians may then infer that Lipitor is more effective in reducing heart disease risks compared with its competitors, even though it does not have direct clinical evidence to prove it yet.

To capture this information spillover story, we develop a structural demand model of correlated learning. First, we define a variable, “efficiency ratio,” which measures how effective a drug can convert *reduction in cholesterol levels* to *reduction in heart disease risks*. Landmark clinical trials provide information about drugs’ efficiency ratios. Most physicians and patients might not actively search for clinical trial results and indirectly learn about scientific information through certain types of media. We allow detailing$^3$ and news coverage (hereafter, we refer it to *publicity*) to play a role in delivering information embedded in clinical trials to physicians and patients. A pharmaceutical representative may inform or remind a physician of the drug’s efficacies. Alternatively, a physician/patient may learn about a drug’s efficacy or the release of an important clinical trial from news media (e.g., Ching et al., 2016).$^4$

Market frictions (e.g., switching costs and refilling costs) are usually ignored in the demand estimation literature using product level market share data (e.g., Azoulay, 2002; Berndt et al., 1996; Berry

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$^3$Detailing is a marketing technique used by pharmaceutical companies wherein a pharmaceutical representative visits a physician and explains efficacies and side-effects of a drug.

$^4$It is also possible that a patient who has exposed to publicity about a drug could ask his physician about the drug, and such an inquiry could motivate his physician to look up clinical evidence for that drug.
et al., 1995; Ching, 2010b; Ching and Ishihara, 2010; Narayanan et al., 2005). However, the relatively high discontinuing rate and low switching rate suggest that ignoring refilling costs and switching costs could lead to serious bias in the demand side parameter estimates.\(^5\) To take the switching and refilling costs into account, we supplement standard market share data with data on switching rates by drug (the percentage of patients who switch from one statin to another statin) and discontinuing rates (the percentage of drug \(j\)’s patients who decide to discontinue the statin treatment). To our knowledge, this is the first structural demand estimation paper that takes these two factors into account. Our estimation strategy is to take the discontinuing and switching decisions as exogeneous. The data on discontinuing and switching rates allow us to decompose the demand for each brand into two components: (i) those due to new patients and switchers; (ii) those due to non-switchers (because they want to avoid the switching costs). The main goal of our research is to develop and estimate a structural model that focuses on the drug choice of new patients and switchers.

The estimation results show physicians’ initial prior belief on the efficiency ratio (ER) is relatively low and they learn about the true efficiency ratio from clinical trials. However, the initial prior on the correlation of ERs across statins is high, and that leads to positive correlated learning across statins. This means after reading the results of a clinical trial, physicians not only learn about the ER of the statin being studied in that trial, but they also update their beliefs about the ERs of other statins not being studied. Moreover, we find that publicity in reducing the heart disease risks dimension increases physicians’ chance to learn about clinical trial results. Our estimation results suggest that there is information spillover of landmark clinical trials across drugs. This, together with the fact that two late entrants, Lipitor and Crestor, are more effective in lowering cholesterol (compared with the older statins), allow them to gain late-mover advantages. Moreover, we find that intensive detailing efforts (via its informative and persuasive roles) also contribute to their successes.

\(^5\)The main problem is that for categories that requires repeated purchases, the product level sales data is a function of new consumers, switchers, quitters, and consumers who routinely choose the same brand because of switching costs/positive state dependence.
How large is the late-mover advantage? To address this question, we conduct a counterfactual experiment to measure how important correlated learning is for Lipitor’s sales. The counterfactual experiment shows that the new patients’ demand for Lipitor would drop by 8% to 23% if physicians do not use the clinical evidence for older statins to update their belief about Lipitor’s efficacy.

Since our model incorporates consumers’ learning about clinical trials, the results can also be used to forecast the returns of landmark clinical trials (measured by how much demand they can generate) which are usually sponsored by pharmaceutical firms. Such results are important for managers who need to decide which clinical trials to fund. Note that Lipitor obtained its own landmark clinical trial results six years after its entry in 1997. How much of the impact did these landmark clinical trials have on Lipitor’s sales? Were the landmark clinical trials worth the investment for Pfizer given that Lipitor were able to (imperfectly) free-ride on the clinical trials conducted by its rivals? Our counterfactual experiment suggests that the sales of Lipitor could drop by 6.2% in Canada without its own landmark trials in 2004. The global sales of Lipitor is $10.9 billion dollars in 2004. If the global market share for statins is similar to the Canadian market, a simple extrapolation suggests that the annual global sales of Lipitor could have decreased by as much as $600 million in 2004 if Pfizer had not invested in the landmark clinical trials. Given that the average cost of a phase 4 clinical trial is $27.8 million for cardiovascular drugs, this suggests that it probably makes sense for Pfizer to invest in post-marketing clinical trials for Lipitor.

The rest of this paper is organized as follows. Section 2 reviews the previous literature. Section 3 describes background information including the market for statins. Section 4 discusses our data set. Section 5 describes the structural model. Section 6 presents the estimation results. Section 7 is the conclusion.

2 Literature Review

Although some papers have developed learning models to study the pharmaceutical market (e.g., Chan et al., 2013; Ching, 2010a,b; Chintagunta et al., 2009; Crawford and Shum, 2005; Narayanan et al., 2005), most of them do not model clinical evidence as a source of quality signals at all. An exception is the study by Ching and Ishihara (2010). But they only use qualitative information of comparison clinical studies (which say whether drug A is better than drug B). In this study, we treat clinical trial results more seriously than previous research. More specifically, we treat the information reported in landmark clinical trials as “observable” signals not only to the agents in the model, but also to researchers. This greatly simplifies the estimation procedure by avoiding the integration of unobserved signals when forming the likelihood, a computationally intensive procedure which is typically needed in previous works. In addition, the clinical trial data also help identify the parameters of the model, as we will discuss later.

It is worth mentioning that our model is related to Chan et al. (2013) who propose a learning model incorporating multi-dimensional attributes and positive state dependence. They investigate physicians’ learning on the effectiveness and side effects of drugs separately through patients’ reported reasons of switching in the erectile dysfunction (ED) category. Similar to their study, we develop a multi-dimensional model. However, the sources of identification are very different. They rely on physician level survey data, while we rely on the content of clinical trials and the variation of the number of prescriptions at the product level.

Our study is also closely related to Janakiraman et al. (2009) – they extend the umbrella branding framework of Erdem (1998) and Erdem and Sun (2002) to investigate correlated learning (information spillover) across competing brands in the anti-depressant market. However, like most of the previous studies, they do not consider the possibility that the release of post-marketing clinical trials may provide more information for the sales representatives to detail. Instead, they follow Erdem and Keane
(1996) and assume that detailing activities always provide physicians with noisy and unbiased signals on product quality. This assumption implies that drug manufacturers are always fully informed of their drugs’ true quality and they can make physicians learn about the true quality of their products after paying the physicians many detailing visits. This implication is inconsistent with the findings of other empirical studies (e.g., Azoulay, 2002; Venkataraman and Stremersch, 2007; Ching and Ishihara, 2010), which show evidence that clinical trial results can affect the effectiveness of detailing. Unlike Janakiraman et al. (2009), our model is able to study the interactions between post-marketing clinical trials and informative marketing activities across drugs.

Our modeling framework significantly extends the study of Ching and Ishihara (2010), which provides a structural modeling framework that allows the effectiveness of informative detailing to vary with clinical trial evidence. Unlike our study, Ching and Ishihara (2010) do not consider correlated learning. Moreover, we use quantitative information of clinical studies, instead of just the qualitative clinical evidence outcomes of comparison studies. It should also be highlighted that most of the studies mentioned above do not take switching costs into consideration, except Chan et al. (2013), who use physician level data. Most importantly, our paper is the first that quantifies late-mover advantages due to correlated learning/information spillover and indirect inference by physicians.

3 Background

There are two main types of cholesterol: LDL (“bad” cholesterol) and HDL (“good” cholesterol). The medical literature has shown that high LDL is a risk factor for heart diseases. Hereafter, we will follow the tradition and use the term “cholesterol” for LDL. Although the main purpose of statins is to reduce heart disease risks, a drug company cannot make the direct claim that its statin can reduce heart diseases risks until it obtains direct evidence from a clinical trial to support the claim. This is because the public health agency is worried that some unknown side-effects of the drug could counter its benefits of lowering cholesterol levels. The information on the effectiveness of a statin in reducing
heart disease risks, however, is usually unavailable when the statin is marketed because it takes a few years to obtain such direct evidence. To obtain the direct scientific evidence, pharmaceutical firms invest in very expensive post-marketing clinical trials, which are called landmark clinical trials. More specifically, the clinical endpoint (the target outcome) of landmark clinical trials for statins is the drugs’ efficacy in reducing heart disease risks. Landmark clinical trials also report how much each statin lowers the cholesterol level. By looking at these two efficacies, the effectiveness in lowering the cholesterol level and in reducing heart disease risks, physicians can learn about the efficiency ratio of a statin. In this research, we assume that landmark clinical trials are the original source of information for the effectiveness in reducing heart disease risks of statins. One might argue that physicians could learn from their patients’ feedbacks. However, a heart attack/stroke is a very rare event and it is very hard for a physician to learn from his/her own patients’ feedbacks. Therefore, we model the efficiency ratios reported in clinical trials as the only quality signals that physicians use to update their prior.

On the other hand, it is much easier and quicker for physicians to learn about a drug’s effectiveness in lowering cholesterol levels. The manufacturer of each statin is required to prove the statin’s ability in lowering cholesterol levels through clinical trials before the drug’s entry to the market. In addition to clinical trials, physicians also learn about the effectiveness in reducing cholesterol levels from his/her own patients’ feedbacks. Once physicians prescribe statins to their patients, they can observe their patients’ cholesterol levels in a relatively short period. Therefore, in this research, as the first approximation, we assume that physicians always know the effectiveness of reducing cholesterol levels for all statins. Table 1 presents a brief summary of the main descriptive statistics for the seven statins, Mevacor, Zocor, Pravachol, Lescol, Liptior, Baycol and Crestor.

