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. Although it lacked 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. We introduce a concept of "efficiency ratio," which measures how efficiently a drug can convert *reduction in the bad cholesterol* to *reduction in heart disease risks*. We assume physicians are uncertain about drugs' efficiency ratios, and allow their initial prior belief to be correlated across drugs. With correlated prior belief, a new clinical trial information signal 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 the bad 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 incumbents.

To estimate our model, we use a data set on market shares, patients' switching rates and discontinuing rates, as well as data on detailing, clinical trials and media coverage from 1993 to 2004. Our estimation results show 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 reducing the bad cholesterol, allows them to gain late-mover advantages and grow much faster in the actual world compared with a counterfactual world where there is no correlated learning/information spillover.

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

1 Introduction

Even when a drug has been introduced to the market for a few years, uncertainty for the drug often remains and deters 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,[1] 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 reduce their bad cholesterol, hoping that it will reduce their heart disease risks. However, before a clinical study on reducing heart disease risks becomes available for a drug, sales representatives can only make a direct claim on its efficacy in lowering the bad cholesterol. Although a positive correlation between bad cholesterol and coronary heart disease risks has been found in medical research, a drug which can reduce the bad cholesterol level effectively does not necessarily mean it can reduce heart disease risks.[2] This is because it might have some unknown side-effects that could raise heart disease risks and counter its benefits of reducing the bad cholesterol. In order to 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, post-marketing clinical trials on reducing heart disease risks take several years to complete, require many participants, and carry large financial costs.

When Lipitor (atorvastatin) entered the market in 1997, there was no clinical evidence to show its efficacy in reducing heart disease risks until Q2 2003. Yet, it was able to expand its market volume steadily and rapidly since its inception. Lipitor's success is puzzling because prior to its entry, three incumbent statins had already established clinical evidence as to their ability in reducing heart disease risks. Assuming that physicians' ultimate goal is to lower patients' chances of having heart attacks or

[1] FDA Code of Federal Regulations Title 21 (http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/CFRSearch.cfm?CFRPart=203&showFR=1&subpartNode=21:4.0.1.1.4.1, accessed on Jan 20, 2018).

[2] For 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 bad cholesterol (LDL) and raising the good cholesterol (HDL) (Park, 2008).

strokes, one would expect they would prefer the older statins with direct clinical evidence as to such efficacy.

In this paper, we propose the following explanation to rationalize this puzzle. Since statins use the same chemical mechanism to reduce the bad cholesterol (Zhou et al., 2006), it is plausible that physicians believe that all statins share an ability to convert *a reduction in the bad cholesterol* to *a reduction in heart disease risks* (i.e., the efficiency ratio). Therefore, when encountering clinical evidence on efficiency ratios from older statins, they may update their beliefs about Lipitor's efficiency ratio as well. In addition, because Lipitor is also more effective in lowering the bad cholesterol, physicians may then infer that Lipitor is more effective in reducing heart disease risks compared with its competitors, even though there is no direct clinical evidence yet to prove this.

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 the bad cholesterol* to *reduction in heart disease risks*. Landmark clinical trials provide information about efficiency ratios for different drugs. However, physicians and patients might not actively search for clinical trial results, and need to learn about this scientific information through different types of media. We therefore allow detailing[3] and news coverage (hereafter, we refer to it as *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]

When estimating our model, we also take the presence of switching costs and refilling costs into account. Such market frictions are usually ignored in the demand estimation literature using product

[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 been 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.

level market share data (e.g., Azoulay, 2002; Berndt et al., 1996; Berry et al., 1995; Ching, 2010b; Ching and Ishihara, 2010; Narayanan et al., 2005). However, the relatively high discontinuing rate and very low switching rate suggest that ignoring refilling costs and switching costs could lead to serious biases in the demand side parameter estimates.[5] To take the presence of switching and refilling costs into account, we supplement standard market share data with data on switching rates (i.e., the percentage of patients who switch from one statin to another statin) and discontinuing rates (i.e., the percentage of drug j's patients who decide to discontinue the statin treatment) by drug. To our knowledge, this is the first structural demand estimation paper that takes these two factors into account. Our estimation strategy is to assume the discontinuing and switching decisions as exogeneous, and we take them as given. The data on discontinuing and switching rates allow us to decompose the demand for each brand into two components: (i) demand due to new patients and switchers; (ii) demand due to stayers. The main goal of our research is to develop and estimate a structural demand model of correlated learning, which focuses on physicians' drug choice for new patients and switchers.

The estimation results show that physicians' initial prior belief on the efficiency ratio is relatively low and they learn about its true value from noisy signals generated by clinical trials. However, the initial prior on the correlation of efficiency ratios across statins is high, and that leads to positive correlated learning across statins. This implies that after reading the results of a clinical trial, physicians will not only learn about the efficiency ratio of the statin being studied, but also update their beliefs about the efficiency ratios of other statins not being studied in the trial. 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 reducing the bad cholesterol (compared with the older statins), allows them to gain late-mover

[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.

advantages, and grow much faster than they otherwise would.

Since our model incorporates consumers' learning about clinical trials, the results can 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 was able to (imperfectly) free-ride on clinical trials conducted by its rivals? Our first counterfactual experiment suggests that the new and switching patients' demand for Lipitor could have dropped by 12% in Canada without its own landmark trials in 2004. This is roughly about 2.5% of the actual total demand. In 2004, global sales of Lipitor totaled $10.9 billion dollars.[6] If the global market share for statins is similar to that of the Canadian market, a simple extrapolation suggests that annual global sales of Lipitor could have decreased by as much as $250 million in 2004 if Pfizer had not invested in the landmark clinical trials. Given that the average cost of a postmarketing 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.

To quantify the late-mover advantages, we conduct the second counterfactual experiment by removing correlated learning/information spillover. In other words, we only allow patients/physicians to learn about a drug's efficiency ratio from its own clinical trials. The counterfactual experiment shows that new and switching patients' demand for late entrants would drop very significantly. In particular, Lipitor would drop by about 50% on average up until Q2 2003 when Lipitor's first clinical trial on its efficacy of reducing heart disease risks were released. if physicians did not use the clinical evidence for older statins to update their belief about Lipitor's efficacy. The result suggests that correlated learning

[6] http://www.theglobeandmail.com/life/higher-doses-of-lipitor-cut-heart-attacks-but-deaths-from-other-causes-increased/article4115969/, accessed on Sep 5, 2016.

is crucial for the early success of Lipitor and Crestor.

In our third counterfactual experiment, we investigate how the evolution of market shares changes if physicians/patients do not face any switching costs. In this counterfactual situation, the market stealing effect is much more immediate for new entrants. It demonstrates that although the first-mover advantage of switching costs does not help incumbents to obtain new patients, it is very successful in protecting incumbents from losing its base patients.

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 8 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 of estimating learning models. In addition, the clinical trial data also helps 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, whereas 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) who 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 the presence of switching costs and refilling costs into consideration. An exception

is Chan et al. (2013), who used physician level data to estimate switching costs. However, they did not take into account the possibility that some patients may discontinue the treatment. 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 a statin is marketed because it takes a few years to obtain such direct evidence. To obtain 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 on how much each statin lowers the cholesterol level. By looking at these two efficacies, the effectiveness in lowering the cholesterol level and the effectiveness in reducing heart disease risks, physicians can learn about the efficiency ratio of a statin. Therefore, 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, heart attack/stroke is a very rare event. Hence, it would be difficult for a physician to tell how well a drug can treat this problem by only observing his/her own patients' feedbacks. Therefore, in our model, we assume clinical trials are

the only source of 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 the bad cholesterol. The manufacturer of each statin is required to prove the statin's ability in reducing the bad cholesterol through clinical trials before the drug can enter the market. In addition to clinical trials, physicians also learn about the effectiveness in reducing the bad cholesterol from his/her own patients' feedback. 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 each statin's effectiveness in reducing the bad cholesterol.

In general, statins do not relieve any acute symptoms for patients and that makes it difficult for patients to experience the real benefits of statins (i.e., their 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). To take the presence of refilling costs into account, we use the data on discontinuing rates.

Conditional 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. Although we do not estimate switching costs here, we take their presence into consideration by supplementing product level data with switching rate data. In subsection 4.2, we provide more details about switching and discontinuing rate data.

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 Ontario Health Insurance Program (OHIP); (iii) landmark clinical trials obtained from published medical journals, and a meta-analysis which summarizes statins' efficacy in the lowering the bad cholesterol; (iv) news articles covering statins collected from Factiva.

4.1 Prescription Volume and Detailing

The product-level data obtained from the marketing 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. Lipitor entered the market in 1997. It did not have any direct evidence about its efficacy in reducing heart disease risks until May 2003. Yet, as shown in Figure 1, Lipitor is able to rise rapidly and surpass its rivals in merely six quarters after its entry. It becomes by-far the most popular statin even before its first landmark clinical trial is released in Q2 2003. Given that the ultimate goal of taking statin is to reduce the chance of having heart diseases or strokes, the success of Lipitor is puzzling.

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 data 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] Note that Mevacor (Q2 1997), Pravachol (Q3 2000), and Zocor (Q1 2003) stopped detailing when the generic

[7]To convert from nominal to real dollars for detailing, we use the Consumer Price Index from Statistics Canada.

substitutes for their own products were introduced in the market. The detailing of Lipitor and Crestor started in Q1 1997 and Q1 2003, respectively, immediately following their launch. Although Pravachol and Zocor spent the similar amount of detailing as Lipitor between Q1 1997 and Q3 1998, Lipitor became the best selling statin in Q4 1998. This fact suggests that although detailing may partially explains its demand, the success of Lipitor cannot be fully explained by detailing spending only.

4.2 Switching and Discontinuing Rate Data

Our data on switching and discontinuing rates are obtained from OHIP (Ontario Health Insurance Program). It consists 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 ($t=47$).[8] 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. Such low switching rates indicates the presence of large switching costs in the statin market. Moreover, switching rates became higher when Lipitor and Crestor were introduced in 1997 and 2003, respectively. 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 rates 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 of average rates in Canada.

[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 swichted to fill a prescription of a different statin in the current quarter.

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 landmark clinical trial outcomes affect physicians' decisions by providing them with information on the efficay of statins. More specifically, we assume the landmark trials reveals the information on the efficiency ratios of statins, i.e., how efficiently a statin can convert reduction in the bad cholesterol to reduction in heart disease risks.

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. The medical literature assumes 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 (see Figure 3 in Cholesterol Treatment Trialists' Collaborators (2005), Figure 1 of Delahoy et al. (2009), Figures 2 and 3 of Silverman et al. (2016), Figure 1 of Wadhera et al. (2016)). This is well captured by our concept of efficiency ratio. Table 1 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 pubic health agencies to prove its statin's ability in lowering cholesterol levels through clinical trials. Moreover, physicians can usually monitor their patients' cholesterol levels within a short period of time after prescribing a statin. We therefore assume that physicians immediately learn about the true efficacy in reducing the bad cholesterol of each stain as soon as it 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 2 shows the mean LDL reduction of each statin by strength.[9] By taking the average of the reported mean LDL reductions across strengthes 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 reducing the bad cholesterol for each statin without the need of estimating them. If we had to estimate the effectiveness of reducing the bad cholesterol, it would be very hard for us to identify the learning parameters. We will discuss the identification strategy in subsection 5.5.

4.3.2 News Coverage

The news coverage data are obtained from Ching et al. (2016), which explains the details. We will briefly describe the data here. The data is collected from news articles 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

[9]While Table 2 uses mmol/L as unit of LDL reduction, the unit in Table 1 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.

extract its headline, source, content and publication date. We first map the information of each article into two multi-dimensional variables: (a) *general* publicity variable ($publicity_t^s$) – 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 ($publicity_{jt}$) – 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 can affect the overall demand for statins, while drug specific publicity influences both total demand for statins and which particular statin to use.[11]

We classify both *general* and *drug specific* publicity into three dimensions: lowering the bad cholesterol (lc), reducing heart disease risks (rh) and side-effects (se). Hereafter, we use (lc_t^s, rh_t^s, se_t^s) 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.

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 ($publicity_{t,l}^s, publicity_{jt,l}$) 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 ($publicity_t^s, publicity_{jt}$) are obtained by simply summing ($publicity_{t,l}^s, publicity_{jt,l}$)

[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 in this paper.

across the news stories appeared in quarter t. For example, $publicity_t^s = \sum_{l=1}^{L_t} publicity_{t,l}^s$.

