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The Effects of Publicity on Demand: The Case of Anti-cholesterol Drugs*

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Abstract

Over the past ten years there has been increased recognition of the importance of publicity as a means of generating product awareness. Despite this, previous research has seldom investigated the impact of publicity on demand. We contribute to the literature by (i) proposing a new method for the interpretation of publicity data, one that maps the information in news articles (or broadcasts) to a multi-dimensional attribute space; (ii) investigating how different types of publicity affect demand; and (iii) investigating how different types of publicity interact with firms' own marketing communication efforts. We study these issues for statins. We find that publicity plays an important role both for expanding the market for statins and for determining which statins patients/physicians choose. We also find evidence that publicity can serve as either a substitute or a complement for traditional marketing channels depending on the complexity of the information type. We argue that the interaction results are driven by the relative strengths of the corroborative and rational inattention functions in publicity. These results suggest that managers should be aware of the interactions between publicity and traditional marketing channels in order to better determine how to allocate their marketing expenditures.

JEL Classification: D12; I11; L65; M30; M31; M32

Keywords: publicity; informative detailing and advertising; information complements and substitutes; corroborative evidence; rational inattention; demand; prescription drugs

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1 Introduction

Over the past ten years, there has been a recognition of the diminished role of traditional marketing channels and the increased importance of publicity as a means of generating awareness about a product (e.g., Ries and Ries, 2009). Publicity, which is generally conveyed to potential customers by a third party in the form of news, is cheaper than traditional marketing channels and can reach a wide range of consumers. Despite the increased interest in publicity, research in marketing has seldom investigated the impact of publicity on product demand or the interaction between publicity and traditional marketing channels. This paper seeks to address this deficiency by (i) proposing a new method for the interpretation of publicity data, one that maps the content of each news article (or broadcast) to a multi-dimensional attribute space; (ii) investigating how different types of publicity affect consumer demand; and (iii) investigating how different types of publicity interact with firms' own marketing communication efforts. We study these issues for a particular class of prescription drugs in Canada – statins, which are the most commonly prescribed group of anti-cholesterol drugs.

There are at least three reasons for our choice of statins. First, publicity features prominently in this class of drugs and much of it is related to post-marketing clinical studies. Therefore, it is plausible that both patients and physicians obtain at least some of their information regarding various drugs' efficacy and side-effects through the media that they can access, e.g., newspapers, TV news, internet, etc.

Second, statin manufacturers (and pharmaceutical manufacturers in general) invest considerable effort in detailing – personal selling to physicians – that also focuses on efficacy, side-effects and the like. From the manufacturers' perspective, understanding the ways that publicity interacts with their own detailing efforts – either enhancing or diminishing detailing's impact – can help them to design more effective marketing strategies.

Third, information relevant to doctors and patients in evaluating a particular statin's efficacy is

heterogeneous in terms of its complexity. In particular, the information necessary to evaluate a statin's ability to lower cholesterol or its side-effects is relatively simple and straightforward: e.g., Lipitor lowers cholesterol by 30 percent, on average. The information necessary to evaluate a statin's ability to reduce heart disease risks, on the other hand, is relatively complicated. Here, information on the nature of the clinical trials is crucial: e.g., the result of each endpoint measure (on average, there are 16 measures reported in each clinical study, and the maximum number is 34), characteristics of the patient pool, the number of years for the study, the group undertaking the study, etc. This heterogeneity provides a unique opportunity to investigate whether "complexity" of information may impact the interactions between different sources of information.

Our approach to quantifying the impact of publicity differs in important ways from earlier methods. Previous research has looked at news stories as uni-dimensional, classifying them as either positive, negative, or neutral (Berger et al., 2010; Chintagunta et al., 2009; Goldenberg et al., 2007; Huang and Chen, 2006; Kalra et al., 2011). We argue that such a uni-dimensional classification can be misleading. For example, a news article might report that an anti-cholesterol drug lowers cholesterol more effectively than do its competitors, but that some patients experience serious side-effects. This article could be coded as positive, negative, or neutral in a uni-dimensional classification depending on the reader's perspective. To reduce ambiguity of context, we code the information of an article into a multi-dimensional attribute space. We consider three attributes for our drugs: (i) their ability to lower cholesterol (short-term efficacy); (ii) their ability to reduce heart disease risks (long-term efficacy); (iii) their side-effects. Continuing with the above example, our algorithm classifies this article as positive in the dimension of lowering cholesterol and negative in the dimension of side-effects. To the best of our knowledge, our research will be the first to use publicity data that is coded in such a precise fashion. This multi-dimensional coding scheme can reduce measurement errors made in uni-dimensional coding schemes used in the previous studies. More importantly, it allows us to separate simple vs. complex

types of information such that we can investigate how different information sources may complement one another or substitute for one another.

We model the statin choice problem as a two-stage process. In the first stage, patients and physicians jointly decide whether or not to use a statin drug to lower cholesterol. In the second stage, physicians, taking patients' utility into account, make prescribing decisions for specific statin drugs. In the latter case, the decision is based on the physician's assessment of the efficacy of each drug. Because the physician is uncertain about any given drug's efficacy, this assessment involves the physician utilizing different information sources to form a "consensus" distribution about each drug's efficacy (see Winkler, 1981). We assume that publicity plays two roles in the formation of this distribution. First, it can act as corroborative evidence for information provided via detailing. Second, by representing an additional source of information itself, it can induce rational inattention on the physician's part when detailers come to call (in the sense of Reis, 2006a,b). The overall impact of publicity on demand represents a balance of these two forces. For complex characteristics – a statin's efficacy at reducing heart disease risk – we expect that the corroborative role of publicity should dominate, because any information sources are necessarily noisy. In this case, we predict that publicity and detailing will be demand complements. For simple characteristics – a statin's efficacy at lowering cholesterol or its side-effects – we expect that the rational inattention effect should dominate, because all information sources are relatively precise. In this case, publicity and detailing will be demand substitutes.

Our analysis reveals the following: (i) Publicity that mentions particular statins by either their brand-names or their chemical names plays an important role in explaining brand choice and the rapid growth of the statin market; (ii) publicity that mentions statins in general without referring to any particular brand also helps expand the statin market; (iii) ignoring publicity or using a uni-dimensional coding scheme leads to biases in parameter estimates and misleading detailing elasticities; and, most importantly, our multi-dimensional coding scheme allows us to show evidence that (iv) publicity about

a drug's ability to lower cholesterol and its side-effects have negative interaction effects with detailing, and (v) publicity about a drug's ability to reduce heart disease risks has positive interaction effects with detailing. These latter two results are consistent with our hypotheses that detailing and publicity are information substitutes (complements) when information is simple (complex).

Our results identify a potential new information benefit of traditional marketing channels: to supplement or reinforce messages that come from publicity. At the same time, our findings suggest that ignoring publicity may lead researchers (both academic and market-based) to overstate the effectiveness of traditional channels and therefore encourage wasteful marketing expenditures. When one recognizes that prescription drugs are not the only class of products in which both publicity generated by third party reviews/assessments and manufacturer's personal selling effort feature prominently, our results take on added importance. Similar issues will be relevant in the cases of medical devices, automobiles, hunting and fishing equipments, stereo equipments, appliances and other durables. As a consequence, what we learn here has broader applicability to a whole host of marketing problems.

The rest of this paper is organized as follows. Section 2 reviews the previous literature. Section 3 provides background of the statin market in Canada. In section 4, we discuss our data sources, and explain the method we use to collect and code the publicity data. We also discuss evidence that supports the notion that information about a drug's ability to reduce heart disease risks is significantly more complex than that about its ability to lower cholesterol and side-effects. Section 5 discusses the econometric model, and develops the hypotheses regarding the interactions between publicity and detailing. Section 6 presents the estimation results and discusses their implications. Section 7 concludes.

2 Literature Review

To our knowledge, this paper is the first one to study the interactions between firms' informative marketing efforts and publicity. Most of the previous empirical studies on publicity focus on the impact of critic- and product-reviews on demand (Basuroy et al., 2003; Berger et al., 2010; Chevalier and

Mayzlin, 2006; Godes and Mayzlin, 2004; Huang and Chen, 2006; Kalra et al., 2011). The results here are mixed. In a study of critical film reviews, Basuroy et al. (2003) find that positive reviews increase box office revenues while negative reviews decrease revenues. Huang and Chen (2006) provide similar findings on the impact of customer reviews and sales volume numbers for on-line products. Berger et al. (2010) examine the impact of reviews on the sales of books and find that both positive and negative reviews can increase book sales. Another closely related work is by Stammerjohan et al. (2005), who study the interaction between informative advertising and positive or negative news stories. However, unlike our research, their study is undertaken in a laboratory setting and uses experimental data rather than actual field data.