In general, statins do not relieve any acute symptoms from patients and that makes it difficult for patients to experience the real benefits of statins (i.e., its ability to reduce heart disease risks). Because patients do not feel any direct discomfort from the discontinuation of statin treatment, a significant
proportion of patients discontinues statin treatment in each period (Neslin et al., 2009). On the contrary, conditioning on continuing the treatment, it is uncommon for patients to switch among statins. This suggests that large switching costs are present in this market. Chan et al. (2013) incorporate switching costs when they model prescription drug choice and find that large switching costs exist in the erectile dysfunction (ED) drug market. While Chan et al. (2013) use physician level data, we only observe product level data which is more readily available to firms. Hence, we do not estimate the switching costs, but we take their presence into consideration by supplementing product level data with switching rate data. In addition, we take the presence of refilling costs into consideration by using discontinuing rate data. More details about the switching and discontinuing rate data will be provided in subsection 4.2.

4 Data

This research makes use of four different data sources: (i) product level quarterly prescription volume and detailing data for the Canadian statin market from IMS Canada; (ii) product level quarterly prescription switching rates between statins and discontinuing rates from statins from Ontario Health Insurance Program (OHIP); (iii) landmark clinical trials obtained from published medical journals and a meta-analysis which summarizes statins’ efficacy in lowering cholesterol levels; (iv) news articles covering statins collected from Factiva.

4.1 Prescription Volume and Detailing

The product-level data obtained from the market-research firm, IMS Canada, consists of quarterly observations of prescription volumes and detailing costs for each statin across Canada from Q2 1993 ($t = 1$) to Q4 2004 ($t = 47$). The market is defined as the national market for quarter $t$. The observation is defined as a molecule-quarter combination.

In Figure 1, we plot the quarterly prescription volumes for seven statins in Canada. The prescription
volume for Lipitor reached around 2.8 million by 2004 while the earlier arrivals, Zocor and Pravachol, had 900,000 and 500,000 quarterly prescriptions, respectively. In 2002, Lipitor achieved estimated annual global sales of $7.4 billion and became the best-selling product in the prescription drug market. When Lipitor hit the market in 1997, Warner-Lambert, the manufacturer of Lipitor, released a head-to-head study supporting superior efficacy of Lipitor in lowering cholesterol levels over existing statins but did not establish any direct scientific evidence that Lipitor is effective in reducing heart disease risks. ASCOT-LLA, the first landmark clinical trial to support Lipitor’s efficacy in reducing heart disease risks, was released in 2003, six years after Lipitor’s entry. On the other hand, the existing statins had well established direct evidence that they are effective in reducing heart disease risks. Figure 1 indicates that Lipitor became very successful before the direct evidence supporting its efficacy in reducing heart disease risks became available. If we believe that the main goal of physicians is to reduce their patients’ chance of getting heart disease, their behavior of prescribing a new drug without direct clinical evidence over drugs with direct evidence, is not easy to apprehend.

Previous research has documented that marketing activities have an influence on physicians’ learning. Since detailing is considered a major promotional activity in the pharmaceutical industry, it is important for us to include detailing expenditures for each drug to see if it can explain the success of Lipitor. Figure 2 graphs the evolution of the quarterly detailing spending for five major statins.\(^7\) The entries of Lipitor (Q2 1997) and Crestor (Q1 2003) are accompanied by their large detailing efforts. Mevacor (Q2 1997), Pravachol (Q3 2000), and Zocor (Q1 2003) stopped detailing when the generic substitutes for their own products were introduced in the market. Note that while on average Pravachol spent more detailing than Lipitor between Q1 1997 and Q4 1998, Lipitor became the best selling statin in Q1 1999. This fact alone suggests that although detailing may partially account for the sales, the success of Lipitor cannot be fully explained by detailing spending only.

\(^7\)To convert from nominal to real dollars for detailing, we use the Consumer Price Index from Statistics Canada.
4.2 Switching and Discontinuing Rate Data

The data obtained from OHIP (Ontario Health Insurance Program) consist of quarterly number of patients who continue using the same statin \((c_{jt})\), number of patients who switch to other statins \((s_{jt})\), and number of patients who discontinue statin medication \((d_{jt})\) at time \(t\) among the patients who use statin \(j\) at time \(t-1\) in the Ontario from Q2 1993 \((t=1)\) to Q4 2004 \((t=47)\). From this dataset, we obtain the switching rate \(S_{jt} = \frac{s_{jt}}{c_{jt} + s_{jt} + d_{jt}}\) and the discontinuing rate \(D_{jt} = \frac{d_{jt}}{c_{jt} + s_{jt} + d_{jt}}\). Figure 3 shows that the switching rates between statins are less than 5% for almost all quarters for all drugs. These low switching rates indicate the existence of switching costs in the statin market. Moreover, switching rates became higher when new drugs, Lipitor (in 1997) and Crestor (in 2003), were introduced. Figure 4 shows that discontinuing rates are almost 15% on average, which are much higher than switching rates. This suggests that the cost of refilling prescriptions is even higher.

Note that prescription volume and detailing data are for the whole Canadian market, while the switching and discontinuing data are for Ontario only. Unfortunately, we are not able to obtain the switching and discontinuing rate data for other provinces. But the population in Ontario is more than one third of the population in Canada, they should serve as a reasonable proxy. Therefore, the sample size should be large enough to represent the population distribution of Canada.

4.3 Publicity: Clinical Trials and News Coverage

4.3.1 Clinical Trials

Following Azoulay (2002), Ching and Ishihara (2010), and Cockburn and Anis (2001) who find evidence that clinical trials have significant impacts on physicians’ prescribing decisions, we hypothesize that clinical trial outcomes affect physicians' decisions by providing them with information on the efficiency ratios of statins, i.e., how efficiently a statin can convert reduction in cholesterol levels to reduction in heart disease risks.

\(^8\)A discontinuing user is one who filled a statin prescription in the previous quarter, but did not fill a prescription for any of the statins in the current quarter. Similarly, a switcher is one who filled a statin prescription in the previous quarter, but switched to fill a prescription of a different statin in the current quarter.
Each landmark clinical trial has slightly different clinical endpoints and follows patients with different conditions for different follow-up periods. But most of them last for several years. Even though clinical endpoints are slightly different across landmark clinical trials, all the landmark clinical trials report mean LDL reduction and relative risk reduction in major vascular events. Major vascular events include major coronary events, coronary revascularization procedures, and strokes. Since medical literature has claimed that there is an overall positive and linear relationship between reduction in LDL and reduction in the risk for major vascular events across landmark clinical trials, we adopt the efficiency ratio as a measure on how efficiently a drug can translate absolute LDL reduction to the reduction in the risks for major vascular events (see Figure 3 in Cholesterol Treatment Trialists’ Collaborators (2005)). Table 2 lists the 14 landmark clinical trials we include in this research.

Every statin is approved as a cholesterol lowering drug because the manufacturer is required by public health agencies to prove its statin’s ability in lowering cholesterol levels through clinical trials. Moreover, physicians can directly observe the cholesterol levels of their patients within a short period of time after prescribing a statin. We therefore assume that physicians immediately learn about the true efficacy in lowering cholesterol levels of each stain as soon as a drug enters the market.

For our analysis, we take the information on each drug’s cholesterol lowering ability from the study of Law et al. (2003) who conducted a meta-analysis summarizing the results of clinical trials which investigate effectiveness of statins on reducing LDL. Law et al. (2003) include all double blind clinical trials reporting mean absolute LDL reductions (mmol/L) in the statin treated group and in the placebo group from Medline, Cochrane Collaboration, and Web of Science databases. They define drug efficacy as the difference between the LDL reductions in the treated and placebo groups, and calculate the drug efficacy for each clinical trial. From the drug efficacy data across clinical trials, they report the mean absolute reduction in LDL of statins including Mevacor, Zocor, Pravachol, Lescol, Liptior and Crestor by dosage (5mg, 10mg, 20mg, 40mg and 80mg) across clinical trials. Since this meta-analysis
does not report the effectiveness in LDL reduction of Mevacor with 5mg dose, we exclude data for all other statins with 5mg dose. Table 3 shows the mean LDL reduction of each statin by strength.\(^9\) The numbers are taken from Table 2 of Law et al. (2003). By taking the average of the reported mean LDL reductions across strengths of each drug, we create a drug specific variable denoting LDL reduction efficacy. The values of this variable is 1.59 for Mevacor, 1.66 for Zocor, 1.28 for Pravachol, 1.16 for Lescol, 2.22 for Lipitor, and 2.44 for Crestor.\(^{10}\) The data from the study of Law et al. (2003) is very important for our research because it allows us to pin down the effectiveness of lowering cholesterol levels for each statin without the need of estimating them. If we had to estimate the effectiveness of lowering cholesterol levels, it would be very hard for us to identify the learning parameters. The identification strategy will be discussed in subsection 5.5.

### 4.3.2 Publicity

The publicity data are obtained from Ching et al. (2016), which explains the details. We will briefly describe it here. The data is collected from news article data covering statins that contain the word “statin” or words related to statin, such as the chemical names or brand names from Factiva from 1986 to 2004, restricted to sources which Canadian patients should have access to. For each article, we extract its headline, source, content and publication date. We first map the information of each article into two multidimensional variables: (a) general publicity variable \((publicity^g)\) – if it has sentences that discuss statins in general without referring to any particular statin by brand or chemical name; (b) drug specific publicity variable \((publicity^d)\) – if it has sentences that refer to one or more statins by either brand or chemical name. Note that an article may contain information that can be mapped onto both variables – it can provide information about statins in general at the beginning, and then later mention which particular statin is the most effective. Ching et al. (2016) finds that general publicity is

\(^9\)While Table 3 uses mmol/L as unit of LDL reduction, the unit in Table 2 is mg/dL. Because molar mass of cholesterol is 386.65g. 1 mmol/L of LDL can be converted to 38.6mg/dL.