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 the market, most news articles report that statins are effective in lowering the cholesterol level and reducing heart disease risks. Table 3 presents a descriptive summary of general publicity variables and drug specific publicity variables with entry quarter for each drug.

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 annual Canadian Heart Health Survey from 1986 and 1992. We multiply it by the total Canadian population for each age group in a given quarter (obtained from 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 to the number of prescriptions, we assume that each patient receives a prescription once per 90 days (see Cosh (2010) and Ching et al. (2016)).

4.5 Preliminary Evidence for Correlated Learning

Our hypothesis is that if correlated learning/information spillover is present, the return of detailing for one drug may benefit from the release of clinical trial for another drug. To look for some preliminary evidence for the presence of correlated learning/information spillover, we regress the number of prescriptions of a drug on brand dummies, brand specific detailing stock and clinical trial stock, and interactions between individual landmark clinical trial and detailing stock. More specifically, we allow the interaction term to be heterogeneous across drugs.

We let STK_detail_{jt} denote the detailing goodwill stock for drug j at time t,

$$STK\_detail_{jt} = \delta_d \cdot STK\_detail_{jt-1} + detail_{jt}, \tag{1}$$

where δ_d is the quarterly carryover rate for detailing; $detail_{jt}$ is detailing spending (in thousand dollars) for drug j in quarter t. We set $\delta_d = 0.95$.[12]

When constructing the clinical stock variable, we weigh each clinical trial by the number of patients. Following Azoulay (2002), we assume that clinical stocks do not depreciate over time. We let $STK\_clinical_{jt}$ denote the goodwill stock of clinical trials for drug j in quarter t,

$$STK\_clinical_{jt} = STK\_clinical_{jt-1} + n_{jt}, \tag{2}$$

where n_{jt} is the number of participants in the landmark trials for drug j released in quarter t (it equals zero if there is no landmark clinical trial for drug j released in quarter t).

Recall that the switching rate is very low. We therefore decide to focus on the number of prescription for new patients and switching patients (i.e., patients who decide not to use the drug they used last period), whose choices should not be affected by switching costs.

The demand from new patients and switching patients, d_{jt}^{ns},[13] can be expressed as :

$$d_{jt}^{ns} = d_{jt} - d_{jt-1} \cdot (1 - S_{jt} - D_{jt}), \tag{3}$$

where S_{jt} and D_{jt} are the switching and discontinuing rates of drug j at time t, respectively. We are able to obtain $\{d_{jt}^{ns}\}$ because we observe S_{jt}, D_{jt} and d_{jt}.

Moreover, with our sample size, we cannot estimate a model which include brand specific interactions between detailing stock and every landmark clinical trial. We therefore decide to run a series of regressions which include one set of interactions per landmark clinical trial. The results are reported

[12]The carryover rate used here is similar to what Berndt et al. (1996) found. We also tried $\delta_d = 0.90$. But the model with $\delta_d = 0.95$ produces much better fit, and hence we decide to use $\delta_d = 0.95$.

[13]Because the switching rate is very low, the vast majority of d_{jt}^{ns} comes from new patients' demand.

in Table 4. The set of drugs we consider includes Lipitor, Zocor, Mevacor, Pravachol and Lescol.[14] [15]

In our reduced form regressions, we consider the set of landmark clinical trials which were released after Liptor's entry date. However, because ALLHAT-LLT and PROSPER are released in the same quarter, and so as ALERT and ASCOT-LLA, we only include the dummies for PROSPER and ASCOT-LLA in the regressions. One should keep in mind that the impact of PROSPER also includes ALLHAT-LLT, and that of ASCOT-LLA includes ALERT as well. All these clinical trials provide positive news about the drug being investigated (see Table 1). Each specification in Table 4 corresponds to a clinical trial, and we present the regressions according to the chronological order of the clinical trials.

We discuss specification (1) first. AFCAPS/TexCAPS is a landmark trial for Mevacor. We will focus on the coefficients for the interaction between detailing stock and the dummy variable which indicates whether AFCAPS/TexCAPS is available in time t.

Interestingly, in terms of boasting the effectiveness of detailing, Lipitor benefits most from AFCAPS/TexCAPS, with positive statistically significant point estimate of 1416.81. Its effect for Zocor and Mevacor are not statistically significant. Both Pravachol and Lescol have negative interaction terms. Judging the point estimates, the ranking of the interaction terms are: Lipitor > Zocor > Mevacor > Pravachol > Lescol. Interestingly, it coincides with their ranking based on the efficacy of lowering cholesterol (see Table 2). These patterns generally hold across landmark clinical trials.

Why some interaction terms are insignificant and some are negative? What could explain this asymmetric effect? On one hand, if correlated learning is important, we expect every drug should benefit from a landmark clinical trial regardless which drug the trial studies. But on the other hand, if

[14] We exclude Crestor because it entered the market near the end of the sample period (Q1 2003). There is only one landmark clinical trial released after its entry and before our sample ends it is CARDS for Lipitor, which was released in Q3 2004. The last observation of our sample period is Q4 2004. So there is only one observation after CARDS, which is not enough to identify is its effect.

[15] We also exclude Baycol. Baycol was withdrawn shortly after its introduction due to serious side-effects. It was on the market from Q1 1998 to Q2 2001, with 13 quarters of observations. [Hyunwoo: Please confirm!]. There were only two landmark clinical trials: LIPID was released in Q4 1998, and GISSI was released in Q4 2000. Hence, we do not have enough observations to identify the impacts of these two clinical trials on the demand for Baycol (we have too few observations before LIPID and after GISSI).

for some reasons, Lipitor benefits most from the information spillover, it generates competition effect which could negatively affect the demand for other statins. Depending on how much information spillover other statins can benefit, this competitive effect may lead to zero net effect or negative net effect in the interaction term.

Let us now focus on the pattern that the order of the interaction terms broadly follows that of drugs' effectiveness in lowering cholesterol. This pattern begs a theory to explain it. This leads us to hypothesize that doctors may be uncertain about to what extent a statin can translate its lowering cholesterol ability to lowering heart disease risks. The landmark clinical trial provides us with information to update the belief about this link for all drugs. Positive information would lead doctors to revise the strength of this link upwards. Consequently, a statin which is more effective in lower cholesterol could end up benefit more from a clinical trial more even though it studies a different statin and the information spillover is imperfect. Our structural model will capture this theory.

We should also point out another interesting pattern. The size of the interaction terms tend to decline as we move from earlier to more recent landmark trials. Such pattern is consistent with a learning model. As the information set accumulates more signals, the later signals would have less impact on it.

5 Model and Estimation

In this section, we propose a structural demand model incorporating physicians' correlated learning about clinical trial outcomes. We also discuss the identification issues and how to construct the likelihood function. 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 belief may suggest another drug is superior in ex-ante.

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

$$U_{ijt} = \omega \cdot q_j^h + \lambda_j + \epsilon_{ijt}, \tag{4}$$

where q_j^h denotes drug j's efficacy in reducing heart disease risks; λ_j captures time-invariant brand specific preference (e.g., price difference across brands[16]); ϵ_{ijt} is an i.i.d. extreme value distributed random shock.

We assume that the physician chooses a drug to maximize his expected utility for the patient plus his utility due to persuasive detailing.[17] 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, we assume that a physician does not receive 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:

$$E[U_{ijt}|I(k,t)] = \omega \cdot E[q_j^h|I(k,t)] + \lambda_j + \epsilon_{ijt}, \tag{5}$$

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.

Let q_j^c be the efficacy in lowering the cholesterol level of drug j, and β_j be the *efficiency ratio*. We define the *efficiency ratio* as a measure on how efficiently a drug can convert *reduction in the bad cholesterol level* to *reduction in heart disease risks*. Then, q_j^h can be expressed as follows:

$$q_j^h = q_j^c \cdot \beta_j. \tag{6}$$

[16] 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.

[17] We will discuss how to model persuasive detailing in subsection 5.2.

We also 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 (β_j).[18] Consequently, physician k's expectation about q_j^h can be expressed as follows.

$$E[q_j^h|I(k,t)] = q_j^c \cdot E[\beta_j|I(k,t)]. \tag{7}$$

How do physicians learn about β_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 the cholesterol level, their initial prior belief about β_j's may be correlated across j. In other words, information about β_j can be useful for updating β_{-j}, and vice versa. Due to this intrinsic correlated prior belief, physicians may infer q_j^h indirectly from the clinical trial evidence on β_{-j}. More specifically, we assume the initial prior is normally distributed and allow the off-diagonal elements (ρ) 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.

Because it is unlikely for physicians to learn about the efficacy in heart disease risks of statins from their patients' experiences, we assume landmark clinical trials (which are specifically designed to prove the efficacy of drugs in heart disease risks) are the only sources of information about this dimension of efficacy. Physicians are assumed to update their beliefs on β_j 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 select patients with different conditions (e.g., one trial may use participants with diabetes, and another trial uses participants who had a heart attack before). Hence, β's obtained from different landmark clinical trials should not be the same even if the number of patients goes to infinity. We assume that the true efficiency ratio associated with the condition studied by trial l ($\bar{\beta}_{jl}$) is capture

[18] 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.

by:

$$\bar{\beta}_{jl} = \beta_j + \epsilon_{jl}, \tag{8}$$

where β_j is the true mean level of the efficiency ratio for drug j; $\epsilon_{jl} \sim N(0, \sigma_\epsilon)$ and is i.i.d. But the exact signals revealed by clinical trial l could differ from $\bar{\beta}_{jl}$ because of sampling errors. More precisely, the signal from clinical trial l for drug j ($\tilde{\beta}_{jl}$) can be expressed as:

$$\tilde{\beta}_{jl} = \bar{\beta}_{jl} + \zeta_{jl}, \tag{9}$$

where ζ_{jl} is a i.i.d. signal noise, and $\zeta_{jl} \sim N(0, \sigma^2_{\zeta l})$. Let σ^2_ζ 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 $\sigma^2_{\zeta l} = \sigma^2_\zeta / N_l$. This implies that the more participants a clinical trial has, the more confidence physicians have about its results. Moreover, we can combine the two equations above and write,

$$\tilde{\beta}_{jl} = \beta_j + \nu_{jl}, \tag{10}$$

where $\nu_{jl} = \epsilon_{jl} + \zeta_{jl}$. This implies that $\nu_{jl} \sim N(0, \sigma^2_{\nu l})$, where $\sigma^2_{\nu l} = \sigma^2_\epsilon + \frac{\sigma^2_\zeta}{N_l}$. 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 procedure.

To explain how physicians update their beliefs through learning about clinical trials, let us provide a simplified example which can be easily generalized.[19] In the appendix, we provide a more general model. 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 β_{jt} be the expected perceived efficiency ratio, and $\sigma^2_{\beta jt}$ be the perceived variance of drug j, conditional on the physician

[19] Our model extends correlated learning models by Erdem (1998); Erdem and Keane (1996); Marcoul and Weninger (2008).

k's information set at time t. The variance-covariance matrix for prior beliefs of physician k at time t becomes,

$$V[\beta_j|I(k,t)] = \begin{pmatrix} \sigma^2_{\beta 1t} & \pi_t \\ \pi_t & \sigma^2_{\beta 2t} \end{pmatrix}. \tag{11}$$

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

$$\beta_{1t+1} = \beta_{1t} + \frac{\sigma^2_{\beta 1t}}{\sigma^2_{\beta 1t} + \sigma^2_{\nu 1l}} \cdot (\tilde{\beta}_{1l} - \beta_{1t}). \tag{12}$$

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

$$\sigma^2_{\beta 1t+1} = \frac{\sigma^2_{\beta 1t}\sigma^2_{\nu 1l}}{\sigma^2_{\beta 1t} + \sigma^2_{\nu 1l}}. \tag{13}$$

With correlated prior beliefs on the efficiency ratio, signals for drug 1 are used to update the posterior mean on drug 2 as well. The posterior mean for drug 2 is given by:

$$\beta_{2t+1} = \beta_{2t} + \frac{\pi_t}{\sigma^2_{\beta 2t} + \sigma^2_{\nu 1l}}(\tilde{\beta}_{1l} - \beta_{1t}), \tag{14}$$

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

The posterior variance on the efficiency ratio of drug 2 at time t becomes

$$\sigma^2_{\beta 2t+1} = \sigma^2_{\beta 2t} - \frac{\pi_t^2}{\sigma^2_{\beta 2t} + \sigma^2_{\nu 1l}}. \tag{15}$$

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

$$\pi_{t+1} = \frac{\pi_t \sigma^2_{\nu 1l}}{\sigma^2_{\beta 1t} + \sigma^2_{\nu 1l}}. \tag{16}$$

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

$$V[\beta_j|I(k,t+1)] = \begin{pmatrix} \frac{\sigma^2_{\beta 1t}\sigma^2_{\nu 1l}}{\sigma^2_{\beta 1t}+\sigma^2_{\nu 1l}} & \frac{\pi_t\sigma^2_{\nu 1l}}{\sigma^2_{\beta 1t}+\sigma^2_{\nu 1l}} \\ \frac{\pi_t\sigma^2_{\nu 1l}}{\sigma^2_{\beta 1t}+\sigma^2_{\nu 1l}} & \sigma^2_{\beta 2t} - \frac{\pi_t^2}{\sigma^2_{\beta 2t}+\sigma^2_{\nu 1l}} \end{pmatrix}. \tag{17}$$

Before leaving this subsection, it is worth making two remarks here. First, one may argue that q_j^c could also have a direct effect in the utility function. We believe this is plausible. However, q_j^c does not vary over time. Hence, its effect cannot be disentangled from the brand dummy, λ_j. When interpreting λ_j, it is important to keep in mind that it includes all drug specific factors that are time-invariant (including q_j^c and price of drug j). Second, one might think that physicians could 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 assume that physicians do not experiment different drugs on their patients.