Our research is also related to the pharmaceutical marketing literature that studies the roles of detailing, journal advertising, and scientific evidence on demand (Azoulay, 2002; Berndt et al., 1996; Chan et al., 2013; Ching and Ishihara, 2010; Cockburn and Anis, 2001; Fischer and Albers, 2010; Leeflang and Wieringa, 2010; Narayanan and Manchanda, 2009; Neslin et al., 2009). This literature has paid very little attention to the role of media coverage in pharmaceutical product choices.[1] To our knowledge, Chintagunta et al. (2009) and Kalra et al. (2011) are the only studies that incorporate data on news coverage when estimating the demand for pharmaceutical products. In contrast to our work, the focus of Chintagunta et al. (2009) is on disentangling learning via one's own experiences from learning via others' experiences. Publicity data is simply used as a control variable in the analysis and is only classified as non-negative or negative based on the title of each article. Chintagunta et al. (2009) find that news articles have a positive influence on prescription choices regardless of the tone of the article's title. They conjecture that this counter-intuitive result could be due to problems in their data-coding design that lead to measurement errors in their publicity variable. Our multi-dimensional coding method could potentially address this shortcoming. Kalra et al. (2011) model the impact of positive

[1] A recent paper by Dunn (2012) estimates the demand for anti-cholesterol drugs in the U.S. and propose a quality-adjusted price index, but he does not use any data on detailing, advertising and publicity.

and negative media news regarding the safety of type II diabetes drugs on physicians' beliefs regarding quality. However, they do not investigate the impact of media coverage regarding the effectiveness of type II diabetes drugs. For this reason, they use a uni-dimensional coding scheme for publicity. In their structural model of physician learning, they assume that every news article from their sources is read by each physician, but rule out the possibility that news articles can influence patients' preferences even though they may play a role in the demand for prescription drugs. In contrast, by using a multi-dimensional coding method, our study investigates the impact of media coverage about both the effectiveness and safety of a drug. Moreover, our reduced form model allows both patients and physicians to be influenced by news articles, without the need to assume that every article is read by every physician/patient. We can also shed light on how publicity influences market expansion and brand choice differently.

Our coding method is closely related to Chandy et al. (2001), who map the advertising content (for an unspecified medical service industry) into a multi-dimensional attribute space.[2] However, their focus is very different from ours: they code ad cues that are related to consumer psychology, while our study is interested in coding the informational content of news articles. We view these two approaches (one for ads, and the other for news) as complementary. Ads are often designed taking consumer psychology into account and do not necessarily contain information that is new from the perspective of consumers. In contrast, news articles and TV news items are usually informative and report using straightforward language. Moreover, creating a data set for publicity is a much more challenging task because articles are seldom the same, but a TV ad is typically shown repeatedly for an extended period. As a result, there are generally many more publicity articles to code compared with ads for any given period of time.[3]

[2]Recently, Bertrand et al. (2010) have extended this approach to study the impact of ad content on the demand for bank loans in a field experiment.

[3]After our research was well underway, we become aware of three other on-going, concurrent, and independent research projects (Anderson et al., 2013; Basuroy et al., 2011; Liaukonyte, 2011), which study the informational content of ads. None of them investigate the impact of news articles or the prescription drug market.

3 Background of the Statin Market

There are two main types of cholesterol: LDL ("bad" cholesterol) and HDL ("good" cholesterol). High amounts of LDL will deposit cholesterol on the artery walls forming plaque. The buildup of plaque narrows the lumen in the arteries and may eventually block blood flow, leading to a possible heart attack or stroke. On the other hand, HDL takes excess cholesterol away and carries it back to the liver to be excreted. It can also remove some of the cholesterol already attached to the artery walls. Statins are the most commonly used group of anti-cholesterol drugs. They lower the level of LDL by blocking the enzyme that synthesizes cholesterol in the liver. Usually, when people simply use the word *cholesterol*, they refer to LDL. We will follow this convention in this paper. Table 1 contains brief background information about the seven statins present in our sample period.

The statin class is notable for a series of high profile landmark clinical studies, the publication of which attracted considerable media attention. Typically, each landmark study focused on one statin and tested its ability to reduce heart disease risks. These studies tend to be expensive to conduct due to the fact that heart attacks and strokes are relatively rare events. As a result, each study needs to follow a large number of patients for four to five years to document any impacts. The design of these landmark studies is also significantly more complicated compared with studies that focus on learning a statin's cholesterol lowering ability. This difference in complexity across clinical trials will be exploited subsequently in our hypothesis development.

One non-landmark clinical trial that received an exceptional amount of publicity is CURVES. The results of this head-to-head comparison study were announced by Pfizer in 1996. CURVES shows that Liptior is more effective in lowering cholesterol than the four older statins: Mevacor, Zocor, Pravachol, and Lescol. Although this study did not establish any direct evidence on Lipitor's ability to reduce heart disease risks, Lipitor's market expanded rapidly after its introduction in 1997.[4] In 2002, Lipitor achieved

[4]Lipitor did not have a landmark clinical trial providing evidence on heart disease risks until 2001. This is noteworthy since the fact that a drug can lower cholesterol effectively does not necessarily mean that it can reduce heart disease risks.

estimated sales of US$7.4 billion worldwide and became the best-selling product in the prescription drug market.

In 2003, AstraZeneca released Crestor, claiming that it lowers cholesterol far more effectively than other statins, including Lipitor. An AstraZeneca sponsored head-to-head clinical study called STELLAR provided clinical evidence that Crestor not only lowers LDL levels but also increases HDL levels significantly more than does Lipitor. Although safety issues for Crestor have been raised, the FDA concluded that Crestor is not riskier than other statins on the market and it has experienced strong growth.

It is worth noting that the most common side-effect of taking statin is mild muscle pain (around 10-15% of people experience it). For most statins, it is extremely rare that it would lead to permanent muscle damage. In 1998, Bayer introduced Baycol with a clinical study showing that it is more effective than Mevacor, Zocor and Pravachol in lowering cholesterol (Davignon et al., 1998). However, evidence shows that Baycol's risk of having permanent muscle damage is 10 times that of the other statins' (Graham et al., 2004). In addition, 52 deaths were reported in the U.S. for patients using Baycol. As a result, in 2001 Bayer "voluntarily" withdrew Baycol from the worldwide market.

4 Data

Our analysis integrates three different data sources: (i) publicity data in the Factiva database covering statins, (ii) landmark clinical trial data for statins, and (iii) monthly product-level prescription volume, detailing and journal advertising data for the Canadian statin market from IMS Canada. The first statin was introduced in 1988; however, as we will discuss in further detail below, our sales data only begin in 1993. In order to avoid any initial conditions problems that this fact may cause when estimating statin demand, we have collected marketing and publicity data for years prior to 1993 (more details can be found in subsection 5.3 below).

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 cholesterol (Park, 2008).

4.1 Publicity Data

To investigate the impact of media coverage on patients' and physicians' demand, we extracted 41,002 news articles from Factiva that refer to statins over the period of 1986 to 2004. Factiva is a division of Dow Jones & Company that provides access to more than 25,000 authoritative sources, including newspapers, journals, magazines, news and radio transcripts, etc. We searched for articles that contain the word "statin" or words related to statin, such as the chemical names or brand names. We restricted the search to articles from *Canadian Accessible Sources*; that is, to sources that Canadian physicians and patients may likely access. These include online sources, Canadian television news programs, Canadian newspapers, and Canadian magazines, as well as U.S. television news programs from the four major television networks (ABC, NBC, CBS and FOX) and CNN, the eight biggest U.S. newspapers with circulation of more than 500,000 daily and the 25 top selling U.S. magazines. We assume that the public might not have direct access to the press releases from news agencies and so we omit articles from news agencies such as Agence France-Presse, The Associated Press, Reuters, etc. In the end, we include 2,754 articles in the analysis. Table 2 shows the number of sources and articles by year. In general, both the number of sources and the number of articles increase over time. One concern is that some of the sources may have existed earlier, but that Factiva only added them to their database later. Given that most of our sources are major newspapers, magazines and TV news networks, this concern is likely valid. We therefore decided to weight the articles differently depending on which year they were published, using the number of sources in 2004 as the base. More precisely, the weight assigned to an article in year Y is $\frac{\#\text{ sources in 2004}}{\#\text{ sources in year Y}}$.[5]

For each article, we extracted its headline, source, content and publication date. After a number of trials and errors, we decided to map the information of each article into two multi-dimensional variables: (a) *general publicity* ($publicity_t^s$) – the article has sentences that discuss statins in general without

[5]We also considered using circulation data to weigh articles. Unfortunately, the historical circulation data proved difficult to obtain.

referring to any particular statin by brand or chemical name; (b) *drug specific publicity* ($publicity_{jt}$) – the article 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 to both variables – it can provide information about statins in general at the beginning and then later mention which particular statin is the most effective. Our intuition is that general publicity is more likely to affect the overall demand for statins, while drug specific publicity should mainly affect which particular statin to use.