\(^{10}\)We have also collected data from CURVES study, with which Pfizer provided the FDA to receive the approval for Lipitor. The results on the LDL reduction abilities are consistent with those of Law et al. (2003). However, CURVES study does not report the efficacy of Crestor. Therefore, we do not use the results from CURVES study.
more likely to affect the overall demand for statins, while drug specific publicity could influence both total demand for statins and which particular statin to use.\textsuperscript{11}

We classify both general and drug specific publicity into three dimensions: lowering cholesterol levels, reducing heart disease risks and side-effects. Hereafter, we use \((lc^s_t, rh^s_t, se^s_t)\) to represent the three dimensions of the general publicity variable, where the superscript \(s\) means that they are for the whole statin class; \(t\) indexes time. For the drug specific publicity variable, we use \((lc_{jt}, rh_{jt}, se_{jt})\), to represent its three dimensions, where \(j\) is an index for drug. For each dimension of both drug specific and general publicity, we use a two-step Likert scale \((+1, -1)\) to assess its tone. We assign “+1” (“-1”) if the article contains sentences which favor (do not favor) the focal drug. Given that information about efficiency ratio should only be mentioned in \(rh_{jt}\), for drug specific publicity, we only use \(rh_{jt}\) in our analysis. We plan to use \((lc_{jt}, se_{jt})\) as controls in our robustness checks.

In our empirical analysis, the length of a period is a quarter. Since there are usually more than one news story published/broadcasted in each quarter, we need to aggregate the outcomes of the news appeared in the same quarter to obtain a quarterly observation. We use the following procedure to do the aggregation. Let \((publicity^s_t,l, publicity_{jt,l})\) denote the publicity variables associated with article \(l\) that is published in quarter \(t\). Also, let \(L_t\) be the total number of news stories appeared in quarter \(t\). Then the values of \((publicity^s_t,l, publicity_{jt,l})\) are obtained by simply summing \((publicity^s_t,l, publicity_{jt,l})\) across the news stories appeared in quarter \(t\). For example, \(publicity^s_t = \sum_{l=1}^{L_t} publicity^s_{t,l}\).

Figure 5 shows the general publicity flow variables. While there are some bad news articles about statins’ side-effects, especially in 2001 when Baycol was removed from market, most news articles report that statins are effective in lowering cholesterol levels and reducing heart disease risks. Table 4 presents a descriptive summary of drug specific publicity variables.

\textsuperscript{11}For drug specific publicity, we sometimes encounter articles that compare drugs. Therefore, we further classify drug specific publicity into comparison (\(c\)) or non-comparison (\(nc\)). But the results of Ching et al. (2016) indicate that comparison publicity does not have much variation. Therefore, we only use non-comparison drug specific publicity.
4.4 Potential Market Size

In order to study market expansion, our model includes an outside good (i.e., we allow patients with high cholesterol to choose treatments other than statins or no treatment at all). We therefore need to measure the potential market size for statins, which includes high cholesterol patients who are on statins and other anti-cholesterol drugs, and those who choose not to take any drugs. In order to estimate the percentage of Canadians with a high cholesterol problem, we follow Ching et al. (2016) and use data from the Canadian Heart Health Survey, recorded between 1986 and 1992. We multiply it by the total Canadian population for each age group in a given quarter, as defined by Statistics Canada, and use the result as a proxy for the total number of potential patients for statins. In order to convert the total population with high cholesterol levels into the number of prescriptions, we assume that each patient visits a physician and receives a prescription once per 90 days.\textsuperscript{12}

4.5 Preliminary Evidence for Correlated Learning

To test whether such a correlated learning (information spill-over) effect exists, we regress the number of prescriptions for Lipitor on the interaction between the cumulative clinical outcomes of Lipitor’s rival drugs (Mevacor, Zocor and Pravachol) and Lipitor’s cumulative detailing stock before Lipitor’s own clinical trials (Q2/2002), controlling for other factors. When creating the cumulative detailing stock, we set the carryover rate to be 90%. We report two specifications here. Table 5 shows the specifications and results.

Specification (1) shows that the interaction term between the rival drug’s cumulative clinical outcomes of Lipitor and Lipitor’s cumulative detailing stock is positive and significant. This is consistent with our hypothesis that the effectiveness of Lipitor’s detailing would increase with the clinical outcomes of Lipitor’s rival drugs. Specification (1) also includes rivals’ cumulative clinical outcomes to control its

\textsuperscript{12}According to Cosh (2010), other than Quebec, the prescription sizes for statins in the rest of Canada are typically 90 days and 180 days. For administrative purposes, the maximum prescription size is one month in Quebec. Since the population in Quebec is roughly a quarter of the entire Canada, we assume that the average prescription size for statins is 90 days in Canada.
direct impact on Lipitor’s sales. This variable turns out to be insignificant. Then, in specification (2), we drop the variable. Qualitatively, the result remains the same. In both specifications, we find that the interaction between the rivals’ cumulative clinical outcomes and Lipitor’s own cumulative detailing to be positive and statistically significant. This provides support for our hypothesis that there is an information spill-over effect.

5 Model and Estimation

In this section, we propose a structural demand model incorporating physicians’ correlated learning about clinical trial outcomes. We also discuss how to construct the likelihood function, and the identification issues. It is important to emphasize that we will only model drug choice for new patients and existing patients who decide to switch. We do not model why consumers decide to quit using this class of drugs, and why the majority of the existing patients keep using the same brand/drug even though their physicians’ prior may suggest another drug is superior in ex-ante.\(^1\)

5.1 Bayesian Learning Model

Consider a situation where physician \(k\) needs to decide which drug to prescribe for patient \(i\). The utility of patient \(i\) who consumes drug \(j\) at time \(t\) is given by

\[
U_{ijt} = \omega \cdot q^h_j + \lambda_j + \epsilon_{ijt},
\]

(1)

where \(q^h_j\) denotes drug \(j\)’s efficacy in reducing heart disease risks; \(\lambda_j\) captures time-invariant brand specific preference, e.g., price difference across brands, efficacy in lowering cholesterol levels;\(^1\) \(\epsilon_{ijt}\) is an i.i.d. random shock and is extreme value distributed.

We assume that the physician chooses a drug to maximize the sum of her patient’s utility conditional on her information set and her utility from marketing spending of pharmaceutical firms such as

\(^1\)We will explain the challenges in section 5.3.

\(^1\)Due to price regulations on prescription drug prices in Canada, prices for statins hardly changed over our sample period. Moreover, we assume that physicians knew the true quality of each statin in lowering cholesterol levels when each statin was marketed.
persuasive detailing. The demand system is obtained by aggregating this discrete choice model of an individual physician’s behavior. Note that physicians/patients are uncertain about $q_j^h$. We therefore assume that physicians make their prescribing decisions based on her expected utility. Let $I(k, t)$ denote physician $k$’s information set at time $t$. For now, let’s assume that a physician does not get utility from persuasive detailing, and hence she acts as a perfect agent on behalf of her patients. Physician $k$’s expected utility of prescribing drug $j$ to patient $i$ at time $t$ will be:

$$E[U_{ijt}|I(k, t)] = \omega \cdot E[q_j^h|I(k, t)] + \lambda_j + \epsilon_{ijt}, \quad (2)$$

where $E[\cdot|I(k, t)]$ denotes the expected value given physician $k$’s information set at time $t$. We assume that physicians make their prescribing decisions based on their current expected utility. One might argue that physicians can be forward-looking and experiment different drugs to learn about $q_j^h$. However, since a heart attack is a very rare event, it is unlikely that physicians can use patients’ experiences to update their prior about $q_j^h$. Consequently, we do not consider that physicians have an incentive to experiment different drugs on their patients.

Let $q_j^c$ be the efficacy in lowering cholesterol levels of drug $j$, and $\beta_j$ be the efficiency ratio. We define the “efficiency ratio” as a measure on how efficiently a drug can convert reduction in cholesterol levels to reduction in heart disease risks. Then, $q_j^h$ can be expressed as follows:

$$q_j^h = q_j^c \cdot \beta_j. \quad (3)$$

Because we assume that physicians have complete information about the efficacy in lowering cholesterol levels of each drug ($q_j^c$) but are uncertain about the efficiency ratio of each drug ($\beta_j$), physicians can easily learn about the efficacy in lowering cholesterol levels of each drug from abundant non-landmark clinical trials or from patients’ consumption experience. On the other hand, physicians can learn about the efficiency ratio only from landmark clinical trials.
Now we turn to explain how physicians learn about $\beta_j$’s. We model physicians’ learning process by adopting the Bayesian learning framework (DeGroot, 1970). Physicians construct their initial prior belief before they learn about the results of landmark clinical trials. As discussed earlier, because all statins use a similar mechanism to lower cholesterol levels, their initial prior belief about $\beta_j$’s may be correlated across $j$. In other words, information about $\beta_j$ can be useful for updating $\beta_{-j}$, and vice versa. Due to this intrinsic correlated belief, physicians may infer $q_j$ indirectly from the clinical trial evidence on $\beta_{-j}$. More specifically, we assume the initial prior is normally distributed and allow the off-diagonal elements ($\rho$) in the variance-covariance matrix for the initial prior beliefs to be non-zero.