5.2 Roles of Detailing and Publicity

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). The official role of detailing is to inform physicians about drug's efficacies and side effect – this is referred to the informative role. However, it's been argued that other non-informative activities (such as offering physicians free meals and gifts) could also cause physicians to favor prescribing the drug being detailed – this is referred to the persuasive role. 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(5) as follows:

$$U_{kjt} = E[U_{ijt}|I(k,t)] + \kappa_d \cdot P\_STK\_detail_{jt}, \tag{18}$$

where $P\_STK\_detail_{jt}$ is a persuasive detailing goodwill stock for drug j at time t. The persuasive detailing stock is defined as:

$$P\_STK\_detail_{jt} = \delta_{d\_per} \cdot P\_STK\_detail_{jt-1} + detail_{jt}, \tag{19}$$

where δ_{d_per} is the quarterly carryover rate for persuasive detailing; $detail_{jt}$ 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 (hereafter, we refer them to *well-informed physicians*). 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 ($\underline{I}_j$).

Let $I\_STK\_detail_{jt}$, $I\_STK\_detail_{-jt}$ and STK_rh_{jt} denote the informative stocks of detailing, competitors' detailing stock, and publicity stock in reducing heart disease risks for drug j at time t, respectively. We model the measure of well-informed physicians of drug j at time t (M_{jt}) as follows.

$$M_{jt} = \frac{exp(\alpha_0 + \alpha_d \cdot I\_STK\_detail_{jt} + \alpha_c \cdot I\_STK\_detail_{-jt} + \alpha_{rh} \cdot STK\_rh_{jt})}{1 + exp(\alpha_0 + \alpha_d \cdot I\_STK\_detail_{jt} + \alpha_c \cdot I\_STK\_detail_{-jt} + \alpha_{rh} \cdot STK\_rh_{jt})}. \tag{20}$$

Note that we allow M_{jt} to depend on $I\_STK\_detail_{-jt}$ to capture the possibility that sales reps of drug j might have discussed about other statin's clinical trials to free-ride on competitors' clinical trial results and take advantage of correlated learning.[20] Moreover, for drug specific publicity, we only include rh_{jt} here because we assume physicians already know lc_{jt}.

The informative detailing stock is defined as:

$$I\_STK\_detail_{jt} = \delta_{d\_inf} \cdot I\_STK\_detail_{jt-1} + detail_{jt}, \tag{21}$$

where δ_{d_inf} is the quarterly carryover rate for informative detailing; $detail_{jt}$ denotes the flow of detailing

[20] Liu et al. (2016) discuss a different type of detailing spillover effect when drugs are sold as a bundle (e.g., HIV combination therapy). There is no consumer uncertainty about drug qualities and hence no consumer learning in Liu et al. (2016).

spending for drug j at time t. The informative stock of competitors' detailing is defined as:

$$I\_STK\_detail_{-jt} = \delta_{d\_inf} \cdot I\_STK\_detail_{-jt-1} + detail_{-jt}, \tag{22}$$

where $detail_{-jt}$ denotes the flow of sum of detailing spending for all statins except for drug j at time t. We define STK_rh_{jt} in a similar way.

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/her 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 his/her patient to get a blood test. Therefore, we model the general publicity and "aggregate" 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:

$$P_t(statin) = \frac{exp(\gamma_0 + \gamma_i \cdot Inclusive_t + \gamma_p \cdot STK\_PUB_t)}{1 + exp(\gamma_0 + \gamma_i \cdot Inclusive_t + \gamma_p \cdot STK\_PUB_t)}, \tag{23}$$

where $Inclusive_t$ is the inclusive value term derived from the brand choice stage; STK_PUB_t denotes a vector of three types of general publicity (rh_t^s, lc_t^s, se_t^s) stocks for the class of statin.[21]

If a potential patient decides to use statins, he/she moves 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

[21] We assume that they all share the same carryover rate.

physician k chooses drug j for a new patient, conditional on prescribing, is expressed as follows:

$$P_t(j|statin, k_{type}) = \frac{exp(U_{kjt}(k_{type}))}{\sum_{r=1}^{J} exp(U_{krt}(k_{type})))}. \tag{24}$$

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 them at all, the total number of physician types is 2^H. 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, $\hat{d}^1_{jt}$, can be expressed as:

$$\hat{d}^1_{jt} = (m_t - \sum_{r=1}^{J} d_{rt-1}) \cdot P_t(statin) \cdot \sum_{k_{type}=1}^{2^H} P_t(k_{type}) \cdot P_t(j|statin, k_{type}), \tag{25}$$

where d_{jt} is the demand for drug j at time t (to be defined later); m_t is the potential market size for statins at time t; $(m_t - \sum_{r=1}^{J} d_{rt-1})$ is the potential patient pool have not yet adopted 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 need to decide whether to stay with the same statin or switch to other statins. If they decide to switch, then they choose a statin. Note that some patients might keep taking the same statin even though there are alternatives which give them better expected utility because of switching costs,[22]

If an existing patient decides to stay with the current statin, we classify the patient as a "stayer." If he decides to switch to another statin, we classify him as a "switcher."

[22]Many statin users are elderly, and they may find it troublesome to switch to a different drug and worried about its potential side-effects.

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

$$\hat{d}^2_{jt} = d_{jt-1} \cdot (1 - S_{jt} - D_{jt}), \tag{26}$$

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

The expected demand for switchers (group 3) can be expressed as :

$$\hat{d}^3_{jt} = \sum_{m=1,\neq j}^{J} \{d_{mt-1} \cdot S_{mt} \cdot \sum_{k_{type}=1}^{2^H} [P_t(k_{type}) \cdot \frac{exp(U_{kjt}(k_{type}))}{\sum_{r=1,\neq m}^{J} exp(U_{krt}(k_{type})))}]\}. \tag{27}$$

Note that once a patient quit using the statin treatment, he will be back to the potential patient pool in the next period. Also, 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:

$$d_{jt} = \hat{d}^1_{jt} + \hat{d}^2_{jt} + \hat{d}^3_{jt} + e_{jt}, \tag{28}$$

where e_{jt} represents a measurement error. $\hat{d}^1_{jt}$, $\hat{d}^2_{jt}$ and $\hat{d}^3_{jt}$ 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 (28) is normally distributed, we can obtain the likelihood function:

$$l(\{d_{jt}\}_{j=1}^{J} | \{\{detail_{j\tau}\}_{j=1}^{J}\}_{\tau=1}^{t}, \{\tilde{\beta}_{jl}\}_{l=1}^{C_t}, \{N_l\}_{l=1}^{C_t}, \{PUB^s_\tau\}_{\tau=1}^{t}, \{\{PUB_{jt}\}_{j=1}^{J}\}_{j=1}^{J}; \theta_d), \tag{29}$$

where θ_d is the vector of parameters; $detail_{jt}$ is detailing spending for drug j at time t; C_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 participants in clinical trial l; PUB_t^s and PUB_{jt} are vectors of general publicity and drug specific publicity, respectively. The likelihood of observing $d = \{\{d_{jt}\}_{j=1}^J\}_{t=1}^T$ is

$$\begin{aligned} & L(d|\{detail_{jt}\}_{j=1}^J\}_{t=1}^T, \{\tilde{\beta}_{jl}\}_{l=1}^{C_t}, \{N_l\}_{l=1}^{C_t}, \{PUB_t^s\}_{t=1}^T, \{\{PUB_{jt}\}_{j=1}^J\}_{t=1}^T; \theta_d) \\ = & \prod_{t=1}^T l(\{d_{jt}\}_{j=1}^J | \{\{detail_{j\tau}\}_{j=1}^J\}_{\tau=1}^t, \{\tilde{\beta}_{jl}\}_{l=1}^{l_t}, \{N_l\}_{l=1}^{l_t}, \{PUB_\tau^s\}_{\tau=1}^t, \{PUB_{jt}\}_{j=1}^J; \theta_d). \end{aligned} \tag{30}$$

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.[23]

Endogeneity Problem of Detailing

When estimating a demand model using product level data, one potential concern is that detailing (firms controlled marketing tool) is endogeneous. From the econometric viewpoint, this would be an issue if there are unobserved demand shocks which firms observe before setting their detailing efforts (e.g., Ching and Ishihara, 2010). In most existing works of estimating the demand model for prescription drugs, the unobserved demand shocks are usually due to omitted information such as release of new

[23]It is unlikely that there is much news about statins available prior to Q1 1986 because the first statin was launched in Q2 1988.

clinical trials results and news shocks (e.g., Azoulay 2002, Ching and Ishihara 2010). Here, we have collected data on clinical trials and news coverage to control for these two sources of demand shocks. As a result, the endogeneity problem should be alleviated.[24]

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 (γ_0, γ_i, γ_{lc}, γ_{rh}, γ_{se}) can be identified by the variation of market share of statins as a whole and the variation of the explanatory marketing variables, such as general publicity and inclusive values.

To understand how to identify the correlation term in the initial prior, it is important to stress that the quality signals from clinical trials are observable to us. Note 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 points of time, the existing stocks of landmark clinical trials faced by them also differ when they entered the market. As long as $\rho > 0$, the entry date prior beliefs will differ across drugs. Hence, after controlling for the observed quality signals from clinical trial outcomes, q_j^c's and detailing stocks, the differences of initial sales across drugs will help us identify ρ (because it is a function of the entry date prior beliefs).

Correlations in the initial prior beliefs (ρ) 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. This is because the change in market shares before and after the release of a clinical trial can be influenced by both the realized quality signal from the clinical trial and the extent of correlated learning. For example, if a

[24] One might argue that landmark clinical trials are funded by drug companies and hence they may also be endogeneous. We note that landmark clinical trials took years to complete. Given that we have also included drug fixed effect, it seems unlikely that the landmark clinical trial's release dates and outcomes are correlated with the demand shocks. Ching et al. (2016) make use of the same argument to justify treating detailing and publicity variables as exogenous from the estimation viewpoint.

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. Note that in principle, one can allow ρ to be drug specific. However, in the current application, we have decided to restrict ρ to be the same across drugs mainly because there are only 14 landmark clinical trials for statins in our sample period.

The parameters that determine the persuasive (κ_d) and informative detailing (α_d) can be separately identified because 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. Therefore, the variation in 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 5 shows the parameter estimates. The first section in the table describes learning parameters. Physicians' initial prior mean on efficiency ratio is 0.0027. As shown in Table 1, 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 low compared with the true efficiency ratios. The initial prior variance (σ_β^2), signal variance from different clinical trial designs (σ_ϵ^2), and the signal variance per 1000 patients (σ_ζ^2), are all statistically significant. The initial prior correlation on efficiency ratio (ρ) is 0.864. 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.

To demonstrate the rate of learning, in Figure 8 we graph the posterior belief of a well-informed physician (i.e., $E[\beta_{jt}]$) over time based on our estimated model. Recall that a well-informed physician 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 were no landmark clinical trials to support statins' efficacy in reducing heart disease risks. Hence, the physician has exactly the same prior belief about Mevacor, Zocor and Pravahol before Q4 1994. In Q4 1994, Zocor received the first landmark clinical trial (4S study) supporting its efficacy in reducing heart disease risks. Then, the well-informed physician updates her beliefs about all statins (not just Zocor). However, because the information spillover is not 100%, the physician's belief on Zocor is slightly higher than those on other statins. We can see similar imperfect information spillover patterns happened when other clinical trials are released.