For every article in each of the publicity groups, we code the article based on its information content. Because an article may well contain multiple pieces of information, we adopt a multi-dimensional coding procedure.[6] This procedure categorizes each article on three dimensions: lowering cholesterol (lc), reducing heart disease risks (rh), and side-effects (se). The procedure categorizes drug specific publicity further based on whether it provides comparisons across drugs – *comparison* (c) – or not – *non-comparison* (nc). In the end, general publicity is a 3-dimensional variable – (rh_t^s, lc_t^s, se_t^s) with the superscript s indicating the entire statin class – while drug specific publicity is a 6-dimensional variable – (rh_c_{jt}, lc_c_{jt}, se_c_{jt}, rh_nc_{jt}, lc_nc_{jt}, se_nc_{jt}) with $\_c$ denoting comparison, $\_nc$ denoting non-comparison, j indexing drug, and t indexing time.

For each dimension of non-comparison publicity (including both *drug specific* and *general* publicity), we use a two-step Likert scale (+1, -1) to assess the article's tone. We assign "+1" ("-1") if the article contains sentences which favor (do not favor) the focal drug. For example, "Lipitor reduces cholesterol quickly" is classified as "+1" in the lc dimension, "Crestor can cause fatal damage to a patient's kidney" is considered to be "-1" in the se dimension, and "Pravachol was well-tolerated" is considered to be "+1" in the se dimension.

For each dimension of drug specific comparison publicity, we score each comparison article similarly. In particular, we assign "+1" to a drug when the article contains sentences which favor that drug most,

[6]For example, an article may state that "Crestor is very effective in lowering cholesterol, but it potentially has serious side-effects." Such an article is difficult to code using uni-dimensional coding scheme.

and "-1" to the drugs to which it is compared. For instance, "Crestor reduces cholesterol faster than Lipitor and Pravachol," is coded as "+1" for Crestor and coded as "-1" for Lipitor and Pravachol in the *lc* dimension.

The coding schemes described above allow us to code most of the articles relatively straightforwardly. We had two undergraduate research assistants (RAs) code each article independently. The agreement rates were 77.6% and 80.62% for non-comparison and comparison articles, respectively. For those articles which were coded inconsistently by the RAs (i.e., the coding outcomes are not the same for these two RAs), we reviewed the disputed articles to determine the final coding outcomes.

In our empirical analysis, the length of a period is a month. Since there is usually more than one news story published/broadcasted in each month, we must aggregate the coded outcomes for each month's publicity to obtain a monthly observation. We construct the monthly publicity variable as follows: Let $(publicity^s_{t,l}, publicity_{jt,l})$ denote the publicity variables associated with article l that is published in month t. Also, let L_t be the total number of articles in month t. Then the values of $(publicity^s_t, publicity_{jt})$ are obtained by summing $(publicity^s_{t,l}, publicity_{jt,l})$ across all articles appearing in month t: $publicity^s_t = \sum_{l=1}^{L_t} publicity^s_{t,l}$, for instance.

Finally, there was a small subset of *se*-dimension articles related to Crestor that proved problematic for our coding scheme. The problem arose because of a report from a watchdog group urging the FDA to remove Crestor from the market based on several incidences of extreme side-effects outcomes.[7] Articles reporting that Crestor might have more serious side-effects also typically stated that the manufacturer is very confident that the drug is safe. The conflicting messages about side-effects made this subset of articles hard to code. In the end, we classified them as a *debatable* articles, coding them as neither positive nor negative. The stock variable (STK_debate_t), is simply the number of debatable articles that appeared in that month. We include STK_debate_t in the utility of choosing Crestor and let the data tell us whether it has positive or negative net impacts on sales.

[7] Chen and Tan (2010) focus on this particular negative news and investigate how firms' detailing efforts react to it.

Table 3 presents a descriptive summary of our drug specific publicity variables. In general, non-comparison articles are much more common than comparison articles, *lc*-type articles are more common than *rh*-type articles, while *se*-type articles are the least common. Most rh_c articles (94%) compare Lipitor and Pravachol. In fact, their publication dates coincide with the release dates of two landmark clinical trials: REVERSAL (March 2004) and PROVE-IT (April 2004), which compare Lipitor and Pravachol in terms of their ability to reduce heart disease risks. A closer examination of the rh_c articles confirms that they indeed discuss these two landmark clinical trials.

We find that comparison articles tend to be more prevalent in the later part of the sample period, while non-comparison articles appear much more evenly over time. Except for lc_c and rh_c, which appear to be similar, the variations in publication timing are quite different across publicity variables. Such variations indicate that we should be able to separately identify the impacts of these variables. While there are some bad news articles about the side-effects of statins, especially in 2001 when Baycol was removed from market, most news articles report that statins are effective in lowering cholesterol and reducing heart disease risks.[8]

4.2 Landmark Clinical Trials

Azoulay (2002), Ching and Ishihara (2010), Cockburn and Anis (2001), and Sood et al. (2014) find evidence that clinical trials have a significant impact on physicians' prescribing decisions. Hence, to control for the impact of clinical evidence, we collect landmark clinical trial data from the U.S. National Library of Medicine (www.medscape.com). Table 4 lists the landmark clinical trials we include. We define landmark trials for statins as follows: a clinical trial is a landmark trial if its main clinical endpoint (the target outcome of the trial) is the drug's efficacy in reducing heart disease risks.[9] This is in contrast to other clinical studies whose aim is to determine a statin's ability to lower cholesterol.

[8]Data plots of various types of publicity variables are available upon request.

[9]We should note that the definition of landmark trial is not universally agreed upon for statins although most medical sources will give a similar set of landmark clinical trials. Our definition is relatively broad. Some sources will further classify our list of landmark trials to: (i) very influential trials; (ii) enrichment trials.

Landmark clinical trials differ from non-landmark clinical trials in a number of dimensions. On average, a landmark clinical trial measures 16 endpoints (see the 7th column of Table 4) while a typical non-landmark clinical trial usually only measures 4 endpoints: levels of LDL, HDL, triglycerides and total cholesterol. The selection of the subject pool for a landmark trial is more complex, with the selection criteria including subjects' i) diabetes problems, ii) previous heart disease histories, and iii) current cholesterol levels. Non-landmark trials usually select patients only based on their cholesterol levels. Also, since the absolute risk of having a heart attack is low in any given month, even for high risk subjects, landmark clinical trials typically include thousands of subjects and most follow them for multiple years to prove the drug's long-term efficacy.[10] Non-landmark trials follow many fewer people for a much shorter time period. Because of the size, scope and length of landmark trials, public health agencies and medical researchers consider these studies to be much more ambitious and important than the non-landmark trials.[11] This also explains why information about a drug's ability to reduce heart disease risks is much more complex than that about its ability to lower cholesterol and side-effects.

We create a stock variable to capture cumulative knowledge about statins. Once an article is published, sales representatives can always refer to it during their detailing activities. We therefore assume that the stock variable for landmark trials does not depreciate, i.e., we set the carryover rate of the landmark trials to be 1. Moreover, the sample size of a clinical trial is often interpreted as a way to measure how significant the study is. We therefore weigh each study by its number of participants when creating the stock variable, $STK\_clinical_{jt}$. More precisely,

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

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

[10] MIRACL is an exception. It followed patients for only 16 weeks because it tried to to show that Lipitor can reduce early recurrent ischemic events if patients takes Lipitor within 24-96 hours after suffering an acute coronary syndrome.

[11] Accounting for these trials also allows patients/physicians to eliminate the concern that statins might not be able to reduce heart disease risks – it is possible that a statin has negative long-term side-effects which could counter their benefits of reducing cholesterol.

4.3 Prescription Volume and Promotional Mix Data

We obtained product-level data from the market research firm IMS Canada. It consists of monthly observations on prescription volumes, detailing costs and journal advertising pages for each statin across Canada. The prescription volumes data are available from March 1993 ($t = 1$) to December 2004 ($t = 130$). Information on detailing and journal advertising – our promotional mix variables – goes back to 1988. The statin market is defined as the national market for month t. An observation is defined as a molecule-month combination. Summary statistics are displayed in Table 5.