As a first step, we assume the initial priors to be the same across drugs. Since it is unlikely for physicians to learn about the efficacy in heart disease risks of statins from their patients’ experiences, landmark clinical trials (which are specifically designed to prove the efficacy of drugs in heart disease risks) become the main sources of information about this efficacy. Physicians are assumed to update their beliefs on the efficiency ratio of each drug when they are exposed to landmark clinical trial results. Note that even when two landmark clinical trials test the same drug, they typically use patients with different conditions. Hence, the efficiency ratios obtained from different landmark clinical trials should not be the same even if the number of patients goes to infinity. We assume that the efficiency ratio associated with each trial $l$ of drug $j$, $\bar{\beta}_{jl}$, is:

$$\bar{\beta}_{jl} = \beta_j + \epsilon_{jl},$$

where $\beta_j$ is the true mean level of the efficiency ratio for drug $j$; $\epsilon_{jl} \sim N(0, \sigma_e)$ and is i.i.d. They believe that a landmark clinical trial $l$ for drug $j$ provides a noisy but unbiased signal for the efficiency ratio. In other words, a signal from clinical trial $l$ for drug $j$, $\tilde{\beta}_{jl}$, can be expressed as:

$$\tilde{\beta}_{jl} = \bar{\beta}_{jl} + \zeta_j,$$

where $\zeta_l$ is a i.i.d. signal noise, and $\zeta_l \sim N(0, \sigma^2_{\zeta})$. Let $\sigma^2_{\zeta}$ be signal variance for one patient, and $N_l$ be the number of patients who participate in landmark clinical trial $l$. As long as the individual signals
are i.i.d. across patients, it can be shown that $\sigma^2_{\mathcal{L}} = \sigma^2_{\zeta}/N_l$. This implies that the more participants a clinical trial has, the more physicians will trust its results. Moreover, we can combine the two equations above and write,

$$\tilde{\beta}_{jl} = \beta_j + \nu_{jl}, \quad (7)$$

where $\nu_{jl} = \varepsilon_{jl} + \zeta_{jl}$. This implies that $\nu_{jl} \sim N(0, \sigma^2_{\epsilon} + \frac{\sigma^2_{\zeta}}{N_l})$ Note that unlike most of the previous research, we are able to observe quality signals by using the information from the landmark clinical trials. As we will explain later, this data will help us identify the correlated learning parameters and simplify the estimation procedures.

To explain how physicians update their beliefs through learning about clinical trials, let us provide a simplified example which can be easily generalized. The general model is provided in the appendix. In this example, we assume that there are two statins $(j = 1, 2)$ and there is only one landmark clinical trial, which investigates drug 1’s efficacy in reducing heart disease risks. Let $\beta_{jt}$ be the expected perceived efficiency ratio, and $\sigma^2_{\beta_{jt}}$ be the perceived variance of drug $j$, conditional on the physician $k$’s information set at time $t$. The variance-covariance matrix for prior beliefs of physician $k$ at time $t$ becomes,

$$V[\beta_j|I(k,t)] = \begin{pmatrix} \sigma^2_{\beta_{1t}} & \pi_t \\ \pi_t & \sigma^2_{\beta_{2t}} \end{pmatrix}. \quad (8)$$

If the physician learns about clinical trial $l$ for drug 1, she will update her beliefs on the efficiency ratio of drug 1 as follows:

$$\beta_{1t+1} = \beta_{1t} + \frac{\sigma^2_{\beta_{1t}}}{\sigma^2_{\beta_{1t}} + \sigma^2_{\xi_{1l}}} \cdot (\tilde{\beta}_{1l} - \beta_{1t}). \quad (9)$$

She also updates her prior variance on the efficiency ratio of drug 1 at time $t$ as follows:

$$\sigma^2_{\beta_{1t+1}} = \frac{\sigma^2_{\beta_{1t}} \sigma^2_{\xi_{1l}}}{\sigma^2_{\beta_{1t}} + \sigma^2_{\xi_{1l}}}. \quad (10)$$
With correlated prior beliefs on the efficiency ratio, signals for drug 1 are used to update beliefs on drug 2 as well. Posterior beliefs for drug 2 are given as:

$$\beta_{2t+1} = \beta_{2t} + \frac{\pi_t}{\sigma_{\beta_{2t}}^2 + \sigma_{\zeta_{1t}}^2} (\bar{\beta}_{1t} - \beta_{1t}),$$  \hspace{1cm} (11)

where $\pi_t$ denotes the off-diagonal element in the variance-covariance matrix of the perceived quality on the efficiency ratio at time $t$.

The variance of her posteriors on the efficiency ratio of drug 2 at time $t$ becomes

$$\sigma_{\beta_{2t+1}}^2 = \sigma_{\beta_{2t}}^2 - \frac{\pi_t^2}{\sigma_{\beta_{2t}}^2 + \sigma_{\zeta_{1t}}^2}. \hspace{1cm} (12)$$

The off-diagonal element of variance-covariance matrix for posterior beliefs becomes

$$\pi_{t+1} = \frac{\pi_t \sigma_{\zeta_{1t}}^2}{\sigma_{\beta_{11}}^2 + \sigma_{\zeta_{11}}^2}. \hspace{1cm} (13)$$

As a result, the variance-covariance matrix for posterior beliefs becomes

$$V[\beta_j | I(k, t + 1)] = \begin{pmatrix} \sigma_{\beta_{11}}^2 & \pi_t \sigma_{\zeta_{11}}^2 \\ \pi_t \sigma_{\zeta_{11}}^2 & \sigma_{\beta_{22}}^2 \end{pmatrix} = \begin{pmatrix} \frac{\sigma_{\beta_{11}}^2 \sigma_{\zeta_{11}}^2}{\sigma_{\beta_{11}}^2 + \sigma_{\zeta_{11}}^2} & \frac{\pi_t \sigma_{\zeta_{11}}^2}{\sigma_{\beta_{11}}^2 + \sigma_{\zeta_{11}}^2} \\ \frac{\pi_t \sigma_{\zeta_{11}}^2}{\sigma_{\beta_{11}}^2 + \sigma_{\zeta_{11}}^2} & \sigma_{\beta_{22}}^2 - \frac{\pi_t^2}{\sigma_{\beta_{22}}^2 + \sigma_{\zeta_{11}}^2} \end{pmatrix}. \hspace{1cm} (14)$$

5.2 Roles of Detailing and Publicity

In this subsection, we explain how detailing influences demand. The economics and marketing literature studying the pharmaceutical industry find evidence that detailing can play both informative and persuasive roles (Ching and Ishihara, 2012; Leffler, 1981; Narayanan et al., 2005). To encourage physicians to prescribe their drugs, detailers might inform physicians of their drug’s efficacies and side effects (informative role). However, they can also persuade physicians to prescribe their drugs regardless of the clinical information about their drugs (persuasive role). For example, detailers provide physicians with free gifts, which can affect physician’s prescribing decisions. We will model both roles and discuss how to separately identify them.

We first describe how we model the persuasive role. Here, we adopt the standard approach by modeling a detailing goodwill stock entering physician $k$’s utility function directly. Therefore, we
modify eq(2) as follows:

\[ U_{kjt} = E[U_{ijt}|I(k,t)] + \kappa_d \cdot P_{STK\_detailjt}, \]  

(15)

where \( P_{STK\_detailjt} \) is a persuasive detailing goodwill stock for drug \( j \) at time \( t \). The persuasive detailing stock is defined as:

\[ P_{STK\_detailjt} = \delta_{d\_per} \cdot P_{STK\_detailjt-1} + \text{detail}_{jt}, \]  

(16)

where \( \delta_{d\_per} \) is the quarterly carryover rate for persuasive detailing; \( \text{detail}_{jt} \) denotes the flow of detailing spending for drug \( j \) at time \( t \).

We now explain how to model the informative role of detailing and publicity. We modify the model proposed by Ching and Ishihara (2010). They model informative detailing as a means to build and maintain the measure of physicians who know the most updated information about drugs. The basic setup of the model is as follows. There is a continuum of physicians with measure one. They are heterogeneous in their information sets. A physician is either well-informed or uninformed about drug \( j \). A well-informed physician knows the current information set maintained by the representative opinion leader \( (I_j(t)) \). An uninformed physician only knows the initial prior \( (I_j) \).

The measure of informed physicians of drug \( j \) at time \( t \), \( M_{jt} \), is a function of \( I_{STK\_detailjt} \), \( I_{STK\_detail-jt} \) and \( STK\_rh_{jt} \), where \( I_{STK\_detailjt} \), \( I_{STK\_detail-jt} \) and \( STK\_rh_{jt} \) denote the informative stocks of detailing, competitors’ detailing and drug specific non-comparison publicity in reducing heart disease risks for drug \( j \) at time \( t \), respectively. If statin manufacturers knew about the correlated learning, their sales reps might have wanted to discuss about other statin’s clinical trials to free-ride on competitors’ clinical trial results. To capture this behavior, we add \( I_{STK\_detail-jt} \). As a result, each drug \( M_{jt} \) is expressed as follows:

\[ M_{jt} = \frac{\exp(\alpha_0 + \alpha_d \cdot I_{STK\_detailjt} + \alpha_c \cdot I_{STK\_detail-jt} + \alpha_{rh} \cdot STK\_rh_{jt})}{1 + \exp(\alpha_0 + \alpha_d \cdot I_{STK\_detailjt} + \alpha_c \cdot I_{STK\_detail-jt} + \alpha_{rh} \cdot STK\_rh_{jt})}. \]  

(17)
The informative detailing stock is defined as:

\[ I_{STK_{detail}} = \delta_{inf} \cdot I_{STK_{detail}} - 1 + detail, \]

where \( \delta_{inf} \) is the quarterly carryover rate for informative detailing; \( detail \) denotes the flow of detailing spending for drug \( j \) at time \( t \). The informative stock of competitors’ detailing is defined as:

\[ I_{STK_{detail}} = \delta_{inf} \cdot I_{STK_{detail}} - 1 + detail, \]

where \( detail \) denotes the flow of sum of detailing spending for all statins except for drug \( j \) at time \( t \). Note that competitor’s detailing stock shares the same carryover rate as own stock of detailing. The informative publicity stock is defined as:

\[ STK_{rh} = \delta_{rh} \cdot STK_{rh} - 1 + rh, \]

where \( \delta_{rh} \) is the quarterly carryover rate for reducing heart disease risks publicity; \( rh \) denotes the flow of drug specific non-comparison publicity in reducing heart disease risks for drug \( j \) at time \( t \). Note that we assume drug specific publicity only plays an informative role.