Before Lipitor releases any landmark clinical trials (i.e., before Q2 2003), $E[\beta_{Lipitor,t}]$ is the lowest

among all existing statins. However, after the second landmark clinical trial of Lipitor is released in Q3 2004, $E[\beta_{Lipitor,t}]$ became the highest among all statins. Figure 8 suggests that Lipitor benefits much from other statins' investment in landmark clinical trials. But it appears that there is still room for Lipitor to improve from its own investment in landmark trials. In one of our counterfactual experiments, we will investigate this further by examining how the evolution of $E[\beta_{j,t}]$ translate to sales.

As expected, we find that firms' own detailing goodwill stock (α_d) has positive and significant effect on sales. The results indicate that detailing plays an informative role in physicians' prescription choices. Brand specific publicity in reducing heart disease risks (α_{rh}) is also positive and significant, indicating that it can help inform physicians (perhaps the pressure of patients prompts physicians to look into the latest clinical evidence of a drug) 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. The results here are consistent with the findings from the reduced form analysis done in Ching et al. (2016).

We also find evidence that rivals' detailing could help inform physicians' about a drug's clinical trials (α_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. Note that this result is not reported in Ching et al. (2016) because their reduced form model does not consider the possibility that a drug's own clinical trials stock can interact with rivals' detailing.

Figure 9 shows how the measure of well-informed physicians changes over time by drug. For all statins, the evolutions appear to be in S-shape. For the earlier half of the sample period, the measure of well-informed physicians about Lipitor are lower than those who are well-informed about Zocor and Pravachol. This is because Lipitor is a late entrant, and it takes time for Lipitor to build up a stock

of physicians who are well-informed about Lipitor. But in Q4 2001, Lipitor has more well-informed physicians than other statins and its measure rises to above 0.9 by Q4 2004. As a comparison, its next competitors, Zocor and Pravachol, reach at around 0.8 and 0.75, respectively by Q4 2004.

As for the utility parameters, we find that the coefficient for the perceived q^h (ω) to be positive and significant. We also find that the persuasive detailing parameter (κ_d) is positive and significant. This indicates that other than its informative role, detailing also influences physicians' decisions via its persuasive role.

Next, we discuss parameters in the adoption decision stage. The estimate of the inclusive value term is positive (1.02) and significant. This indicates that detailing and publicity stocks in the brand choice stage has positive influence on the adoption decision in the first place. The stock of general publicity in reducing heart disease risks ($Stk\_rh_t^s$) is also estimated to be positive and significant, but the stock of lowering cholesterol ($Stk\_lc_t^s$) is insignificant. However, we do not want to read too much into the estimates of these two variables because these two stock variables are highly correlated. Interestingly, the coefficient on the stock of general publicity in side-effects ($Stk\_se_t^s$) 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 and switching patients, we will focus on studying how the demand for new patients and switchers would change in the first two counterfactual experiments. But the third counterfactual experiment will examine the total demand when we remove the switching costs.

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 shed some light on this question, 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 and switching patients' demand for statins. The dotted lines denote the counterfactual new and switching patients' demands. Without its own landmark clinical trials, the counterfactual demand for Lipitor due to new and switching patients is 9% 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. This is roughly about 2.5% of the actual total demand in Canada.

The magnitude of the change might appear to be small. However, Liptior's global annual sales is almost $10.9 billion in 2004. Therefore, even just 2.5% loss in sales would cost about $250 million per year. According to U.S. Department of Health and Human Services, the average cost of Phase 4 clinical trials (i.e., postmarketing clinical trials) for cardiovascular drugs is $27.8 million (https://aspe.hhs.gov/sites/default/files/pdf/77166/rpt_erg.pdf).[25] Hence, Lipitor's investment in its own post-marketing clinical trials appears to be justifiable from profits viewpoint.

It is also worth pointing out that the counterfactual demand for other drugs also drops but at a

[25]Phase 4 refers to postmarketing studies to provide additional information such as treatment's risks, benefits, and optimal use.

smaller magnitude. For Crestor, the new and switching patients' demand decreases by 4% - 7% during Q4 2002 to Q3 2004. For Zocor and Pravachol, it also drops but by a even smaller percentage. We see negative effects on other drugs because we take away the positive externality generated by the clinical trials of Lipitor. The reason why the effects are asymmetric is because (i) the information spillover is not perfect; (ii) β works like a multiplier, and how a change in β affects a drug's q^h (and ultimately its demand) depends on its q^c. Since $q^c_{crestor} > q^c_{zocor} > q^c_{pravachol}$, the magnitudes of the changes in demand across drugs reflects this order.

Experiment 2 (Removing Correlated Learning)

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 generate 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 (ρ_0) to be zero. Figure 12 presents the benchmark and counterfactual demand due to new and switching patients. It is striking to see that Lipitor's counterfactual demand is almost 50% lower than its benchmark demand from the beginning of sample period until Q1 2003. After Lipitor's first landmark clinical trial is launched in Q2 2003, the difference between the counterfactual and benchmark demand sharply drops and then quickly converges to about 1% in Q3 2004 (amounts to about 4,700 prescriptions). For Crestor, the counterfactual demand is consistently about 46-52% lower than the benchmark demand for most quarters. The difference is very substantial, and it indicates that correlated learning plays a very important role for the early success of both Lipitor and Crestor.

However, note that even without correlated learning, the demand for Lipitor from new and switching patients keeps increasing over time. This suggests that correlated learning is not the only driving force

for the early success of Lipitor. So what else can contribute to its success? Because Lipitor had the highest q^c before Crestor was launched, Lipitor could still have a high $E[q^h]$ even with a relatively low $E[\beta]$ (see Eq(3) and (4)). 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 in the absence of correlated learning, Lipitor has the lowest $E[q^h]$ up until Q2 2003. Hence, we can rule out this explanation. When examining our parameter estimates closer, we see that increasing demand for Lipitor in the counterfactual situation is due to its large brand dummy. The large value of Lipitor's brand dummy may partly capture the possibility that q^c has a direct effect on physicians' or patients' utility, which our current specification does not explicitly model.

It is interesting that the demand for Zocor also drops over time. This shows that Zocor also benefits from correlated learning (i.e., the information spillover due to clinial trials done by other drugs). However, we see that the demand for Pravachol hardly changes in the counterfactual situation. During 1997-1998, Mevachol releases two landmark clinical trials. However, they benefit Pravahcol very little because of its small q^c. Again, we can see the asymmetric effects of information spillover on sales aligns with the asymmetry of q^c.

Experiment 3 (Removing Switching Costs)

We have argued that correlated learning generates late-mover advantage. But we should not overlook that there is a source of first-mover advantage in this market, namely *switching cost* as evident from the low observed switching rates. Although we do not estimate switching costs, we can still conduct a counterfactual experiment to shed light on its importance by assuming that all existing statin users make their statin choice in each period as if the switching costs do not exist. In other words, for an existing statin user, if he/she decides to keep taking a statin, then in each period the doctor would treat him/her like a new patient and choose a statin out of the entire set of statins available in the market. However, we still take the observed discontinuing rate as given and apply it to this counterfactual

scenario.

Figure 14 shows the counterfactual demand under this scenario. Without the switching costs, it is clear that when a new drug with more superior $E[q^h|I(t)]$ (compared with incumbent drugs) can become the market leader much quicker in the counterfactual world. For Lipitor, our simulation result shows that it would become the market leader as soon as it entered the market. Moreover, the counterfactual demand for Zocor and Pravachol are higher than that for Mevacor at the beginning of the sample period (Q1 1993); but in the actual world, Zocor and Pravachol did not surpass Mevacor until Q2 1996. We also see that when Crestor entered, it would be able to steal a significant portion of demand from Lipitor and other statins immediately (we see the discrete jumps in the figure).

It is also worth noting that Zocor raised its detailing expenditure during 2001-2002 (see Figure 2), possibly because the release of a landmark clinical trial, HPS. We see that the counterfactual demand for Zocor is much more responsive to the increase in detailing, and it went up much faster than the benchmark demand. Then the counterfactual demand drops quickly upon Crestor's entry, while the benchmark demand is much more resistent to Crestor. It demonstrates that although the first-mover advantage of switching costs does not help incumbents to obtain new patients, it is very successful in protecting incumbents from losing its base patients.

7 Extension

We also consider a version of the model where patients (and hence physicians) directly care about q_j^c. The utility of patient i who consumes drug j at time t is given by

$$U_{ijt} = \omega_h \cdot q_j^h + \omega_c \cdot q_j^c + \lambda_j + \epsilon_{ijt}, \tag{31}$$

where q_j^h and q_j^c denote drug j's efficacy in reducing heart disease risks and in lowering cholesterol levels, respectively.

Instead of assuming all physicians have complete information about q_j^c, we assume that only the

well-informed physicians know the true q_j^c and the uninformed physicians have an initial prior belief, $\underline{q}_j^c$. Hence, physician k's expected utility of prescribing drug j to patient i at time t will be:

$$E[U_{ijt}|I(k,t)] = \omega_h \cdot E[q_j^h|I(k,t)] + \omega_c \cdot E[q_j^c|I(k,t)] + \lambda_j + \epsilon_{ijt}, \tag{32}$$

We further assume that q_j^c and β_j are independent. Consequently, physician k's expectation about q_j^h can be expressed as follows.

$$E[q_j^h|I(k,t)] = E[q_j^c|I(k,t)] \cdot E[\beta_j|I(k,t)], \tag{33}$$

We estimate this extension and the results are reported in Table 6. In this version, we have also included more publicity variables to explain the measure of well-informed physicians. In general, the results are quite similar to what we report earlier. In particular, we find that the correlated learning parameter is still significant, and at the magnitude similar to our benchmark model. However, we also see that the utility weight for q_j^c is not significant. This provides a justification for our assumption that patients and physicians primarily care about q_j^h.

8 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 late movers (especially for 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 using the clinical trial

results. By observing the information signals, it also helps us identify the correlated learning parameters. Our data on the content of clinical trials also help us identify the informative effect of detailing.

In addition to using product level market share data, we supplement them with switching rates and discontinuing rates. These data allow us to take the presence of switching costs and refilling costs into consideration, and focus on estimating the demand model for new patients and switchers and avoid a serious source of misspecifying the model.

Based on our results, it maybe tempting to advise managers that they should postpone launching their products to free-ride information generated by the incumbents. But such advices overlook the importance of switching costs, which allows early entrants to gain first-mover advantages by locking in early adopters of statins. Another point to note is that pharmaceutical companies usually file for patents protection for their drugs before gaining approval for selling them. Since the patent protection will expire after a fixed period of time, this factor would discourage firms to delay launching their drugs because that would shorten the duration of their monopoly position for selling them.

So to find out the optimal entry time, it is important to evaluate these trade-offs. A complete analysis would require us to understand the nature of switching costs, and project the future demand for the drugs (taking into consideration of the remaining patent life and generic competition afterwards) under different entry times. Although such an analysis is beyond the scope of this paper, we hope our research has taken us one step further in this research direction.

Last but not least, we should point that the model developed here could 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

may then infer that a Samsung phone is better than an iPhone. This can explain why Samsung has quickly become the largest smartphone player even though it entered the smartphone market after Apple.