In Figure 1, we plot the monthly prescription volumes for statins in Canada. The prescription volume for Lipitor reached almost one million by 2001 while the earlier arrivals, Zocor and Pravachol, had 300,000 and 150,000 monthly prescriptions, respectively.

Previous research has documented that marketing activities have an influence on the prescription choices of physicians. To control for the impact of detailing and journal advertising, we incorporate information on detailing expenditures and journal advertising pages for each drug. Figures 2 and 3 graph the monthly detailing and journal advertising, respectively, for the top four statins in terms of average market shares.[12] Note that: (i) the market entries of Lipitor (March 1997) and Crestor (February 2003) coincide with large detailing and journal advertising efforts; (ii) Mevacor (April 1997), Pravachol (July 2000) and Zocor (January 2003) stopped detailing and journal advertising when their generic counterparts were introduced in the market.

DTCA in Canada has been subject to much stricter regulation than in the United States and so, in general, there has been very little DTCA spending (Mintzes et al., 2009). We obtained Canadian DTCA data for statins from A.C. Nielsen. The data show that spending in this category is basically zero for all firms throughout our sample period until 2003. In 2003 and 2004, we observe some DTCA spending but the amount is only one thousandth of the U.S. spending. Therefore, we omit DTCA in

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

Canada from our reported regression results but we can confirm that results are qualitatively unchanged even if it is included.[13] These results are available upon request. Unfortunately, this also implies that we will not be able to study the interactions between DTCA and publicity, which is a limitation of this research.

For most product categories, price is an important factor that affects consumers' purchase decisions. However, price regulation for prescription drugs in Canada means that prices tend to change only infrequently.[14] For statins, we find that prices have hardly changed at all over the sample period. For this reason, we do not include prices in our demand system. This (essentially) fixed price regime also should alleviate concerns regarding the endogeneity of prices (the data suggest that prices are hardly correlated with unobserved demand shocks that vary over time). The inclusion of brand-fixed effects in our model should suffice for capturing the impact of prices.

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 use data from the Canadian Heart Health Survey, recorded between 1986 and 1992. The study suggests that 33% of the total Canadian population aged 16 to 65 and 85% of those over 65 have a high-cholesterol (i.e., high LDL) problem. We multiply these numbers by the total Canadian population for each age group in a given month, as defined by Statistics Canada, and use the result as a proxy for the total number of potential patients

[13] For research that study the effects of DTCA on the demand for prescription drugs in the U.S., see, e.g.,Calfee et al. (2002), Iizuka and Jin (2005), Jayawardhana (2013), Liu and Gupta (2011), and Stremersch et al. (2013).

[14] In Canada, prices of patented prescription drugs are strictly regulated. Health Canada introduced a government agency, the Patented Medicine Prices Review Board (PMPRB), through amendments to the Patent Act in 1987. This board regulates the prices of drugs that are still under patent protection by establishing maximum allowed prices (Anis and Wen, 1998; Paris and Docteur, 2006).

for statins. In order to convert the total population with high cholesterol levels into the number of prescriptions, we assume that each patient visits a physician and receives a prescription once per 90 days.[15] Based on this measure of potential market size, we calculate the total statins share in each month. Figure 4 plots the potential market size, and the total demand for statins over time.

5 Econometric Model

5.1 Demand Model

We view the choice problem for statins as a two-stage process in which decisions are jointly made by the patient and the physician. In stage 1, the two decide whether the patient should use a statin drug of any sort; in stage 2 they decide which statin the patient should use, conditional on the stage 1 decision to use statins as a therapy to lower cholesterol. We call the first stage the *market expansion stage* and the second stage the *brand choice stage*. We assume that, in making their decisions, patients and physicians have access to various sources of information: publicity, detailing, journal advertising and clinical trials.

Our modeling framework is a generalization of both the standard nested multinomial logit model and the models of Berndt et al. (1996) and Gupta (1988), the latter two treating the two-stage demand process as two separate reduced form models. We choose this generalized framework because it allows us to treat the joint patient/physician decision in a flexible way. Our specific concern is that, while both the patient and the physician are likely involved in determining the demand for statins, it is possible that the relative importance of these two players is different in the two stages. In the brand choice stage, it seems likely that the physician plays a relatively more important role in determining which statin to use. The physician likely has access to more information than the patient – both detailing and the experiences of other patients – and is likely better able to assess the efficacy of the different

[15] According to Cosh (2010), other than Quebec, the prescription sizes for statins in the rest of Canada are typically 90 days and 180 days. For administrative purposes, the maximum prescription size is one month in Quebec. Since the population in Quebec is roughly a quarter of the entire Canada, we assume that the average prescription size for statins is 90 days in Canada.

statins. Ultimately, the physician is also the one who has the authority to write the prescription (i.e., the physician is the final decision maker in the brand choice). Of course, the patient can still work to influence the prescribing decision but is likely less influential at this stage. In the market expansion stage, it is likely that the patient plays a more significant role in deciding whether or not to use a statin to lower cholesterol. At this choice level, a patient must decide whether to visit a doctor – information from *general* publicity may trigger both this decision and the decision to request a cholesterol level check. Our modeling approach allows for such a possibility and lets us shed light on the relative importance of the physician and the patient in these two stages (this can be done via the difference in the relative importance of publicity versus standard marketing activities in these two stages). Moreover, if general publicity turns out to be unimportant in the market expansion stage, our estimation results will reproduce the nested multinomial logit model.[16] The details of the choice problem and the demand model are given below.

In the first stage – the market expansion stage – we assume that whether a potential patient adopts statins depends on the general publicity that statins have generated and the expected maximum utility in the brand choice stage. The latter is referred to as *the inclusive value* in the discrete-choice literature. It should be noted that the inclusive value term is a "weighted" average of drug specific detailing, journal advertising, publicity and clinical trial evidence, where the weights are determined by the utilities in the second stage. The inclusive value term captures the idea that physicians play a role in determining whether a patient will be on statins. Since the general publicity variables do not mention any statins by name, they should only affect the likelihood that a patient would adopt statins. In particular, we

[16]We should highlight that standard marketing activities (such as detailing and journal advertising) could also play a role in market expansion. These marketing activities help educate/remind doctors of the risks of having high cholesterol levels. This in turn may encourage them to test their patients' cholesterol levels and convince them to take statins. This effect will be captured by the inclusive value term generated from the brand-choice stage.

assume that the relative share of statin adopters and non-adopters can be specified as:[17]

$$ln(\frac{S_{statin,t}}{S_{outside,t}}) = g(STK\_publicity_t^s, Inclusive_t) + \nu_t,$$

where $Inclusive_t = \ln(\sum_j \exp(\tilde{f}_{jt}+\tilde{\xi}_{jt}))$; $STK\_publicity_t^s = \{STK\_lc_t^s, STK\_rh_t^s, STK\_se_t^s\}$ represents the general publicity stock variables; $\tilde{f}_{jt}$ and $\tilde{\xi}_{jt}$ are the normalized mean utility of choosing brand j and its normalized demand shock, respectively, and we will explain them below.

The second stage choice problem – the brand choice stage – determines how the overall statin demand (from the market expansion stage) is allocated across specific statin products. We adopt a discrete-choice model to determine statin choice and so the conditional market shares of each of the statin products. Our approach is similar to Berry (1994), except that we do not have an outside option at this stage. As is typical, the conditional market shares are determined as the aggregate outcome of all of the individual statin choice problems. We assume that, in choosing among statins, the physician's and patient's objectives are perfectly aligned and that the "common" preferences can be represented by an indirect utility function. For ease of reference, we call this function the patient utility function in what follows.

The indirect utility of patient i from choosing statin j at time t is given by:

$$U_{ijt} = f_j(STK\_promo_{jt}, STK\_publicity_{jt}, STK\_clinical_{jt}) + \xi_{jt} + \epsilon_{ijt},$$

where $f_j(\cdot)$ gives the average utility of statin j given the available information at time t. The variable STK_promo_{jt} is a vector of promotional activities that includes both detailing and journal advertising – $STK\_promo_{jt} = \{STK\_detail_{jt}, STK\_journal_{jt}\}$ – and is meant to capture the impact of information from traditional marketing sources on the joint assessment of the efficacy of any particular statin.[18] Similarly, the variable $STK\_publicity_{jt}$ is a vector of publicity variables for statins

[17] This can be derived by assuming: (i) a potential patient i's utility of adopting statins as $V_{i1t} = g(STK\_publicity_t^s, Inclusive_t) + \nu_t + \zeta_{i1t}$, where ζ_{i1t} is i.i.d. extreme value distributed; (ii) the mean utility of choosing the outside good is normalized to zero.