5.3 Prescribing Decisions

Based on patients’ choices in the previous period \((t - 1)\), we classify patients at time \( t \) into two groups, “potential patients” and “existing patients.” First, we will explain the decision making process of “potential patients.” As Figure 6 depicts, our model assumes that their decision making process consists of two stages. The first stage (adoption decision stage) determines whether a potential patient will use statins. The decision in this stage could be jointly made by the patient and his physician. For example, news articles reporting the problem of high cholesterol levels or the benefits of taking statins could entice the patient to see a physician. Alternatively, a physician detailed by pharmaceutical representatives might recommend her patient to get a blood test. Therefore, we model that sum of publicity and sum of detailing spending affect the decision making process in this stage. The probability that a physician
prescribes one of the statins to her potential patients at time $t$, $P_t(statin)$, is expressed as follows:

$$P_t(statin) = \frac{\exp(\gamma_i \cdot Inclusive_i + \gamma_p \cdot STK_{PUB} + \gamma_0)}{1 + \exp(\gamma_i \cdot Inclusive_i + \gamma_p \cdot STK_{PUB} + \gamma_0)},$$

(21)

where $Inclusive_i$ is the inclusive value term derived from the brand choice stage; $STK_{PUB}$ denotes a vector of three types of general publicity ($rh_t^*, lc_t^*, se_t^*$) stocks for the class of statin. We assume that they all share the same carryover rate.

If a potential patient decides to use statins, then we move to the second stage (statin choice stage), which determines which statin to be prescribed. The physician evaluates all the statins available given her information set and chooses the most appropriate statin for her patient. The probability that physician $k$ chooses drug $j$ for a new patient, conditional on prescribing, is expressed as follows:

$$P_t(j|statin, k_{type}) = \frac{\exp(U_{kjt}(k_{type}))}{\sum_r \exp(U_{krt}(k_{type}))}.$$  

(22)

Note that the information set of physician $k$, $I(k, t)$ is a function of physician $k$’s type at time $t$. Because we assume that for each drug $j$, a physician is either informed about the most updated landmark clinical trials for this drug, or he is uninformed about any of them, the total number of physician types is $2^H$.

Note that although there are seven statins, $H = 5$ in our application because only five of them have landmark clinical trials.

Let $P_t(k_{type})$ denotes the probability of physician $k$ being a particular type. The expected “new patients demand” (group 1) for drug $j$ at time $t$, $d^1_{jt}$, can be expressed as:

$$d^1_{jt} = (m_t - \sum_r d_{rt-1}) \cdot P_t(statin) \cdot \sum_{k_{type}=1}^{2^H} P_t(k_{type}) \cdot P_t(j|statin, k_{type}),$$

(23)

where $d_{jt}$ is the demand for drug $j$ at time $t$, which we will define later; $(m_t - \sum_r d_{rt-1})$ it the potential patient pool for statins at time $t$.

For “existing patients,” their decisions are more complicated than “potential patients”. Figure 7 depicts the decision tree of existing patients. In the first stage, they decide to either quit or keep taking statins. Once they decide to keep taking statin, they will decide either to stay with the same statin
or switch to other statins. If they decide to switch, they will choose which statin to switch to. Note that some patients might keep taking the same statin even though there are alternatives to give him a better expected consumption utility. This may be because many elder patients with high cholesterol problems are taking many drugs, and they may find it very troublesome to remember another drug and to understand its potential side-effects. If an existing patient decides to stay with the current statin, we classify the patient as a “stayer.” If he decides to switch to one of other statins, we classify him as a “switcher.”

The expected demand for stayers (group 2) can be expressed as:

$$d^2_{jt} = d_{jt-1} \cdot (1 - S_{jt} - D_{jt}),$$

(24)

where $S_{jt}$ and $D_{jt}$ denote switching and discontinuing rates of drug $j$ at time $t$ which is from our data set. It should be highlighted that the whole sequence of $\{d^2_{jt}\}$ will be determined by the equation above because we observe $S_{jt}$, $D_{jt}$ and $d_{jt}$.

The “switchers” (group 3) are patients who took a statin other than drug $j$ in the previous period but take statin $j$ at period $t$. Switchers do not consider the same drug which they chose in the previous period. The estimated demand for switchers can be expressed as:

$$d^3_{jt} = \sum_{m=1, m \neq j}^J \left[ d_{mt-1} \cdot S_{mt} \cdot \sum_{k_{type}=1}^{2H} P_t(k_{type}) \cdot \frac{\exp(U_{kjt}(k_{type}))}{\sum_r^{J} \exp(U_{krt}(k_{type}))} \right].$$

(25)

Note that once a patient leaves from taking statin treatment, he will be back to the potential patient pool in the next period.

We should also highlight that we do not use nested logit framework for the switchers’ decision making process. Because we do not model $S_{jt}$ as a function of physicians’ expected utilities in prescribing each statin, the decision on whether to switch is independent of the decision on which drug to prescribe. We do not model $S_{jt}$ as a function of physicians’ expected utilities for the following reasons:

1. It is very hard for a patient or a physician to directly observe the efficacy in reducing heart disease
risks from patient’s experience. Therefore, the patient’s or physician’s switching decision could mainly rely on factors other than heart disease risks.

2. If we model the switching rate as a function of physicians’ expected utilities, the model will become too complex to estimate. If the switching rate is a function of physicians’ expected utilities, the patients of physicians who evaluate drug \( j \) the least will switch from drug \( j \) to other statins first. Then, the physicians of remaining patients with drug \( j \) will have a different distribution of utilities from the physicians of switching patients from drug \( j \). To model these switching or staying behaviors, we have to simulate a very large number of physicians and patients and then follow their decisions at each period. Such a simulation procedure will significantly increase the computational burden of estimating the model.

Because we treat \( S_{jt} \) and \( D_{jt} \) as exogeneous, we do not estimate the switching costs and refilling costs parameters.

### 5.4 Estimation

#### Likelihood

The quantity demand \( d_{jt} \) at time \( t \) for drug \( j \) can be expressed as:

\[
d_{jt} = \hat{d}_{jt}^1 + \hat{d}_{jt}^2 + \hat{d}_{jt}^3 + e_{jt},
\]

where \( e_{jt} \) represents a measurement error. \( \hat{d}_{jt}^1 \), \( \hat{d}_{jt}^2 \) and \( \hat{d}_{jt}^3 \) denote the estimated demand for group 1, 2 and 3, respectively. Note that Subsection 5.3 describes how we model estimated demand for each group.

Assuming that the measurement error, \( e_{jt} \) in equation (26) is normally distributed, we can obtain the likelihood function:

\[
l(\{d_{jt}\}_j^j\{\{detail\}_{j}^{j}\}_{j=1}^J\{\{\beta_{jt}\}_{i=1}^{i}\}_{i=1}^I\{N_i\}_{i=1}^{i}\{PUB\}_{\tau=1}^{\tau}\{\{PUB_{jt}\}_{j=1}^{j}\}_{j=1}^J\theta_d),
\]

(27)
where $\theta_d$ is the vector of parameters; $\text{detail}_{jt}$ is detailing spending for drug $j$ at time $t$; $l_t$ denotes the number of landmark clinical trials up to time $t$; $\tilde{\beta}_{jl}$ is a level of quality signal from landmark clinical trial $l$; $N_l$ is the number of patients which clinical trial $l$ follows; $\text{PUB}_s^t$ and $\text{PUB}_jt$ are vectors of general publicity and drug specific publicity, respectively. The likelihood of observing $d = \{\{\text{detail}_{jt}\}_{j=1}^J\}_{t=1}^T$ is

$$L(d|\{\text{detail}_{jt}\}_{j=1}^J\}_{t=1}^T, \{\tilde{\beta}_{jl}\}_{l=1}^L, \{N_l\}_{l=1}^L, \{\text{PUB}_s^t\}_{t=1}^T, \{\text{PUB}_jt\}_{j=1}^J, \theta_d)$$

$$= \prod_{t=1}^T l(\{d_{jt}\}_{j=1}^J|\{\text{detail}_{jt}\}_{j=1}^J\}_{t=1}^T, \{\tilde{\beta}_{jl}\}_{l=1}^L, \{N_l\}_{l=1}^L, \{\text{PUB}_s^t\}_{t=1}^T, \{\text{PUB}_jt\}_{j=1}^J, \theta_d).$$ (28)

We estimate parameters by maximizing the log-likelihood function. Unlike the previous literature on learning models, all the quality signals are “observable” in our model. Therefore, we can simply construct the likelihood function without adopting any simulation method.