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Table 1: Landmark Clinical Trials for Statins

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

Table 2: Statins' Mean Cholesterol Reduction by Strength (mmol/L)

	Daily Dose (mg)					Mean
	5	10	20	40	80	
Mevacor	N/A	1.02	1.40	1.77	2.15	1.59
Zocor	1.08	1.31	1.54	1.78	2.01	1.66
Pravachol	0.73	0.95	1.17	1.38	1.60	1.28
Lescol	0.46	0.74	1.02	1.30	1.58	1.16
Lipitor	1.51	1.79	2.07	2.36	2.64	2.22
Crestor	1.84	2.08	2.32	2.56	2.80	2.44

Table 3: Summary of Publicity Variables

Dimension	# of Quarters	General Publicity			
		Mean	Std. Dev.	Min.	Max.
Lowering Cholesterol Levels	47	11.59	14.07	0	65.00
Reducing Heart Disease Risks	47	4.84	6.37	0	25.00
Side Effects	47	-0.37	2.20	-13	2.00

Drug	Entry Quarter	# of Quarters	Drug Specific Publicity in Reducing Heart Disease Risks			
			Mean	Std. Dev.	Min.	Max.
Mevacor	Q3/1988	47	0.84	1.77	-1.58	7.13
Zocor	Q3/1990	47	2.01	3.15	-3.00	14.25
Pravachol	Q4/1990	47	1.97	2.68	0	12.67
Lescol	Q1/1994	44	0.07	0.25	0	1.00
Lipitor	Q1/1997	32	2.98	4.28	0	16.15
Baycol	Q1/1998	14	0.07	0.27	0	1.00
Crestor	Q1/2003	8	0.84	1.07	0	2.85

Table 4: Preliminary Evidence for Correlated Learning

Vairables	(1)		(2)		(3)		(4)	
	Estimates	S.E.	Estimates	S.E.	Estimates	S.E.	Estimates	S.E.
STK_Detail_{jt} (In Thousand Dollars)	**1,519.20**	185.47	**1,478.52**	168.58	**1,245.33**	133.88	**1,829.82**	139.54
$STK\_Clinical_{jt}$	**2.15**	0.23	**2.24**	0.24	**1.51**	0.26	**1.23**	0.36
Lipitor	**50,551.16**	10,535.49	**69,405.80**	8,964.80	**105,966.20**	7,025.45	**118,377.50**	8,176.97
Zocor	-2,040.35	6,668.62	-502.93	6,437.79	4,713.42	5,900.29	-3,901.97	6,634.14
Mevacor	**-19,332.84**	7,620.78	**-19,384.58**	7,233.03	**-11,906.08**	5,916.81	**-28,900.97**	5,964.95
Pravachol	**-23,779.38**	7,344.16	**-23,313.41**	7,088.09	**-18,683.57**	6,642.86	**-33,235.43**	7,450.66
Lescol	1,420.78	6,823.54	-1,851.94	6,412.28	**-11,020.88**	5,585.86	**-16,956.12**	6,156.08
STK_Detail_{jt} * Lipitor * AFCAPS	**1,416.81**	198.45						
STK_Detail_{jt} * Zocor * AFCAPS	119.81	125.34						
STK_Detail_{jt} * Mevacor * AFCAPS	-259.21	325.13						
STK_Detail_{jt} * Pravachol * AFCAPS	**-480.69**	130.99						
STK_Detail_{jt} * Lescol * AFCAPS	**-775.64**	179.19						
STK_Detail_{jt} * Lipitor * LIPID			**1,265.78**	163.14				
STK_Detail_{jt} * Zocor * LIPID			132.64	118.26				
STK_Detail_{jt} * Mevacor * LIPID			-238.78	379.41				
STK_Detail_{jt} * Pravachol * LIPID			**-545.25**	130.31				
STK_Detail_{jt} * Lescol * LIPID			**-666.31**	173.43				
STK_Detail_{jt} * Lipitor * GISSI					**1,207.84**	106.87		
STK_Detail_{jt} * Zocor * GISSI					**497.01**	118.83		
STK_Detail_{jt} * Mevacor * GISSI					54.87	548.94		
STK_Detail_{jt} * Pravachol * GISSI					-134.16	157.49		
STK_Detail_{jt} * Lescol * GISSI					-312.07	204.60		
STK_Detail_{jt} * Lipitor * LIPS							**708.43**	108.28
STK_Detail_{jt} * Zocor * LIPS							**492.72**	180.14
STK_Detail_{jt} * Mevacor * LIPS							1,377.55	884.96
STK_Detail_{jt} * Pravachol * LIPS							170.22	298.22
STK_Detail_{jt} * Lescol * LIPS							51.64	336.07
R Squared	0.955		0.954		0.953		0.935	
Adjusted R Squared	0.953		0.952		0.950		0.932	
Number of Observations	211		211		211		211	

Estimates shown in bold are significant at 5% level.

Definition of Variables are as follows:

STK_Detail_{jt}: Cumulative Stock of Detailing for Drug j at quarter t. Carryover rate is 95%.

$STK\_Clinical_{jt}$: Cumulative Clinical Oucomes for Drug j at quarter t. Carryover rate is 100%.

Mevacor, Zocor, Pravachol, Lescol and Lipitor denote a brand dummy for each drug.

AFCAPS, LIPID, GISSI and LIPS indicate observation periods after each clinical trial.

Table 4: Preliminary Evidence for Correlated Learning (Cont'd)

Vairables	(5)		(6)		(7)		(8)	
	Estimates	S.E.	Estimates	S.E.	Estimates	S.E.	Estimates	S.E.
STK_Detail_{jt} (In Thousand Dollars)	**1,891.49**	137.56	**2,023.55**	138.86	**2,149.10**	132.19	**2,260.77**	113.49
$STK\_Clinical_{jt}$	**1.25**	0.36	**1.41**	0.34	**1.45**	0.30	**1.83**	0.24
Lipitor	**118,758.70**	8,290.16	**119,225.90**	8,584.49	**118,557.60**	8,631.76	**119,245.70**	8,590.44
Zocor	-5,479.44	6,640.60	-8,337.09	6,859.05	-12,056.87	6,835.29	**-15,855.79**	6,572.21
Mevacor	**-30,470.27**	5,829.98	**-34,651.05**	5,899.36	**-37,809.74**	5,660.18	**-40,506.29**	5,081.96
Pravachol	**-35,190.41**	7,371.81	**-40,772.01**	7,557.44	**-45,701.84**	7,439.80	**-55,103.45**	7,085.52
Lescol	**-17,751.28**	6,141.47	**-18,203.34**	6,286.96	**-19,463.76**	6,190.17	**-19,945.32**	5,909.42
STK_Detail_{jt} * Lipitor * HPS	**671.29**	107.78						
STK_Detail_{jt} * Zocor * HPS	**524.90**	188.01						
STK_Detail_{jt} * Mevacor * HPS	1,510.35	950.21						
STK_Detail_{jt} * Pravachol * HPS	174.28	329.47						
STK_Detail_{jt} * Lescol * HPS	119.84	362.98						
STK_Detail_{jt} * Lipitor * PROSPER			**524.19**	111.79				
STK_Detail_{jt} * Zocor * PROSPER			**439.36**	198.91				
STK_Detail_{jt} * Mevacor * PROSPER			1,705.66	1,055.23				
STK_Detail_{jt} * Pravachol * PROSPER			139.36	340.45				
STK_Detail_{jt} * Lescol * PROSPER			176.43	406.43				
STK_Detail_{jt} * Lipitor * ASCOT					**432.26**	115.35		
STK_Detail_{jt} * Zocor * ASCOT					**546.73**	222.71		
STK_Detail_{jt} * Mevacor * ASCOT					2,100.46	1,256.25		
STK_Detail_{jt} * Pravachol * ASCOT					254.17	364.43		
STK_Detail_{jt} * Lescol * ASCOT					327.87	489.29		
STK_Detail_{jt} * Lipitor * CARDS							**679.37**	212.93
STK_Detail_{jt} * Zocor * CARDS							865.96	546.95
STK_Detail_{jt} * Mevacor * CARDS							2,670.29	3,304.06
STK_Detail_{jt} * Pravachol * CARDS							377.21	815.98
STK_Detail_{jt} * Lescol * CARDS							639.67	1,303.26
R Squared	0.934		0.929		0.927		0.924	
Adjusted R Squared	0.931		0.925		0.923		0.919	
Number of Observations	211		211		211		211	

Estimates shown in bold are significant at 5% level.

Definition of Variables are as follows:

STK_Detail_{jt}: Cumulative Stock of Detailing for Drug j at quarter t. Carryover rate is 95%.

$STK\_Clinical_{jt}$: Cumulative Clinical Oucomes for Drug j at quarter t. Carryover rate is 100%.

Mevacor, Zocor, Pravachol, Lescol and Lipitor denote a brand dummy for each drug.

HPS, PROSPER, ASCOT and CARDS indicate observation periods after each clinical trial.

Table 5: Parameter Estimates for the Benchmark Model

Variable Descriptions	Estimates	S.E.
Statin Choice Stage		
Learning Parameters		
$\underline{\beta}$ (Initial Prior Belief on Efficiency Raito)	0.0851	0.0210
σ_{β}^2 (Initial Prior Variance on Efficiency Raito)	1.2435	0.0962
σ_{ε}^2 (Signal Variance from Different Design)	0.5227	0.2069
σ_{ζ}^2 (Signal Variance from 1,000 Patients)	5.4968	2.2340
ρ_0 (Correlated Learning Parameter in Initial Prior)	0.8247	0.0607
Parameters Determining Measure of informed Physicians		
α_0 (Constant)	-5.7250	0.6151
α_d (Informative Detailing)	2.5943	0.2180
α_{d_c} (Informative Detailing of Competitors)	1.3840	0.0562
α_{lc} (Informative Publicity in Lowering Cholesterol Levels)	0.0216	0.3156
α_{lc_c} (Informative Publicity of Competitors in Lowering Cholesterol Levels)	-0.0094	0.0434
α_{lc_g} (Informative General Publicity in Lowering Cholesterol Levels)	0.3865	0.1662
α_{rh} (Informative Publicity in Reducing Heart Disease Risks)	1.2032	0.1640
α_{rh_c} (Informative Publicity of Competitors in Reducing Heart Disease Risks)	0.2504	0.3271
α_{rh_g} (Informative General Publicity in Reducing Heart Disease Risks)	0.1176	0.1986
δ_{d_inf} (Carryover Rate of Informative Detailing in Statin Choice)	0.8999	0.0143
δ_{rh_inf} (Carryover Rate of Informative Publicity in Statin Choice)	0.2142	0.0290
δ_{d_per} (Carryover Rate of Persuasive Detailing in Statin Choice)	0.9272	0.0077
Utilty Parmaeters		
ω_h (Coefficient of Perceived Quality in Reducing Heart Disease)	2.0144	0.3112
κ_d (Persuasive Detailing)	1.0735	0.0828
Brand Dummies		
Zocor	1.2683	0.0352
Pravachol	1.0809	0.0560
Lescol	-0.1991	0.2524
Lipitor	1.7644	0.0175
Baycol	0.3259	0.1135
Crestor	1.0314	0.0610
Adoption Decision Stage		
γ_0 (Constant)	-6.9597	0.1059
γ_i (Inclusive Value)	1.0395	0.0385
γ_{lc} (General Publicity Stock in Lowering Cholesterol Levels)	0.0434	0.0979
γ_{rh} (General Publicity Stock in Reducing Heart Disease Risks)	0.3272	0.1095
γ_{se} (General Publicity Stock in Side Effects)	-0.0297	0.0152
δ_p (Carryover Rate of Publicity in Adoption Decision)	0.9262	0.0053
Additional Parameter		
Standard Deviation of e_{jt} (in Hundred Thousand)	0.2319	0.0111
Log Likelihood	-2695.46	

Table 6: Parameter Estimates for the Extended Model

Variable Descriptions	Estimates	S.E.
Statin Choice Stage		
Learning Parameters		
$\underline{\beta}$ (Initial Prior Belief on Efficiency Raito)	0.0777	0.0193
σ_β^2 (Initial Prior Variance on Efficiency Raito)	1.2200	0.0967
σ_ε^2 (Signal Variance from Different Design)	0.5349	0.2086
σ_ζ^2 (Signal Variance from 1,000 Patients)	5.5081	2.3210
ρ_0 (Correlated Learning Parameter in Initial Prior)	0.8258	0.0642
Initial Priors on Efficacy in Lowering Cholesterol Levels		
Mevacor	1.6662	0.5516
Zocor	1.6360	0.3061
Pravachol	1.2312	0.5158
Lescol	1.1528	0.6955
Lipitor	2.2321	0.4991
Baycol	2.2073	0.9468
Crestor	2.4352	0.5280
Parameters Determining Measure of Well-informed Physicians		
α_0 (Constant)	-5.7230	0.4238
α_d (Informative Detailing)	2.5831	0.2284
α_{d_c} (Informative Detailing of Competitors)	1.4021	0.0581
α_{lc} (Informative Publicity in Lowering Cholesterol Levels)	0.0068	0.3339
α_{lc_c} (Informative Publicity of Competitors in Lowering Cholesterol Levels)	-0.0122	0.0445
α_{lc_g} (Informative General Publicity in Lowering Cholesterol Levels)	0.3196	0.2134
α_{rh} (Informative Publicity in Reducing Heart Disease Risks)	1.2007	0.1641
α_{rh_c} (Informative Publicity of Competitors in Reducing Heart Disease Risks)	0.2504	0.3383
α_{rh_g} (Informative General Publicity in Reducing Heart Disease Risks)	0.0920	0.2132
δ_{d_inf} (Carryover Rate of Informative Detailing in Statin Choice)	0.8999	0.0130
δ_{rh_inf} (Carryover Rate of Informative Publicity in Statin Choice)	0.2139	0.0290
δ_{d_per} (Carryover Rate of Persuasive Detailing in Statin Choice)	0.9265	0.0078
Utilty Parmaeters		
ω_h (Coefficient of Perceived Quality in Reducing Heart Disease Risks)	2.0043	0.2598
ω_c (Coefficient of Perceived Quality in Lowering Cholesterol Levels)	-0.0191	0.0590
κ_d (Persuasive Detailing)	1.0647	0.1089
Brand Dummies		
Zocor	1.2152	0.0584
Pravachol	1.0211	0.0668
Lescol	-0.2171	0.2620
Lipitor	1.7417	0.0232
Baycol	0.3292	0.1218
Crestor	1.0531	0.0652
Adoption Decision Stage		
γ_0 (Constant)	-7.0022	0.1031
γ_i (Inclusive Value)	1.0854	0.0453
γ_{lc} (General Publicity Stock in Lowering Cholesterol Levels)	0.0392	0.0522
γ_{rh} (General Publicity Stock in Reducing Heart Disease Risks)	0.3546	0.1058
γ_{se} (General Publicity Stock in Side Effects)	-0.0289	0.0065
δ_p (Carryover Rate of Publicity in Adoption Decision)	0.9250	0.0057
Additional Parameter		
Standard Deviation of e_{jt} (in Hundred Thousand)	0.2285	0.0113
Log Likelihood	-2692.00	