[18] As discussed in the previous section, there is very little variation in prices. Therefore, instead of including prices in our model, we will simply allow for drug-specific fixed effects.

that includes *lc*-, *rh*-, and *se*-type comparison and non-comparison articles – $STK\_publicity_{jt} = \{STK\_lc\_c_{jt}, STK\_rh\_c_{jt}, STK\_se\_c_{jt}, STK\_lc\_nc_{jt}, STK\_rh\_nc_{jt}, STK\_se\_nc_{jt}\}$. It is meant to capture the impact that information from various forms of publicity have on the joint assessment of the efficacy of any particular statin. Finally, $STK\_clinical_{jt}$ is an index that captures the amount of accumulated landmark clinical trial evidence available up to time t and the impact that information from clinical trials has on the joint assessment of efficacy. In all of these instances, we use stock rather than flow variables to capture whatever long-term impacts accumulated information flows may have on demand. The stock variable for detailing is defined as:

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

where δ denotes the carryover rate for detailing. Other stock variables are defined similarly but may have different carryover rates. We assume that ϵ_{ijt} is i.i.d. extreme value distributed.

With these assumptions, conditional market shares are given as:

$$ln(\frac{S_{j,t}}{S_{Mevacor,t}}) = f_j(.) - f_{Mevacor}(.) + \tilde{\xi}_{jt} = \tilde{f}_j(.) + \tilde{\xi}_{jt},$$

where $\tilde{\xi}_{jt} = \xi_{jt} - \xi_{mevacor,t}$. We choose Mevacor as the "reference" drug because it appears in the entire sample period.

5.2 Theoretical Foundation for Understanding the Roles of Publicity and Detailing

At the brand choice stage, there are two questions we want to address with this demand model: i) how do the direct demand impacts of the various forms of publicity compare to those of more traditional marketing activities such as detailing and journal advertising? ii) how, if at all, do the different types of publicity interact with detailing to affect the impact that detailing has on demand? From above, the potential answers to these two questions depend on how different information stocks affect the utility function at this stage. Given that the physician likely plays the key role at the brand choice stage, this issue is best addressed by considering how the physician assesses the efficacy of any given statin.

To economize on space, we provide an intuitive discussion here about how publicity and detailing affect the physician's assessment of drug efficacy. A more formal discussion is presented in Appendix A. Our basic premise is that the physician is uncertain about a statin's efficacy (either at reducing cholesterol or heart disease risk, or causing side-effects) and is confronted with noisy information about efficacy from both publicity and detailing. We assume that the physician combines information signals from the various sources to form a consensus distribution about efficacy. As in Winkler (1981), when forming the consensus distribution, the weight the physician attaches to any given information signal depends on the confidence the physician has in its source (which, in turn, depends on the noisiness of the source).[19]

In contrast to Winkler's framework, which considers only a direct information role for each source, we assume that publicity serves two additional functions. First, publicity serves as corroborative evidence (see Godden, 2010), strengthening the physician's confidence in the information content of detailing signals. Second, to the extent that publicity has already given the physician much of the required information on efficacy, it induces rational inattention (in the sense of Reis, 2006a,b) by physicians when detailers call at the office. Depending on the relative strengths of these two effects, detailing and publicity may serve either as substitutes for one another (when the inattention effect dominates the corroborative effect) or complements (when the corroborative effect dominates the inattention effect).

We assume that the relative strengths of these two effects depends on how complex is the statin characteristic that the physician is assessing. In the case of a statin's ability to lower cholesterol, this is a simple characteristic in the sense that such information can be easily communicated and easily understood by the physician. In this case, the information signals are not very noisy in either source and so the physician can have confidence in the information content of both the publicity and detailing signals. As a result, publicity's role of providing corroborative evidence is likely small in this case.

[19] Other papers in this literature, such as Clemen and Winkler (1999) and Winkler and Clemen (2004), have examined the performance of other types of weighting methods.

Rational inattention, however, potentially looms large. Information from publicity is cheaply available to the physician; he/she can be exposed to publicity when reading newspapers/watching TV news, or hear about the news from patients during their appointments. Since the information signals from publicity are also reasonably precise for simple information, it is plausible that most of the uncertainty can be resolved just based on the publicity source. As a result, the marginal gain to the physician of paying attention to sales reps is likely to be small and so physicians may choose not to incur the attention costs to obtain information signals from detailing. In this case, the detailer may spend many minutes with the physician but actually communicate almost no information. Moreover, this inattention effect is likely to be larger the higher the level of publicity, making detailing less and less effective. This, then, yields our first hypothesis: *publicity and detailing are substitutes when the information is simple to communicate.*

In the case of the statin's ability to reduce heart disease risk, this is a complex assessment for the physician to make. Signals from both publicity and detailing will be much noisier: such information is harder to describe and understand and the space/time/target audience constraints of the news media prevent the publicity source from going into any detail.[20] As a result, there is likely still a significant degree of uncertainty remaining even after physicians have received the information signals from publicity. Consequently, the additional gain to the physician from consulting the detailing source will likely be large, making the rational inattention effect small in this case. At the same time, given the significant degree of remaining uncertainty, publicity may serve an important corroborative effect, bolstering a physician's confidence in the sales reps' claims (e.g., Godden, 2010; Walton and Reed, 2008).[21] This yields our second hypothesis: *in the case of complicated information, with the rational*

[20] When one of the most detailed news articles covered a particularly important landmark clinical trial, 4S (Scandinavian Simvastatin Survival Study Group, 1994), it only mentioned 4 endpoints, while the trial actually reports 34 endpoints (http://www.nytimes.com/1994/11/17/us/study-finds-cholesterol-lowering-drug-may-save-lives.html, accessed on Oct 25, 2014). In Appendix B (Table 9), we list all the endpoints measured in 4S.

[21] The following example from Godden (2010) can illustrate this point: "Several independent witnesses testify to some fact. In this case, not only does each new piece of testimonial evidence count as a primary reason for the conclusion, but it also bolsters the probative value of some other piece of testimony."

inattention effect small, the corroborative evidence effect dominates and so publicity and detailing become complements.

Based on this analysis, we expect both that publicity has different demand impacts from traditional marketing variables and, more importantly, that different types of publicity have different effects (substitute or complement) on the impact of detailing. To capture and assess these possibilities, we adopt the following functional form specification for $f_j(\cdot)$:

$$f_j = \gamma_j + \gamma_{promo} \cdot STK\_promo_{jt} + \gamma_{publicity} \cdot STK\_publicity_{jt} + \gamma_{int} \cdot STK\_detail_{jt} \cdot STK\_publicity_{jt} + \gamma X, \quad (2)$$

where X is a set of additional control variables, including $STK\_clinical_{jt}$ and other variables that we will explain later. Note that the interaction terms between STK_detail_{jt} and different types of publicity stocks allow us to investigate which types of publicity serve as substitutes or complements for detailing.

5.3 Initial Condition Problem and Endogeneity Problem

Before we discuss the estimation results, three estimation issues are worth noting here. First, unlike previous studies that fix the carryover rates of the stock variables (e.g., Azoulay, 2002; Berndt et al., 1996), we estimate them, along with other parameters, using maximum likelihood. Second, our data set for prescription volume starts only in March 1993. Mevacor, Zocor and Pravachol were introduced before that time and so, by March 1993, these three drugs should have accumulated stocks of detailing, journal advertising and publicity. If we do not have detailing and journal advertising data prior to March 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 July 1988, when the first statin (Mevacor) was introduced, and use these data to construct the initial values of detailing and journal advertising stock in March 1993. Similarly, for the publicity variables, we use the pre-sample period data from Jan 1986 to Feb 1993 to construct the

initial values of publicity stocks in March 1993.[22]

The third issue is the potential endogeneity of detailing and journal advertising. The endogeneity problem arises if there are drug-specific demand shocks that are observed by the firms, but not by researchers. In this situation, detailing and journal advertising may be correlated with the error terms in the model. There are three ways to address this concern. First, researchers can look for instruments that are correlated with detailing and journal advertising, but are uncorrelated with the demand shocks, and use instrumental variable techniques to estimate the model (e.g., Berry, 1994). Second, since the endogeneity problem here can be viewed as an omitted variable bias problem, researchers can use the control function approach to address it (e.g., Petrin and Train, 2010). Third, following the argument that this is an omitted variable problem, researchers can collect extra data to explicitly control for the demand shocks. The last approach is seldom used because data on omitted variables are usually not readily available (this is why they are omitted), and collecting them is very time-consuming and costly. Despite such difficulties, the third approach is what we use in this paper.