**Initial condition problem**

Note that our data set for prescription volume starts only in Q2 1993. Mevacor, Zocor and Pravachol were introduced before that time and so, by Q2 1993, these three drugs should have accumulated stocks of detailing, and publicity. If we do not have detailing and journal advertising data prior to Q2 1993, the detailing and journal advertising stocks will be subject to the classic initial condition problem (Heckman, 1981). To address this, we have collected monthly detailing and journal advertising data going back to Q3 1988, when the first statin (Mevacor) was introduced, and use these data to construct the initial values of detailing stock in Q2 1993. Similarly, for the publicity variables, we use the pre-sample period data from Q1 1986 to Q1 1993 to construct the initial values of publicity stocks in Q2 1993.$^{17}$

**5.5 Identification**

In this subsection, we provide some intuitions about how the parameters of our model can be identified. The parameters in the adoption decision stage ($\gamma_0$, $\gamma_i$, $\gamma_{lc}$, $\gamma_{se}$) can be identified by the variation of

$^{17}$It is unlikely that there is much news about statins available prior to Q1 1986 because the first statin was launched in Q2 1988.
market share of statins as a whole and the variation of the explanatory marketing variables, such as general publicity and inclusive values.

Correlations in the initial prior beliefs ($\rho$) can be identified from the observed (to researcher) quality signals on efficiency ratios from clinical trial outcomes and the timing of each clinical trial release as well as the changes in relative market shares of statins before and after the release of each clinical trial. In identifying the correlation parameter, the observed quality signals play a pivotal role because the change in market shares before and after the release of a clinical trial can be moderated by both the realized quality signal from the clinical trial and the extent of correlated learning. For example, if a drug does not gain relative market share after the release of its own clinical trial, there are two possible explanations: (i) The realized quality signal from the clinical trial is the same as physicians’ current perceived quality for the drug, and there is no correlated learning (i.e., $\rho = 0$). Or, (ii) the realized quality signal is higher than physicians’ current perceived quality, but the extent of correlated learning is extremely high (i.e., $\rho \simeq 1$); consequently, physicians update their prior beliefs about the qualities of both drugs by the same amount. By explicitly using the information reported in a clinical trial, we can observe the realized quality signals. This is how we can tell which explanation plays a bigger role, and hence identify the correlation parameter.

It is important to stress that the “initial prior” in our model captures the physicians’ belief prior to the release of any landmark clinical trials. Since drugs entered the market at different point of time, the existing stocks of landmark clinical trials faced by them also differ at time when they entered the market. As long as $\rho > 0$, the entry date prior beliefs will differ across drugs. Hence, after controlling for $q_j^c$’s and detailing stocks, the differences of initial sales across drugs will help us identify $\rho$ (because it is a function of the entry date prior beliefs).

The parameters that determine the persuasive ($\kappa_d$) and informative detailing ($\alpha_d$) can be separately identified because (i) they enter the model in two very different structural ways, and (ii) we assume
that clinical trial outcomes only affect the informative detailing and we explicitly use the information from clinical trials. As a result, clinical trials provide exclusion restrictions needed to disentangle the persuasive and the informative effects of detailing. It is worth emphasizing that clinical trials differ in terms of (a) which drugs they study; (b) number of subjects (patients); (c) reported mean efficiency ratio; (d) release time. All of these would only change the way informative detailing affects physicians’ expected utility associated with different drugs. For example, the observed clinical trial results and their release timings help identify the informative detailing parameter and physicians’ perceived quality by type in each period. Therefore, the variation of the market shares, and the corresponding variation of detailing help identify the proportion of each physician type and informative detailing parameters.

Note that the change in physicians’ information sets would also change the impact of persuasive detailing on physician’s choice in our random utility modeling framework, but in a very specific way determined by the model. Therefore, the persuasive detailing parameters are essentially acting as “free” parameters to help fit the variation of market shares that cannot be fully explained by informative detailing and learning.

6 Results

6.1 Parameter Estimates

Table 6 shows the parameter estimates. The first section in the table describes learning parameters. Physicians’ initial prior mean on efficiency ratio is 0.016. As shown in Table 2, most signals on the efficiency ratios from landmark clinical trials are between 0.1 and 0.3. Therefore, it appears that physicians’ initial prior beliefs on statins’ efficiency ratios is quite a bit lower than the true efficiency ratios. The initial prior variance ($\sigma_\beta^2$), signal variance from different clinical trial designs ($\sigma_\epsilon^2$), and the signal variance per 1000 patients ($\sigma_\epsilon^2$), and all statistically significant. The initial prior correlation on efficiency ratio ($\rho$) is 0.776. This implies that if one statin receives a new clinical trial result, physicians update their beliefs about the efficiency ratio of not only the focal statin in the clinical trial, but
also other statins. We also find that the coefficient for the perceived quality ($\omega$) to be positive and significant.

To demonstrate the rate of learning, in Figure 8 we graph how the well-informed physician learns about the efficiency ratios (i.e., $E[\beta_{jt}]$) over time based on our parameter estimates. The well-informed physician refers to a physician who has learned about all the clinical trial results available up to time $t$. The figure shows that the physician updates her beliefs about all the drugs whenever the clinical trial is released. Because of correlated learning (information spillover), she learns about the efficacies of not only a drug studied in the clinical trial, but also other statins. Before Q4 1994, there has been no landmark clinical trial to support statins’ efficacy in reducing heart disease risks. The physician has exactly the same prior belief about Mevacor, Zocor and Pravachol before Q4 1994. In Q4 1994, Zocor received a new clinical trial (4S study) supporting its efficacy in reducing heart disease risks. Then, the physician updates her beliefs about all statins not just about Zocor due to the correlated learning. However, because the information spillover is not 100%, the physician’s belief on Zocor is slightly higher than those on other statins. Lipitor’s first landmark clinical trial was released in Q2 2003. Before Q2 2003, the physician has the lowest belief on Lipitor among all existing statins. However, after several clinical trial results for Lipitor are released, $E[\beta_{\text{Lipitor},t}]$ became the highest among all statins. The graph suggests that Lipitor benefits much from other statins’ investment in landmark clinical trials but there is still room for its own investment. In one of our counterfactual experiments, we will investigate this further.

Figure 9 shows how the proportion of informed physicians changes over time by drug. Note that for the earlier half of the observations, the measure of physicians who are informed about Lipitor’s most updated clinical trials are lower than those who are informed about Zocor and Pravachol. However, the sales of Lipitor has surpassed those of its competitors very early on. This illustrates that Lipitor’s strong $q_j^c$, together with physicians’ initial prior on its $\beta_j$, have already led their $E[q_j^h|I(t)]$ to be better
than other drugs’.

We find that both informative ($\alpha_d$) and persuasive ($\kappa_d$) detailing parameters are positive and significant. The results indicate that detailing has both persuasive and informative roles in physicians’ prescription choices. Brand specific non-comparison publicity in reducing heart disease risks ($\alpha_{rh}$) is also positive and significant, indicating that it can help inform physicians (perhaps indirectly via the pressure of patients) about the drug’s clinical trials. This could happen if patients who are exposed to publicity in heart disease risks encourage their physicians to read clinical trial results. We also find evidence that rivals’ detailing could help informing physicians about a drug’s clinical trials ($\alpha_c$ is positive and significant). This is consistent with correlated learning. For instance, when its own landmark clinical trials are still underway, Lipitor may want to take advantage of correlated learning and uses its detailing to inform physicians about the landmark trials of other drugs. This is one way to enhance its late-mover advantages.

Next, we discuss parameters in the adoption decision stage. The estimate of the inclusive value term is positive (1.29) and significant at the 10% level. The stock of general publicity in reducing heart disease risks ($Stk_{rh}^s$) is also estimated to be positive and significant, but the stock of lowering cholesterol ($Stk_{lc}^s$) turns out insignificant. However, we do not want to read too much into these two variables because they are highly correlated. Interestingly, the coefficient on the stock of general publicity in side-effects ($Stk_{se}^s$) is negative and significant, indicating that consumers are generally worried about side-effects.

### 6.2 Counterfactual Experiments

Now we turn to counterfactual experiments. Because our model is designed to capture the choice of new patients, we will focus on studying how the patients’ demand would change under the counterfactual experiments. Our model also applies to switchers. But recall that switching rates are less than 5% for almost all quarters for all drugs. Such low switching rates imply that switchers would contribute very
little to changes in total demand.

**Experiment 1 (Quantifying the Return of Lipitor’s Landmark Clinical Trials)**

A clinical trial to prove efficacy in reducing heart disease risks requires medical researchers to follow up on thousands of patients for a few years. Therefore, sponsoring such a clinical trial is a very large investment for the firm. If physicians can indirectly learn about the ability of a new statin in reducing heart disease risks through incumbents’ clinical trials, it might not be worthwhile for the manufacturer of the new statin to sponsor another landmark clinical trial for its own statin. This could be the case for Lipitor. Prior to its entry in 1997, several incumbent firms had already obtained landmark clinical results for reducing heart disease risks. Lipitor obtained its own landmark clinical trial results several years after its introduction in 1997. Were the landmark clinical trials for its own drug, Lipitor, worth the investment of the drug company?

To address these questions, we use our model to forecast the demand for Lipitor in a counterfactual situation where Lipitor does not receive any clinical results supporting that it reduces heart disease risks by shutting down Lipitor’s landmark clinical trials. Figure 11 graphs the benchmark and counterfactual new patients’ demand for statins. The dotted lines denote the counterfactual new patients’ demands. Without its own landmark clinical trials, the counterfactual demand for Lipitor due to new and switching patients is 10% to 15% lower than the benchmark demand for most quarters from Q2 2003 to Q4 2004 (note that the first landmark clinical trial for Lipitor was released in May 2003). The counterfactual demand due to new and switching patients is about 58,000 prescriptions lower than the benchmark counterpart in Q4 2004.