Figure 1: Quarterly Number of Total Prescriptions for Statins

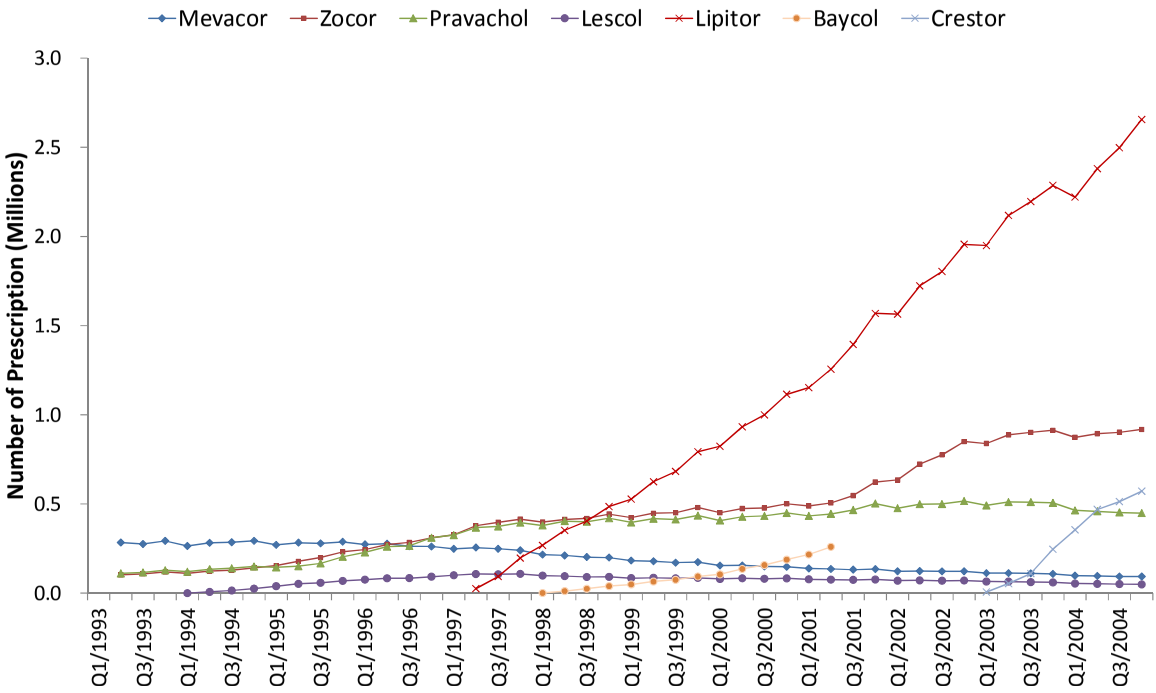


Figure 2: Quarterly Detailing Spending for Leading Statins

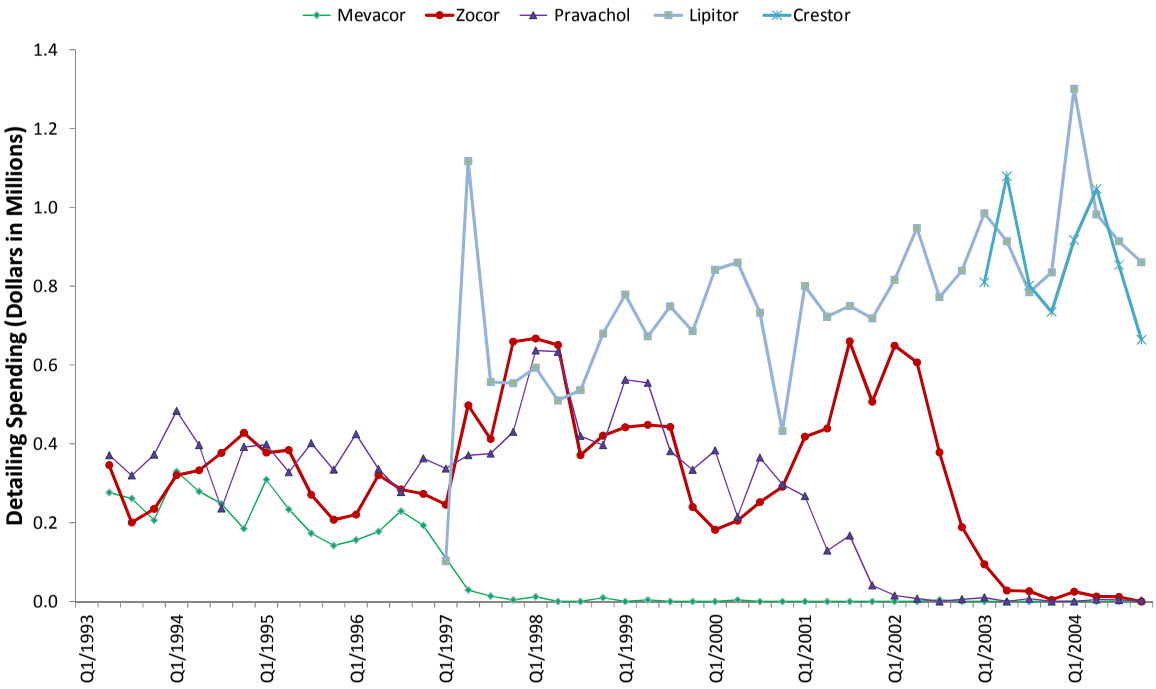


Figure 3: Quarterly Switching Rate for Leading Statins

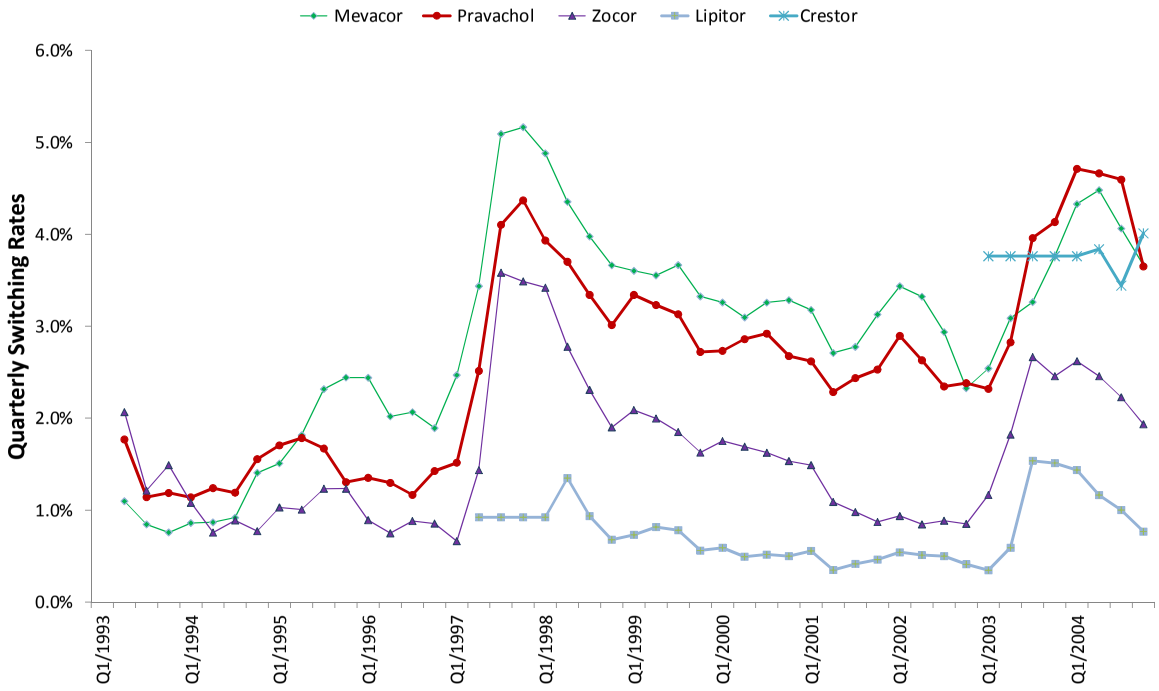


Figure 4: Quarterly Discontinuing Rate for Leading Statins