By including clinical trial results and publicity data in our demand model we alleviate the endogeneity problem related to detailing and journal advertising because trials and publicity are two important sources of demand shocks that were typically omitted in previous research.[23] Incorporating the clinical trial outcomes and the multi-dimensional publicity variables allows us to control for unobserved demand shocks better than did previous studies. Therefore, we believe the endogeneity problem of promotional variables should be less of a concern here. Later we present evidence to support the notion that incorporating publicity variables can help correct this endogeneity problem.

Since our focus is on the effects of publicity, we must also consider the possibility that it is also endogenous. One particular concern might be that publicity arises when demand is elevated and focuses

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

[23]As we discussed in the literature review, there are only handful of studies that incorporate clinical trial outcomes. Moreover, there are only two studies that includes news variables, but in a much simpler way compared with our study.

mostly on how well a given drug is doing in terms of sales. To address this concern, in our analysis, we have dropped all publicity that references sales of the various drugs. We have also controlled for clinical trials, detailing, journal advertising, and drug fixed effects, which we believe are all of the factors that are of first-order importance in determining demand for statins. We cannot think of other obvious demand shocks that might be highly correlated with publicity. Finally, we have included the number of statins on the market in order to control for the introduction of new drugs at the market expansion stage.

In some broad economic sense, one can argue that the landmark clinical trials could also be endogenous because many of them are funded by the industry. However, considering the time required to complete the research, which is usually several years, uncertainty about publication dates and research outcomes, and the fact that we will include drug fixed effects in our model, the remaining unobserved demand shocks should be relatively uncorrelated with the clinical trial results. In particular, it seems unlikely that the release dates and outcomes of clinical trials would be correlated with monthly demand shocks that occur several years after the start of the trial. We now turn to the estimation results.[24]

6 Results

6.1 Market Expansion Stage

Table 6 reports the estimation results for the market expansion stage. We present five specifications. The first specification includes the inclusive value term, the general publicity variables and the number of statins. The inclusive value term is generated from the main specification in the brand choice stage (i.e., specification (2) in Table 7, to be discussed in subsection 6.2). The estimate of the inclusive value term is positive and significant. The number of statins is not significant. This is not surprising because the inclusive value term is already a function of the number of statins but captures its effect in a more

[24]When estimating the model, we re-scale some variables: Detailing stock is divided by 100,000; ad journal stock is divided by 100,000; landmark clinical trial stock variable is divided by 1,000; all publicity stock variables are divided by 100. The re-scaling allows us to get the parameter estimates in the similar order of magnitudes, and facilitate the implementation of maximum likelihood.

structural way. The second specification drops the number of statins and its log-likelihood value and parameter estimates hardly change.

In both the first and the second specifications, none of the general publicity variables are significant. However, the general publicity carryover rate is 0.982 and significant. This fact suggests that the general publicity variables may be jointly significant. In the third specification, we only include the inclusive value term. The log-likelihood value drops by 86 points and the Durbin-Watson (D-W) test statistic changes from 1.63 to 0.50. This confirms that the general publicity variables are indeed jointly significant and ignoring them would lead to the error term being serially correlated.

One possible reason why the general publicity variables are not individually significant is that some of them are highly correlated (i.e., a multi-collinearity problem). We check the correlation of the general publicity variables and find that the correlation between $STK\_lc_t^s$ and $STK\_rh_t^s$ is higher than 0.99.[25] In the fourth specification, we estimate the model by just dropping $STK\_rh_t^s$. As expected, the $STK\_lc_t^s$, becomes positive and significant.[26]

To get a sense of the relative importance of the inclusive value term and the general publicity variables, we estimate the fifth specification in which we drop the inclusive value. The results show that the log-likelihood value drops by 120 points. By comparing it to the third specification, we can see that the results indicate that even without any general publicity effects, the regular patient-physician interactions due to drug specific publicity and promotional mix (captured by the inclusive value) are able to explain a large proportion of market expansion. But we should note that the general publicity effects are still important and should not be ignored.

6.2 Brand Choice Stage

In estimating the brand choice stage, we use the functional form for $f_j(.)$ in equation (2). For identification purposes, we normalize $\gamma_{mevacor}$ to zero. We also supplement the specification with several

[25]The correlation between $STK\_lc_t^s$ and $STK\_se_t^s$ is -0.54.

[26]The results of dropping $STK\_lc_t^s$ and keeping $STK\_rh_t^s$ is similar, but it produces slightly lower log-likelihood value.

additional control variables. To capture the impact of landmark clinical evidence on reductions in heart disease risks, we include an interaction term $STK\_clinical_{jt} * STK\_detail_{jt}$. The non-landmark clinical evidence has established that there is a difference between statins in terms of their effectiveness in lowering cholesterol. To capture this, we introduce a drug-specific quality variable, $LC\_quality_j$, using information on each drug's cholesterol lowering ability from a meta-analysis by Law et al. (2003), and we interact it with STK_detail_{jt}.[27]

We first estimate the model by assuming that the error terms are i.i.d. However, the Durbin and Watson (D-W) test statistics are less than 0.5 for all specifications that we tried. This indicates that the error terms are highly serially correlated so that the estimates may be very unreliable. To take the serial correlation of the error terms into account, we assume they follow an AR(1) process and use the Cochrane-Orcutt procedure (Cochrane and Orcutt, 1949; Pindyck and Rubinfeld, 1997) to estimate the model.

We report the results in Table 7. Specification (2) is our main specification. As expected, we find that STK_detail is positive and significant. The interaction term, $LC\_quality_j * STK\_detail_{jt}$, is also positive, but not significant.[28] In contrast, $STK\_clinical_{jt} * STK\_detail_{jt}$ is positive and significant. This suggests that to some degree physicians rely on detailing to learn about the landmark clinical trials and each statin's ability to reduce heart disease risks – this is consistent with the notion that it is much harder to learn about heart disease risks from patients' experiences, possibly because heart attacks or strokes are rare events.

We now investigate the relative importance of the promotional variables (which are directly controlled by firms) and publicity variables (over which firms have much less control) in explaining the demand for brand choice. Specification (1) drops the publicity variables. Compared with specification

[27] $LC\_quality$ is constructed by averaging the mean LDL reductions across strengths of each drug reported in Table 2 of Law et al. (2003).

[28] The insignificance could be because $LC\_quality$ only measures a drug's ability to reduce LDL. As mentioned earlier, some statins can also increase HDL, but $LC\_quality$ does not capture it.

(2), which is our full model, the log-likelihood decreases by about 70 points. On the other hand, by comparing specifications (2) and (3), which drops the detailing variables, the log-likelihood decreases by more than 240 points. The difference suggests that detailing is more important than publicity in explaining brand choice.

We should also highlight that omitting the publicity variables could yield misleading estimates of the impact of promotional mix in the brand choice stage. In specification (1), $STK\_clinical_{jt} * STK\_detail_{jt}$ now becomes insignificant, $LC\_quality_j * STK\_detail_{jt}$ becomes negative and significant, and the carryover rate for detailing and journal advertising becomes larger than 1. The results from specification (2) (our full specification) do not have any of these counter-intuitive estimates.

6.3 Are Detailing and Publicity Complements or Substitutes?

We now address the question of whether different types of publicity serve as substitutes or complements for a manufacturer's own marketing communication efforts (i.e., detailing in this case). Studying the interaction terms between STK_detail_{jt} and different types of publicity stocks in the brand choice stage allows us to answer this question.[29]

From before, our hypothesis (i) is that: for simple efficacy issues such as a statin's ability to lower cholesterol or its side-effects, publicity about these issues and detailing should be substitutes. Our hypothesis (ii) is that: for complicated efficacy issues such as a drug's ability to reduce heart disease risks, publicity about these issues and detailing should be complements.

Referring to specification (2) of Table 7, we see that $STK\_lc\_nc_{jt}$ is positive and significant, while the interaction term, $STK\_lc\_nc_{jt} * STK\_detail_{jt}$, is negative and significant. This indicates that the higher STK_detail_{jt}, the smaller the marginal impact of $STK\_lc\_nc_{jt}$, and vice versa, suggesting that detailing and publicity are substitutes in this case. These results are consistent with our hypothesis (i).

Looking next at the impact of publicity about heart disease risks, we see that the coefficient on

[29]Note that we did not include any interaction terms between the inclusive value and general publicity in the market expansion stage because the multi-collinearity problem of the general publicity variables make it difficult for us to disentangle them.

$STK\_rh\_nc_{jt}$ is negative and significant while the coefficient on its interaction term with STK_detail_{jt} is positive and significant. Given the observed values of STK_detail_{jt}, the marginal effect of $STK\_rh\_nc_{jt}$ is almost always positive. This provides evidence to support our hypothesis (ii) that detailing and publicity about heart disease risks are complements.