The magnitude of the change might appear to be insignificant. However, Lipitor’s global annual sales is almost $13 billion in 2003. Therefore, even just 1% loss in sales would cost more than $100 million per year. In a post-marketing clinical trial, following one patient would cost roughly tens of thousand dollars for a few years. Even with rough calculation, the 10% sales difference per year is
probably large enough to justify Lipitor’s investment in its own post-marketing clinical trials.\(^{18}\)

**Experiment 2 (Quantifying Late-Mover Advantages)**

Our estimation results suggest that (i) there is information spillover of landmark clinical trial results across drugs, and (ii) Lipitor (and Crestor) can gain late mover advantage by free-riding on the information provided by its rivals’ clinical trials. Therefore, we are interested in quantifying the importance of correlated learning. How much did Lipitor benefit from the clinical trials conducted by other drug companies? To answer the above questions, we forecast the demand for each statin in a counterfactual situation where there is no correlated learning. Under this counterfactual experiment, we set the correlated learning parameter \((\rho_0)\) to be zero. Figure 12 presents the benchmark and counterfactual demand due to new and switching patients. For Lipitor, the counterfactual demand is almost 23% lower than the benchmark demand early on, and the difference diminishes over time to about 8% in Q4 2004 (amounts to about 35,000 prescriptions). For Crestor, the counterfactual demand is consistently about 33-40% lower than the benchmark demand for most quarters. The difference is very substantial, and it indicates that correlated learning plays an very important role for the success of Lipitor.

However, note that even without correlated learning, the demand for Lipitor from new patients keeps increasing over time. This suggests that correlated learning cannot be the only driving force for the early success of Lipitor. So what else can contribute to its success? One possibility is its superior efficacy in lowering cholesterol levels. Because Lipitor has the highest \(q^c\) (except Crestor), Lipitor could still have a high \(E[q^h]\) even with a relatively low \(E[\beta]\). To investigate this possibility, in Figure 13 we graph the most updated physician’s \(E[q^h]\) over time under the condition that there is no correlated learning. It shows that Lipitor has a fairly low \(E[q^h]\) in the absence of correlated learning up until Q2 2003. Therefore, it seems likely that the early success of Lipitor and Crestor is also driven by persuasive

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\(^{18}\)A report to U.S. Department of Health and Human Services states that average cost of Phase 4 (3) clinical trials for cardiovascular drugs is $27.8 (25.2) million (http://aspe.hhs.gov/sp/reports/2014/ClinicalTrials/rpt_erg.pdf). Note that the nature of statin’s landmark clinical trials fits into the cardiovascular drugs category. Note further that Phase 3 is the final phase of pre-marketing clinical studies to confirm safety and efficacy (it tests on 1000-3000 patients); Phase 4 refers to postmarketing studies to provide additional information such as treatment’s risks, benefits, and optimal use.
7 Conclusion and Future Research

We develop a new structural model of physicians’ prescribing decisions under uncertainty where physicians can learn about the quality of drugs through correlated learning. We define a variable, “efficiency ratio,” which measures how efficiently a drug can convert reduction in cholesterol levels to reduction in heart disease risks. We assume that physicians learn about the efficiency ratio for each drug from landmark clinical trials and allow physicians’ initial prior perceptions of the efficiency ratio to be correlated across drugs. We find that the initial prior perceptions on the efficiency ratio are positively correlated. This information spillover allows a late mover (such as Lipitor and Crestor) to significantly benefit from incumbents’ clinical trials on proving their drugs’ efficacy in reducing heart disease risks.

Unlike the previous literature which assumes that quality signals from clinical trials are unobservable to researchers, we treat quality signals from clinical trial results as observable by taking a careful look at clinical trial results and extracting detailed information from the clinical trials. By treating clinical trials in this manner, we are able to clearly identify the correlated learning parameters.

In addition to using product level market share data, we supplement them with switching rates and discontinuing rates. These data switching rate data allow us to take the presence of switching costs and refilling costs into consideration.

Our model also allows detailing to have both persuasive and informative roles in physicians’ prescribing decisions. Our data on the content of clinical trials allows us to tease out the informative effect of detailing. The results are useful for marketing managers who need to allocate their detailing budgets optimally according to how much information physicians have.

We should point out that switching rates or discontinuing rates can differ under some counterfactual conditions. However, because we do not explicitly model switching rates or discontinuing rates, we assume that switching rates or discontinuing rates will remain unchanged in the counterfactual ex-
periments. A fruitful future research direction is to examine more micro level data, and use it to model switching rates and discontinuing rates as a function of switching costs, refilling costs, and physicians' information set.

We should also emphasize that the model developed here can also be applied to settings other than prescription drugs. For instance, when iPhone entered the market, it is a very innovative product. The heavy promotion done by Apple has informed a large population about what a touchscreen phone can accomplish (like a mini-computer). When Samsung entered the market later (as a late entrant), it leveraged what consumers already know about basic ideas of this product, and launched its own android based phones with larger screen sizes, more memory, and faster processors. Some consumers then infer that a Samsung phone is better than iPhone. This can explain why Samsung became the largest smartphone player even though it entered the market after Apple.
References


Table 1: Summary of Statins

<table>
<thead>
<tr>
<th>Brand</th>
<th>Molecule</th>
<th>Entry Date</th>
<th>Generic Entry</th>
<th>Manufacturer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pravachol</td>
<td>pravastatin</td>
<td>Oct-1990</td>
<td>Jul-2000</td>
<td>Bristol-Myers Squibb</td>
</tr>
<tr>
<td>Lescol</td>
<td>fluvastatin</td>
<td>Mar-1994</td>
<td>N.A.¹</td>
<td>Novartis</td>
</tr>
<tr>
<td>Lipitor</td>
<td>atorvastatin</td>
<td>Mar-1997</td>
<td>N.A.¹</td>
<td>Pfizer</td>
</tr>
<tr>
<td>Baycol</td>
<td>cerivastatin</td>
<td>Mar-1998</td>
<td>N.A.²</td>
<td>Bayer</td>
</tr>
<tr>
<td>Crestor</td>
<td>rosuvastatin</td>
<td>Feb-2003</td>
<td>N.A.¹</td>
<td>AstraZeneca</td>
</tr>
</tbody>
</table>

1 - The patent expiration date is beyond our sample period.
2 - Baycol was withdrawn in August 2001 before its patent expires.

Table 2: Landmark Clinical Trials for Statins

<table>
<thead>
<tr>
<th>Title</th>
<th>Publication Date</th>
<th>Drugs Studied</th>
<th># of Subjects</th>
<th>Follow-up Period</th>
<th>Efficiency Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>4S</td>
<td>Dec, 1994</td>
<td>Zocor</td>
<td>4,444</td>
<td>5.2 years</td>
<td>0.21</td>
</tr>
<tr>
<td>WOSCOPS</td>
<td>Nov, 1995</td>
<td>Pravachol</td>
<td>6,595</td>
<td>4.8 years</td>
<td>0.27</td>
</tr>
<tr>
<td>CARE</td>
<td>Oct, 1996</td>
<td>Pravachol</td>
<td>4,159</td>
<td>4.8 years</td>
<td>0.22</td>
</tr>
<tr>
<td>Post-CABG</td>
<td>Jan, 1997</td>
<td>Mevacor</td>
<td>1,351</td>
<td>4.2 years</td>
<td>0.22</td>
</tr>
<tr>
<td>AFCAPS/TexCAPS</td>
<td>May, 1998</td>
<td>Mevacor</td>
<td>6,605</td>
<td>5.3 years</td>
<td>0.30</td>
</tr>
<tr>
<td>LIPID</td>
<td>Nov, 1998</td>
<td>Pravachol</td>
<td>9,014</td>
<td>5.6 years</td>
<td>0.20</td>
</tr>
<tr>
<td>GISSI Prevention</td>
<td>Dec, 2000</td>
<td>Pravachol</td>
<td>4,271</td>
<td>1.9 years</td>
<td>0.23</td>
</tr>
<tr>
<td>LIPS</td>
<td>Jun, 2002</td>
<td>Lescol</td>
<td>1,677</td>
<td>3.1 years</td>
<td>0.24</td>
</tr>
<tr>
<td>HPS</td>
<td>Jul, 2002</td>
<td>Zocor</td>
<td>20,536</td>
<td>5 years</td>
<td>0.21</td>
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<td>PROSPER</td>
<td>Nov, 2002</td>
<td>Pravachol</td>
<td>5,804</td>
<td>3.2 years</td>
<td>0.13</td>
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<tr>
<td>ALLHAT-LLT</td>
<td>Dec, 2002</td>
<td>Pravachol</td>
<td>10,355</td>
<td>4.8 years</td>
<td>0.12</td>
</tr>
<tr>
<td>ASCOT-LLA</td>
<td>May, 2003</td>
<td>Lipitor</td>
<td>10,305</td>
<td>3.2 years</td>
<td>0.28</td>
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<tr>
<td>ALERT</td>
<td>Jun, 2003</td>
<td>Lescol</td>
<td>2,102</td>
<td>5.1 years</td>
<td>0.11</td>
</tr>
<tr>
<td>CARDS</td>
<td>Aug, 2004</td>
<td>Lipitor</td>
<td>2,838</td>
<td>3.9 years</td>
<td>0.32</td>
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</table>
### Table 3: Statins’ Mean Cholesterol Reduction by Strength (mmol/L)

<table>
<thead>
<tr>
<th></th>
<th>Daily Dose (mg)</th>
<th></th>
<th></th>
<th></th>
<th>Mean</th>
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<tr>
<td></td>
<td>5</td>
<td>10</td>
<td>20</td>
<td>40</td>
<td>80</td>
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<tr>
<td>Mevacor</td>
<td>N/A</td>
<td>1.02</td>
<td>1.40</td>
<td>1.77</td>
<td>2.15</td>
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<tr>
<td>Zocor</td>
<td>1.08</td>
<td>1.31</td>
<td>1.54</td>
<td>1.78</td>
<td>2.01</td>
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<tr>
<td>Pravachol</td>
<td>0.73</td>
<td>0.95</td>
<td>1.17</td>
<td>1.38</td>
<td>1.60</td>
</tr>
<tr>
<td>Lescol</td>
<td>0.46</td>
<td>0.74</td>
<td>1.02</td>
<td>1.30</td>
<td>1.58</td>
</tr>
<tr>
<td>Lipitor</td>
<td>1.51</td>
<td>1.79</td>
<td>2.07</td>
<td>2.36</td>
<td>2.64</td>
</tr>
<tr>
<td>Crestor</td>
<td>1.84</td>
<td>2.08</td>
<td>2.32</td>
<td>2.56</td>
<td>2.80</td>
</tr>
</tbody>
</table>