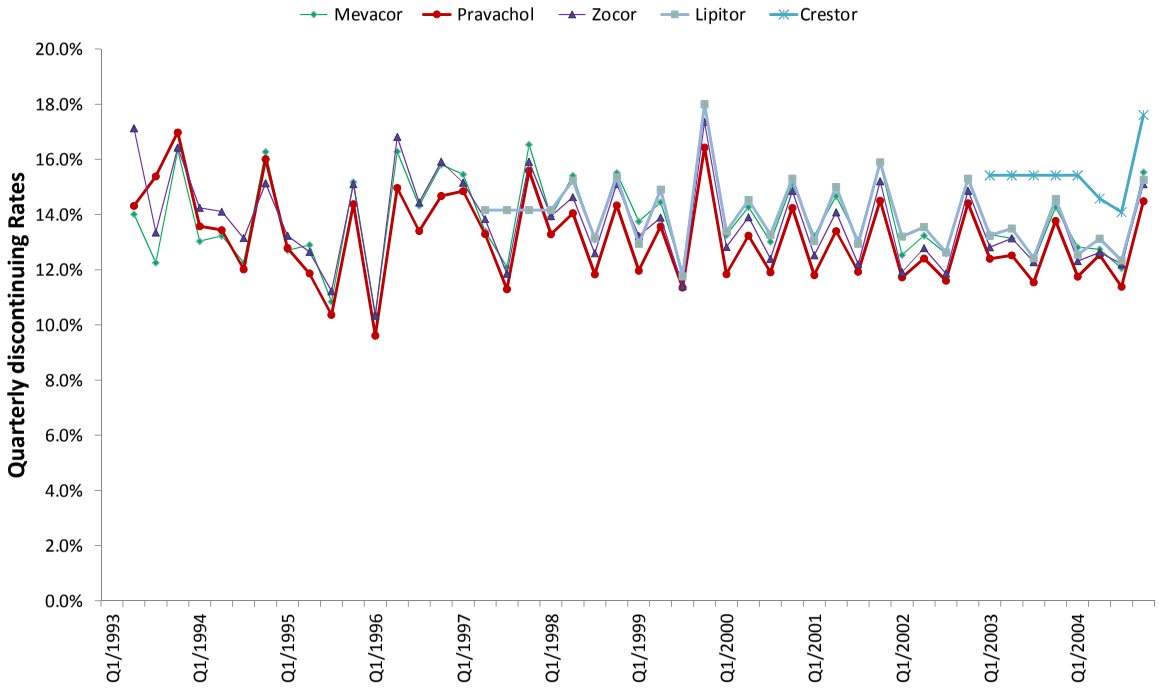


Figure 5: Quarterly Flow of General Publicity

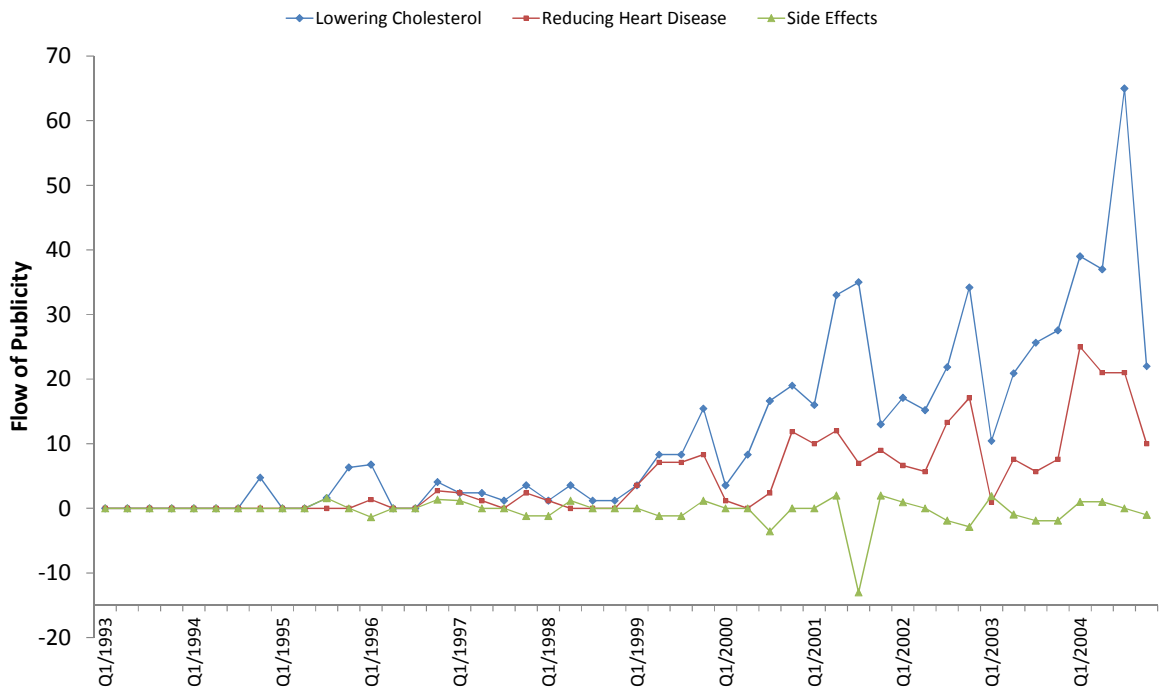


Figure 6: Decision Process of Potential Patient

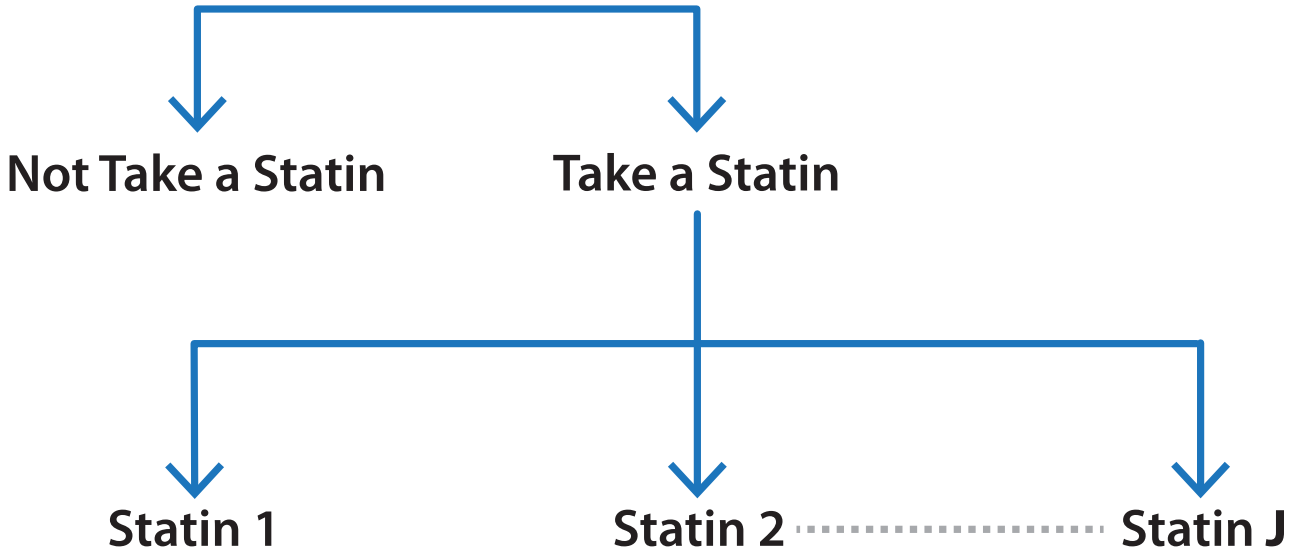


Figure 7: Decision Process of Existing Patient

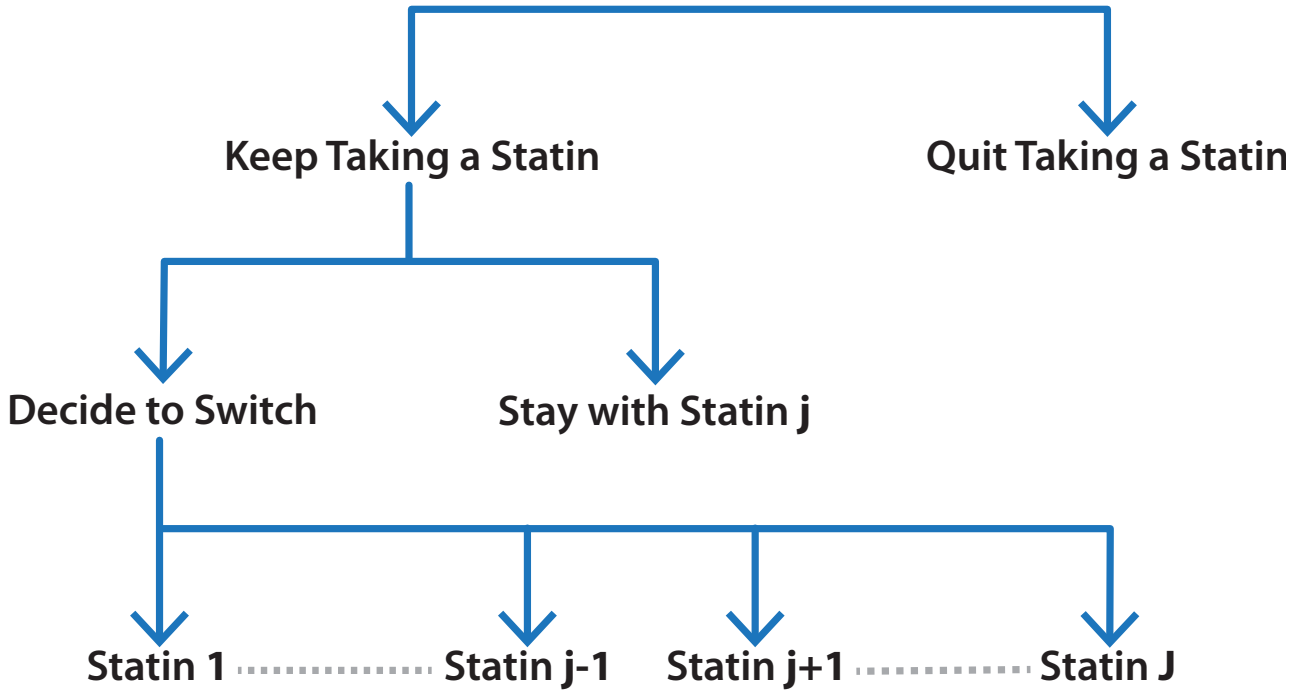
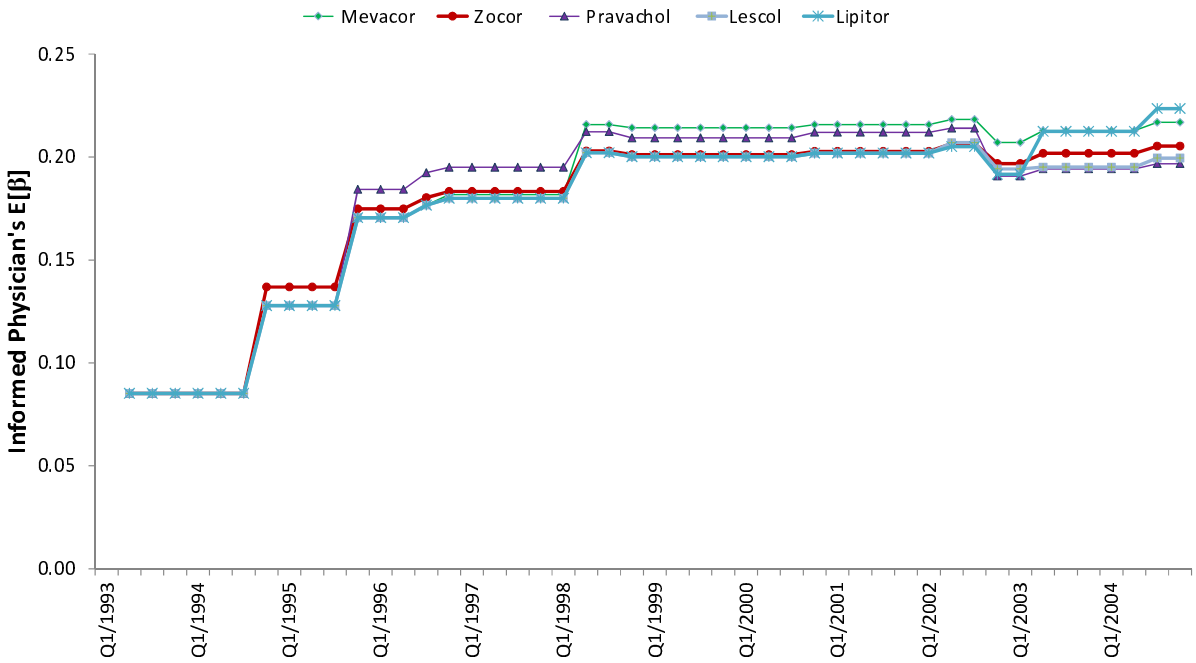