We also find that the coefficient on $STK\_se\_nc_{jt}$ is positive and significant while the coefficient on its interaction with STK_detail_{jt} is negative and significant. The latter implies that publicity about side-effects and detailing are also substitutes. Since most of this publicity is of the form "drug X has very few side-effects" which is quite simple, it is plausible that, as with publicity about lowering cholesterol, this sort of publicity and detailing are also substitutes.

Interestingly, the coefficient on $STK\_se\_c_{jt}$ is negative and significant and its interaction with STK_detail_{jt} is positive and significant. At first blush, these results seem counterintuitive. But by examining the news articles more carefully, we find that most *se_c* articles mentioned that Baycol has potentially severe side-effects (it can potentially cause permanent renal muscle damage) and hence needed to be withdrawn from the market, while other statins are relatively safe. With this information, we assign -1 to Baycol and +1 to other statins, with the idea that such news should allow other statins to gain market share at the expense of Baycol. However, these articles actually appeared after 2001, the year when Baycol was withdrawn.[30] As a result, the relative effects of the *se_c* variable cannot be realized because Baycol no longer belongs to the choice set. On the other hand, because Baycol's side-effects are so severe, it is conceivable that those articles create an overall negative impression of statins. Consequently, the average own effect of $STK\_se\_c_{jt}$ may turn out to be negative. But when a patient sees a physician and raises his concerns about side-effects of statins, the likelihood that the physician can correct such a misconception would increase with detailing, provided that the sales representative discusses the relative severity of the side-effects of the drug being promoted. This may explain why the

[30] Articles about Baycol reappeared after its exit because some clinical trial data is only available later, and Vioxx, a pain-relief drug, was found to increase heart disease risks (and finally was withdrawn in 2004). This leads to a public concern whether the FDA is monitoring the safety of drugs properly, and examples like Baycol was cited.

interaction term, $STK\_se\_c_{jt} * STK\_detail_{jt}$, is positive.

Finally, before we leave this subsection, it is worth noting that both $STK\_lc\_c_{jt}$ and $STK\_rh\_c_{jt}$ are insignificant. To try to understand why, we examined the data patterns more carefully and found that lc_c and rh_c are highly correlated. This is because the publicity that these two variables represent is largely related to two landmark clinical studies: REVERSAL (published in November 2003) and PROVE-IT (published in March 2004). These two studies compare Lipitor with Pravachol and find that Lipitor is more effective in both lowering cholesterol and reducing heart disease risks. If publicity related to REVERSAL and PROVE-IT has little impact on demand, $STK\_lc\_c_{jt}$ and $STK\_rh\_c_{jt}$ can turn out to be insignificant. This may well have been the case given how late these studies appear. By late 2003/early 2004, six years after the inception of Lipitor, it might have become well-known that Lipitor is more effective in lowering cholesterol; in addition, most statin users might have concluded that Lipitor was the most effective drug in reducing heart disease risks. In this case, articles related to these two studies merely confirmed consumer's beliefs and so had little real demand effects. Finally, the variable, STK_debate_{jt}, is insignificant, indicating that the debatable articles about Crestor had no impact on its demand.

6.4 Limitations

Before we discuss the managerial and public policy implications of our results, it is important for us to recognize the limitations of our research. The main limitation is that we do not observe the content of detailing. Therefore, when we construct the detailing stock variable, we implicitly assume that it is a stable construct, in the sense that the proportion of detailing effort devoted to each information dimension remains the same over time. This is a strong assumption and our inference about whether publicity and detailing are substitutes or complements depends on this assumption. Suppose that when the stock of publicity about reducing heart disease risks rises, pharmaceutical companies direct their sales forces to devote more or less effort to discussing a drug's ability to lower cholesterol, or its side-

effects. Such a reallocation of effort may change the marginal return of detailing, even if publicity has no interaction with detailing. If it happens that it leads to an increase in the marginal return of detailing, we would mis-infer that publicity about reducing heart disease risks and detailing are complements. Similar inference mistakes may occur with other types of publicity stocks.[31]

In an effort to address this issue, we have studied the medical literature to seek guidance on the endogeneous reallocation of sales efforts in detailing. However, we have been unable to find any research that investigates this topic, perhaps because research on publicity is still at the early stage. It is also unclear to what extent pharmaceutical companies track publicity.[32] If pharmaceutical companies do not have a reliable way to measure the multi-dimensional nature of publicity, then the allocation of sales effort should not depend on the publicity stocks much. This should alleviate the concern that our results on the interaction terms are biased. Of course, the best way to address this potential problem is to collect information on the content of detailing over time and create a new data set on the multi-dimensional information aspect of detailing. Such an investigation is beyond the scope of this paper, and we leave it for future research.

6.5 Managerial and Public Policy Implications

Subject to the above caveats, our results suggest that manufacturers might find it worthwhile to track the amount of publicity for different product characteristics. Then, if a manager also knows which types of publicity are substitutes or complements for traditional marketing channels, he/she can allocate his/her sales force more efficiently over time. Another implication is that a manager could potentially direct his/her sales force to focus more on certain types of information, depending on the extent to which each has already been covered by the news. For instance, according to our analysis, if there is a surge in news coverage about drug A's ability to lower cholesterol, the marketing manager might want

[31] Note, however, that we have allowed detailing to interact with landmark clinical trial stocks and the interaction is positive and significant. So, to some extent, we have controlled for the changes in return of detailing over time, potentially mitigating some of these concerns.

[32] We have also checked the databases available for purchase at IMS Health, the company that specializes in collecting marketing data in the pharmaceutical industry. To the best of our knowledge, none of its databases track publicity.

to instruct the sales force to focus their presentation on other aspects of the drug. This is because a patient/physician may have already learned the information from the news. On the other hand, when more news about drug B's ability to reduce heart disease risks comes out, a sales representative can be more effective by spending relatively more time discussing this aspect of the drug with physicians. This could allow detailing to have an even stronger impact on the patient-physician interaction.

In this regard, it is interesting to compare the elasticities of detailing between specifications (1) and (2) of the brand choice stage in Table 7. This difference illustrates the extent of the bias generated by failing to include drug specific publicity variables in the brand choice stage. By comparing rows 1 and 2 in Table 8, we find that omitting publicity variables leads to much higher detailing elasticities for Mevacor, Zocor, Pravachol and Lipitor but lower elasticities for Baycol and Crestor. This indicates that ignoring publicity variables and their interactions with detailing could lead to non-trivial biases in estimates of the effectiveness of detailing. The directions of the biases are difficult to predict a priori because the signs of the publicity variables and the interaction terms are mixed. But it is important to highlight the managerial implication: marketing research that ignores publicity may well overstate the effectiveness of detailing, thus leading to wasteful (from a profit perspective) detailing expenditures.

An important question in health-care management is how to control the rapid increase in prescription drug costs. Many have suggested that a major factor driving up prescription drug prices is high marketing costs, especially detailing costs. Based on the belief that detailing done by manufacturers is primarily persuasive, as opposed to informative, some have proposed to limit the amount of detailing. Some countries have gone even further and banned detailing. Opponents argue that such policies ignore the informational benefits provided by detailing and that bans would slow down the learning process and hinder the adoption by physicians of the best drugs for their patients.

Our results shed light on this debate (e.g., Chan et al., 2013; Ching and Ishihara, 2012; Narayanan and Manchanda, 2009). We provide new evidence that detailing is informative – otherwise, the interac-

tion terms between detailing and different types of publicity should be insignificant. More importantly, we identify a new informative benefit of detailing (and potentially for marketing communication in general) – when the information is complicated to explain, informative marketing communication could complement the information provided in the news media.

6.6 Robustness Checks

6.6.1 Does a Multi-Dimensional Coding Scheme Matter?

In this subsection, we compare the results from a multi-dimensional coding scheme with those from a uni-dimensional coding scheme. To compare the results from two different coding schemes, we create a uni-dimensional publicity variable as follows. For each dimension, we assign "+1" ("-1") if an article contains sentences which favor (do not favor) the focal drug. Then we sum up the scores across the three dimensions for each article. If the sum is positive (negative), the article is assigned as "+1"("-1"). By summing up the score for each article per month, we create a uni-dimensional publicity variable.

Specification (4) in Table 7 uses two uni-dimensional variables: STK_c_{jt} (comparison) and STK_nc_{jt} (non-comparison). The results are largely consistent with specification (2). In particular, STK_nc_{jt} is positive and significant and its interaction with STK_detail_{jt} is negative and significant. Note, though, that $LC\_quality_j * STK\_detail_{jt}$ now becomes negative. The log-likelihood of this specification drops by 15 points compared with specification (2) (i.e., the full specification), so it still provides reasonable fit to the data. However, it should be emphasized that the multi-dimensional coding scheme offers the advantage of allowing us to learn the impact of different forms of publicity. Without it, we cannot tell which type of publicity will serve as a substitute/complement for detailing, and hence cannot provide the managerial and public policy implications that we discussed above.