### Table 4: Summary of Publicity Variables

<table>
<thead>
<tr>
<th>Reducing Risks of Heart Disease</th>
<th># of Quarters</th>
<th>Values</th>
<th></th>
<th></th>
<th>Mean</th>
<th>Std. Dev.</th>
<th>Min.</th>
<th>Max.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mevacor</td>
<td>47</td>
<td>0.84</td>
<td>1.77</td>
<td>-1.58</td>
<td>7.13</td>
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<tr>
<td>Zocor</td>
<td>47</td>
<td>2.01</td>
<td>3.15</td>
<td>-3.00</td>
<td>14.25</td>
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<tr>
<td>Pravachol</td>
<td>47</td>
<td>1.97</td>
<td>2.68</td>
<td>0</td>
<td>12.67</td>
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</tr>
<tr>
<td>Lescol</td>
<td>44</td>
<td>0.07</td>
<td>0.25</td>
<td>0</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Lipitor</td>
<td>32</td>
<td>2.98</td>
<td>4.28</td>
<td>0</td>
<td>16.15</td>
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<tr>
<td>Baycol</td>
<td>14</td>
<td>0.07</td>
<td>0.27</td>
<td>0</td>
<td>1.00</td>
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<td></td>
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<tr>
<td>Crestor</td>
<td>8</td>
<td>0.84</td>
<td>1.07</td>
<td>0</td>
<td>2.85</td>
<td></td>
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</table>
Table 5: Preliminary Evidence for Correlated Learning

<table>
<thead>
<tr>
<th>Variables</th>
<th>(1) Estimates</th>
<th>S.E.</th>
<th>(2) Estimates</th>
<th>S.E.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>140533.63</td>
<td>71309.93</td>
<td>21032.56</td>
<td>33218.78</td>
</tr>
<tr>
<td>Cum_Detail&lt;sub&gt;i&lt;/sub&gt;</td>
<td>693.63</td>
<td>1531.91</td>
<td>1789.48</td>
<td>1513.34</td>
</tr>
<tr>
<td>Rival_Cum_Clinical&lt;sub&gt;i&lt;/sub&gt; X Cum_Detail&lt;sub&gt;i&lt;/sub&gt;</td>
<td><strong>0.15</strong></td>
<td>0.05</td>
<td><strong>0.06</strong></td>
<td>0.03</td>
</tr>
<tr>
<td>Rival_Cum_Clinical&lt;sub&gt;i&lt;/sub&gt;</td>
<td>-6.38</td>
<td>3.43</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R Squared</td>
<td>0.875</td>
<td>0.848</td>
<td></td>
<td></td>
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<tr>
<td>Adjusted R Squared</td>
<td>0.852</td>
<td>0.830</td>
<td></td>
<td></td>
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<tr>
<td>Number of Observations</td>
<td>20</td>
<td>20</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Estimates shown in bold are significant at 5% level.

Definition of Variables are as follows:

Cum_Detail<sub>i</sub>: Cumulative Stock of Detailing for Lipitor at quarter \( t \). Carryover rate is 90%.

Rival_Cum_Clinical<sub>i</sub>: Rivals' Cumulative Clinical Outcomes (Mevacor, Zocor and Pravachol). Carryover rate is 100%.
Table 6: Parameter Estimates

<table>
<thead>
<tr>
<th>Variable Descriptions</th>
<th>Estimates</th>
<th>S.E.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Statin Choice Stage</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Learning Parameters</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\bar{b}$ (Initial Prior Belief on Efficiency Raito)</td>
<td>0.0161</td>
<td>0.0087</td>
</tr>
<tr>
<td>$\sigma_\theta^2$ (Initial Prior Variance on Efficiency Raito)</td>
<td>0.5199</td>
<td>0.1442</td>
</tr>
<tr>
<td>$\sigma_e^2$ (Signal Variance from Different Design)</td>
<td>1.5607</td>
<td>0.2499</td>
</tr>
<tr>
<td>$\sigma_x^2$ (Signal Variance from 1,000 Patients)</td>
<td>4.0027</td>
<td>1.2653</td>
</tr>
<tr>
<td>$\rho_0$ (Correlated Learning Parameter in Initial Prior)</td>
<td>0.7757</td>
<td>0.0433</td>
</tr>
<tr>
<td>$\omega$ (Coefficient of Perceived Quality)</td>
<td>1.6727</td>
<td>0.0394</td>
</tr>
<tr>
<td>Parameters related to detailing and publicity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\alpha_0$ (Constant)</td>
<td>-0.6525</td>
<td>0.7071</td>
</tr>
<tr>
<td>$\alpha_d$ (Informative Detailing)</td>
<td>3.1985</td>
<td>0.2796</td>
</tr>
<tr>
<td>$\alpha_c$ (Informative Detailing of Competitors)</td>
<td>0.1915</td>
<td>0.0767</td>
</tr>
<tr>
<td>$\alpha_{rh}$ (Informative Publicity in Reducing Heart Disease Risks)</td>
<td>1.1468</td>
<td>0.0058</td>
</tr>
<tr>
<td>$\kappa_d$ (Persuasive Detailing)</td>
<td>1.3129</td>
<td>0.0861</td>
</tr>
<tr>
<td>$\delta_{d,inf}$ (Carryover Rate of Informative Detailing in Statin Choice)</td>
<td>0.8905</td>
<td>0.0142</td>
</tr>
<tr>
<td>$\delta_{rh,inf}$ (Carryover Rate of Informative Publicity in Statin Choice)</td>
<td>0.2117</td>
<td>0.0184</td>
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<tr>
<td>$\delta_{d,per}$ (Carryover Rate of Persuasive Detailing in Statin Choice)</td>
<td>0.9154</td>
<td>0.0092</td>
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<tr>
<td><strong>Brand Dummies</strong></td>
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<tr>
<td>Zocor</td>
<td>0.8932</td>
<td>0.0514</td>
</tr>
<tr>
<td>Pravachol</td>
<td>0.6792</td>
<td>0.0639</td>
</tr>
<tr>
<td>Lescol</td>
<td>-0.6851</td>
<td>0.2195</td>
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<tr>
<td>Lipitor</td>
<td>1.3537</td>
<td>0.0484</td>
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<tr>
<td>Baycol</td>
<td>-0.0451</td>
<td>0.1684</td>
</tr>
<tr>
<td>Crestor</td>
<td>0.7304</td>
<td>0.0905</td>
</tr>
<tr>
<td><strong>Adoption Decision Stage</strong></td>
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<td></td>
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<tr>
<td>$\gamma_0$ (Constant)</td>
<td>-7.2446</td>
<td>0.1916</td>
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<tr>
<td>$\gamma_I$ (Inclusive Value)</td>
<td>1.2886</td>
<td>0.0699</td>
</tr>
<tr>
<td>$\gamma_k$ (General Publicity Stock in Lowering Cholesterol Levels)</td>
<td>-0.0920</td>
<td>0.0545</td>
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<tr>
<td>$\gamma_{rh}$ (General Publicity Stock in Reducing Heart Disease Risks)</td>
<td>0.5872</td>
<td>0.1099</td>
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<tr>
<td>$\gamma_{se}$ (General Publicity Stock in Side Effects)</td>
<td>-0.0286</td>
<td>0.0058</td>
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<tr>
<td>$\delta_p$ (Carryover Rate of Publicity in Adoption Decision)</td>
<td>0.9113</td>
<td>0.0236</td>
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<tr>
<td><strong>Additional Parameter</strong></td>
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<tr>
<td>Standard Deviation of $e_{ij}$ (in Hundred Thousand)</td>
<td>0.2152</td>
<td>0.0084</td>
</tr>
<tr>
<td>Log Likelihood</td>
<td>-2746.24</td>
<td></td>
</tr>
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</table>
Figure 3: Quarterly Switching Rate for Leading Statins

Quarterly Switching Rates

- Mevacor
- Pravachol
- Zocor
- Lipitor
- Crestor

Figure 4: Quarterly Discontinuing Rate for Leading Statins

Quarterly Discontinuing Rates

- Mevacor
- Pravachol
- Zocor
- Lipitor
- Crestor
Figure 5: Quarterly Flow of General Publicity

![Chart showing the quarterly flow of general publicity with lines for Lowering Cholesterol, Reducing Heart Disease, and Side Effects.]

Figure 6: Decision Process of Potential Patient

- **Not Take a Statin**
  - Statin 1
- **Take a Statin**
  - Statin 2
  - Statin J
Figure 7: Decision Process of Existing Patient

- Keep Taking a Statin
- Quit Taking a Statin
- Decide to Switch
- Stay with Statin j

- Statin 1
- Statin j-1
- Statin j+1
- Statin J

Figure 8: The Posterior Beliefs of Well-Informed Physicians

†A well-informed physician means a physician who know all clinical trials available at time $t$. 
Figure 9: Measure of Informed Physicians of Statin $j$

Figure 10: Fit: Actual and Simulated Prescription Volume
Figure 11: Counterfactual Experiment 1 (Removing Lipitor’s Landmark Trials)

Figure 12: Counterfactual Experiment 2 (Removing Late-mover Advantages)
Figure 13: The Most Updated Physician’s $E[q^t]$ over Time (No Correlated Learning)