Figure 8: The Posterior Beliefs of Well-Informed Physicians†



†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

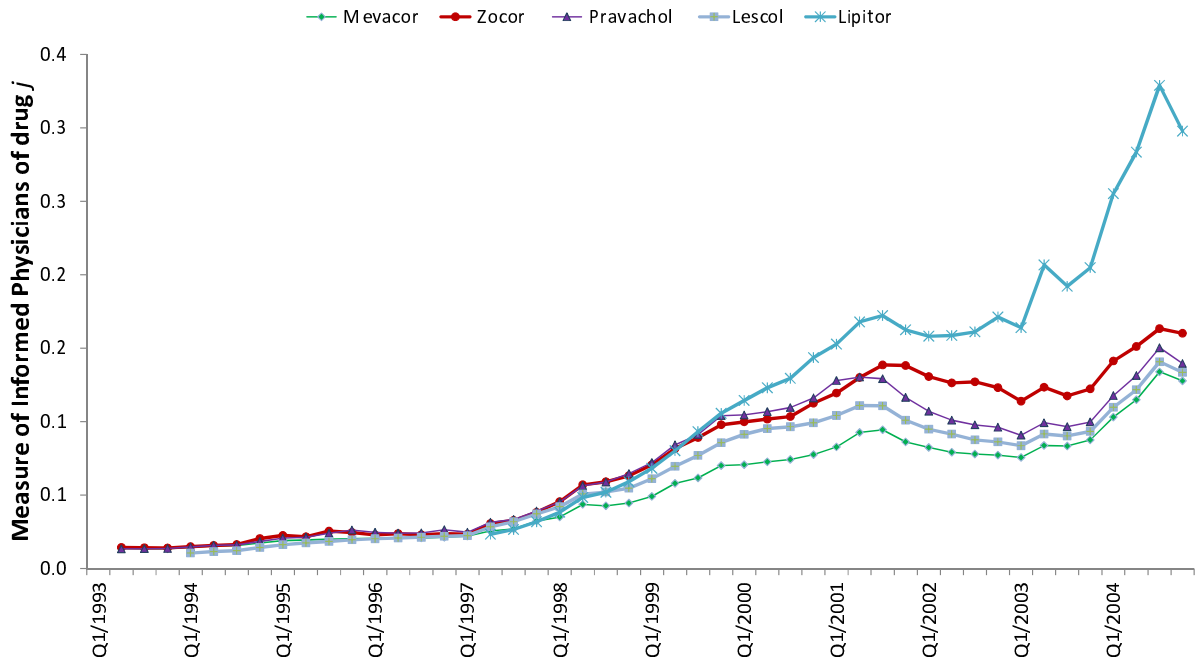


Figure 10: Fit: Actual and Simulated Prescription Volume

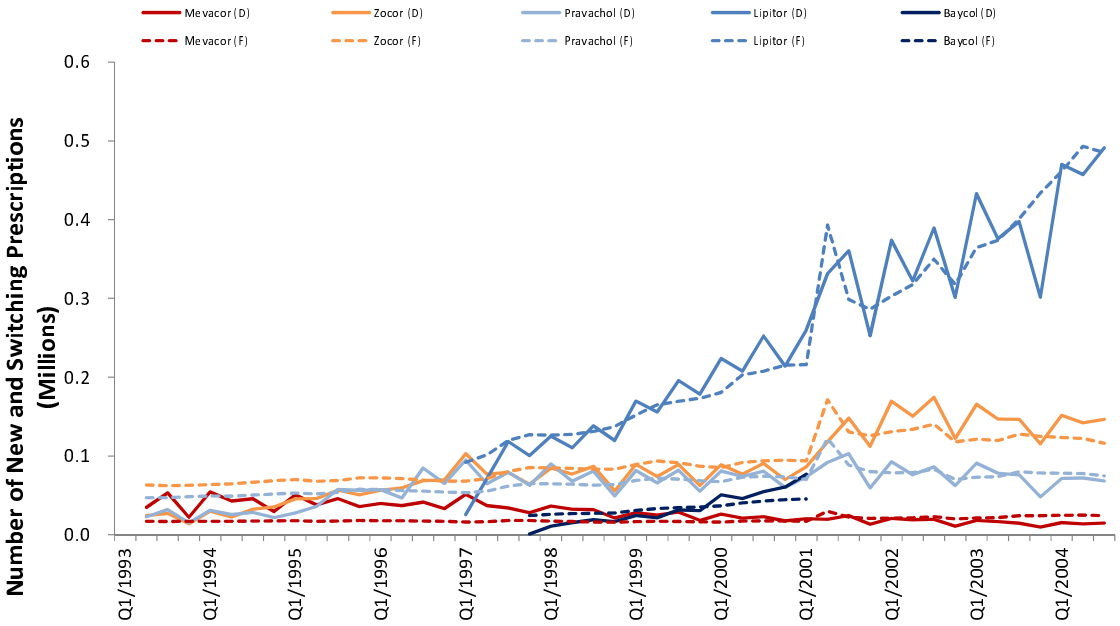


Figure 11: Counterfactual Experiment 1 (Removing Lipitor's Landmark Trials)

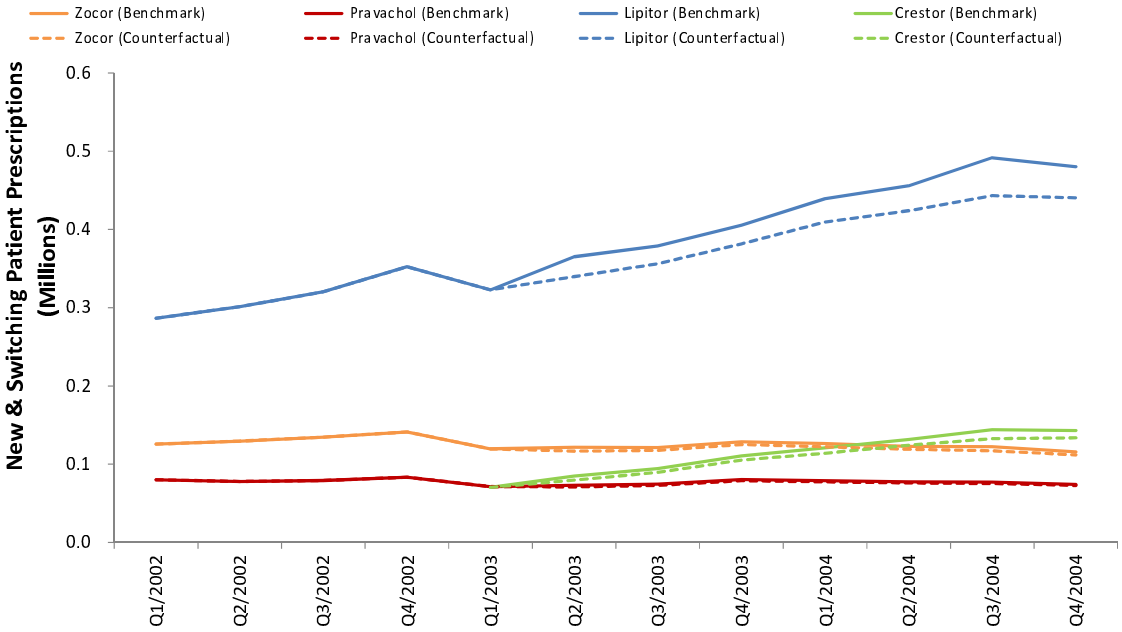


Figure 12: Counterfactual Experiment 2 (Removing Correlated Learning)

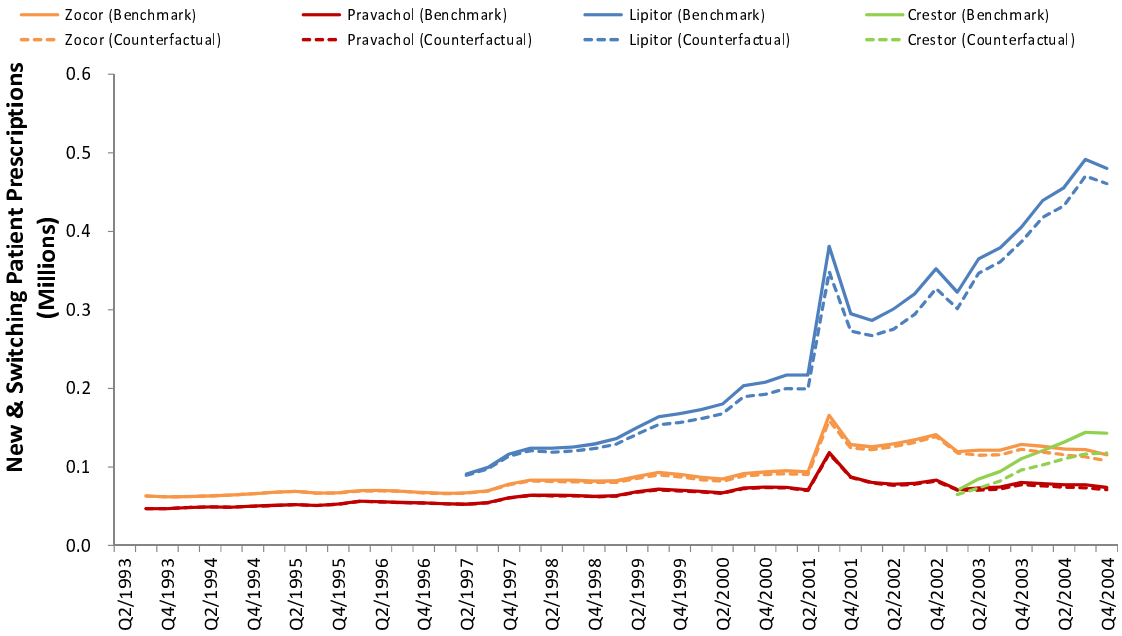


Figure 13: The Most Updated Physician's E[q^h] over Time (Counterfactual Experiment 2)

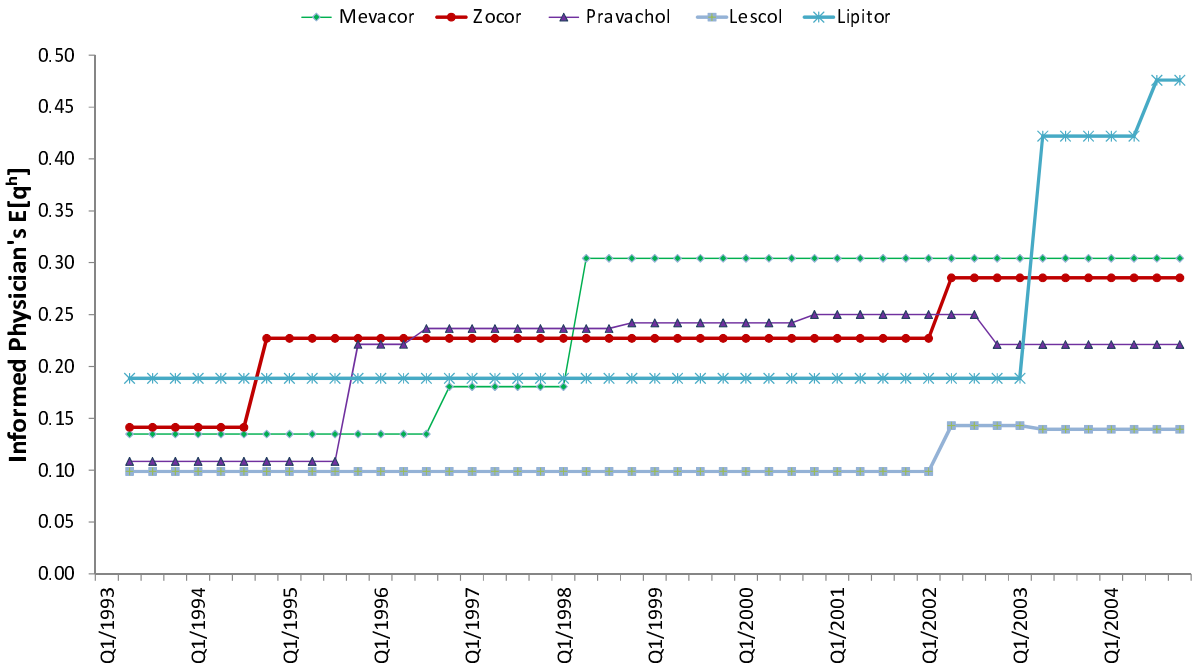
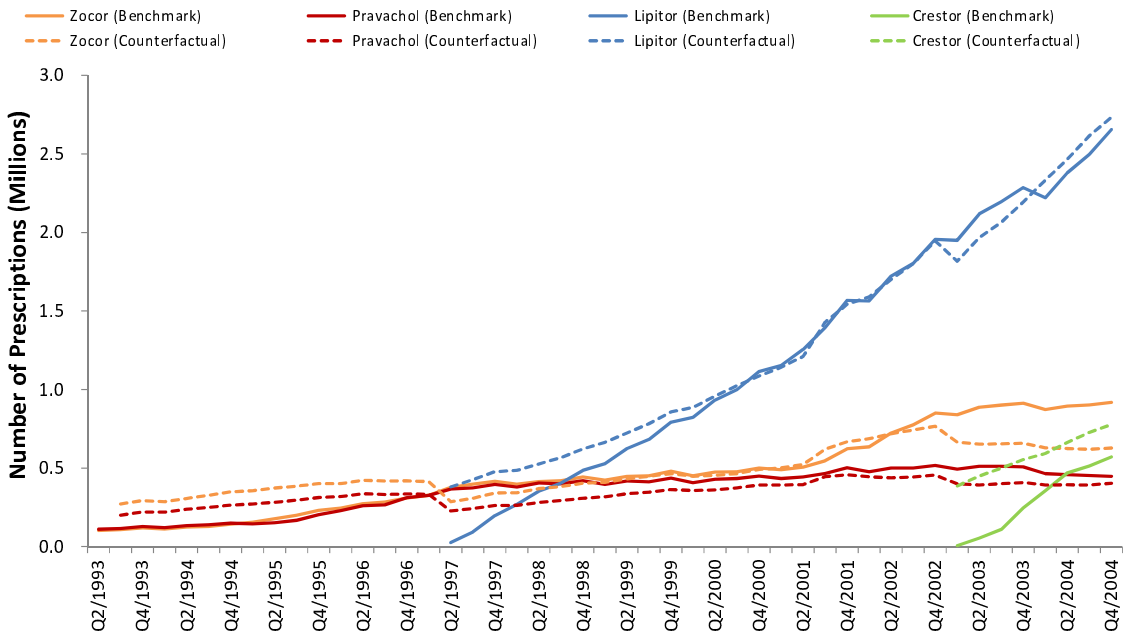


Figure 14: Counterfactual Experiment 3 (Removing Switching Cost)†



†(B) denotes Benchmark demand from the full model; (C) denotes Counterfactual demand