Table 8 compares the detailing elasticities generated from specifications (2) and (4). The differences are in the range of 10% (Lipitor) to 23% (Baycol), with the exception that the detailing elasticity for Mevacor more than doubles. This provides further support for using the multi-dimensional coding

scheme that we propose here.

6.6.2 Positive vs. Negative Publicity

The way we construct the publicity variables is to allow positive and negative publicity to have equal weights when we add them up. However, it is possible that consumers assign them different weights. To investigate this alternative way to interpret the data, we attempt to construct positive and negative publicity variables. Unfortunately, we find that most of the publicity for lc and rh is positive (more than 95%), while publicity for se is more evenly distributed. Therefore, we only construct positive and negative publicity variables for side-effects (se_p and se_n). The estimation results are presented in specification (5) in Table 7. We find that STK_se_p is positive and significant, and STK_se_n is not significant. Moreover, all other results which we discuss above remains qualitatively unchanged.

7 Conclusion

This research proposes a new way to interpret publicity data and studies the effects of publicity on the demand for prescription drugs. To capture the multi-dimensional messages contained in a publicity event (e.g., a news article), we map information contained in each event into a multi-dimensional attribute space. Our approach allows us to (i) avoid many ambiguities encountered by the traditional uni-dimensional coding method, (ii) investigate the relative importance of different types of publicity, and (iii) investigate which types of publicity are substitutes or complements for firms' marketing communication efforts. Our methodology is particularly important for marketing experience goods, where informative marketing communication tools play an important role in helping consumers to choose the right product for them. Manufacturers can use this method to interpret news in a systematic way, and therefore analyze their impacts using standard regression analysis.

We apply our coding method to the statin market. Our results suggest that non-comparison publicity regarding a drug's ability to lower cholesterol and side-effects are a substitute for detailing activity.

We argue that, in both cases, the information that the publicity conveys is rather straightforward and precise, so publicity can be an effective substitute for detailing.

On the other hand, we find evidence that publicity regarding a drug's ability to reduce heart disease risks and detailing are complements. We argue that, in this case, the noisiness of an information source would be a primary issue because it is much more complicated to explain and understand heart disease risks. This empirical result is consistent with the hypothesis that publicity acts as corroborative evidence to bolster physicians' confidence about the detailing source. It is worth noting that Luan and Sudhir (2010) also find evidence that word of mouth (WOM) and advertising are complements in the U.S. movie DVD market. This suggests that the idea of corroborating effects from multiple marketing communication channels can be extended to other markets as well. Future research should investigate whether the strength of the corroborative effects depends on the complexity of information in other contexts.

By incorporating publicity and clinical trial data into the demand analysis, we also control for the demand shocks that are typically unobserved in previous research. This allows us to alleviate the endogeneity problem that is present for marketing-mix variables. In particular, we find evidence that the estimated effects of detailing could be very misleading if we fail to control for publicity.

Finally, although our coding scheme is designed specifically for the statin market, the idea of a multi-dimensional coding scheme to capture the different aspects of publicity is more general. Many products have multiple characteristics that could be highlighted by news articles. Automobile publicity, for instance, could focus on safety or environmental impact, or any other different features. Focusing attention on a single dimension could be misleading in such a context. It is conceivable that our coding method could be extended to study its effects in other markets. We leave this topic for future research.

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Table 1: Summary information on Statins

Brand	Molecule	Entry Date	Generic Entry	Manufacturer
Mevacor	lovastatin	Jul-1988	Apr-1997	Merck & Co.
Zocor	simvastatin	Aug-1990	Jan-2003	Merck & Co.
Pravachol	pravastatin	Oct-1990	Jul-2000	Bristol-Myers Squibb
Lescol	fluvastatin	Mar-1994	N.A.[1]	Novartis
Lipitor	atorvastatin	Mar-1997	N.A.[1]	Pfizer
Baycol	cerivastatin	Mar-1998	N.A.[2]	Bayer
Crestor	rosuvastatin	Feb-2003	N.A.[1]	AstraZeneca

1 - The patent expiration date is beyond our sample period.

2 - Baycol was withdrawn in August 2001 before its patent expires.

Table 2: Number of Sources and Articles Covering Statins by Year

Year	# of Sources	# of Articles
1986	7	6
1987	8	49
1988	8	12
1989	9	8
1990	10	10
1991	11	11
1992	11	7
1993	12	46
1994	13	38
1995	13	57
1996	18	44
1997	20	80
1998	22	113
1999	22	131
2000	22	148
2001	26	320
2002	30	433
2003	31	481
2004	31	760
Total		2,754

Table 3: Summary of Publicity Variables

Lowering Cholesterol Levels	# of Months	Non-comparison					Comparison				
		# of Articles	Values				# of Articles	Values			
			Mean	Std. Dev.	Min.	Max.		Mean	Std. Dev.	Min.	Max.
Mevacor	198	255	1.80	1.93	0	9	6	-0.04	0.20	-1	0
Zocor	173	470	3.31	3.55	0	17	14	-0.04	0.29	-1	1
Pravachol	171	262	1.85	2.00	0	11	35	-0.22	1.45	-13	1
Lescol	130	33	0.24	0.62	-1	3	2	0.00	0.12	-1	1
Lipitor	94	707	7.53	7.00	0	28	58	0.15	1.57	-4	11
Baycol	41	9	0.22	0.65	0	3	0	0.00	0.00	0	0
Crestor	23	120	5.22	5.95	0	20	16	0.70	1.11	0	4
Reducing Risks of Heart Disease	# of Months	Non-comparison					Comparison				
		# of Articles	Values				# of Articles	Values			
			Mean	Std. Dev.	Min.	Max.		Mean	Std. Dev.	Min.	Max.
Mevacor	198	41	0.25	0.73	-1	4	0	0	0	0	0
Zocor	173	94	0.56	1.39	-3	9	2	0.01	0.12	0	1
Pravachol	171	81	0.54	1.11	-1	7	32	-0.23	1.49	-15	0
Lescol	130	3	0.02	0.15	0	1	0	0	0	0	0
Lipitor	94	92	0.98	2.18	0	15	32	0.32	1.83	-1	15
Baycol	41	1	0.02	0.16	0	1	0	0	0	0	0
Crestor	23	7	0.30	0.63	0	2	0	0	0	0	0
Side-Effects	# of Months	Non-comparison					Comparison				
		# of Articles	Values				# of Articles	Values			
			Mean	Std. Dev.	Min.	Max.		Mean	Std. Dev.	Min.	Max.
Mevacor	198	5	0.04	0.22	0	2	5	0.01	0.22	-1	2
Zocor	173	16	0.08	0.48	-1	4	4	0.01	0.21	-1	2
Pravachol	171	15	0.08	0.38	-1	3	10	0.08	0.39	0	2
Lescol	130	0	0.00	0.00	0	0	5	0.04	0.23	0	2
Lipitor	94	20	0.07	0.42	-1	1	9	0.03	0.40	-2	2
Baycol	41	2	0.05	0.31	0	2	0	0	0	0	0
Crestor	23	72	-0.07	0.49	-5	0	1	-0.04	0.21	-1	0

Table 4: Landmark Clinical Trials

Title	Publication Date (mm/yy)	Journal	Drugs Studied	# of Subjects	Follow-up Period	# of Endpoints	Results	Sponsors
4S	12/94	Lancet	Zocor	4,444	5.4 yrs	34	Reduce risk of coronary events and improve survival	Merck & Co.
WOSCOPS	11/95	NEJM	Pravachol	6,595	4.9 yrs	20	Reduce MI and death from cardiovascular causes	Bristol-Myers Squibb
CARE	10/96	NEJM	Pravachol	4,159	5 yrs	11	Reduce coronary events	Bristol-Myers Squibb
AFCAPS/ TexCAPS	05/98	JAMA	Mevacor	6,605	5.2 yrs	8	Reduce risk for the first acute coronary event	
LIPID	11/98	NEJM	Pravachol	9,014	6.1 yrs	25	Reduce the cardiovascular events and death	Bristol-Myers Squibb
MIRACL	04/01	JAMA	Lipitor	3,086	16 wks	15	Reduce recurrent ischemic events	Pfizer Inc.
LIPS	06/02	JAMA	Lescol	1,677	3.9 yrs	7	Reduce the risk of major adverse cardiac events	Novartis
HPS	07/02	Lancet	Zocor	20,536	5 yrs	28	ReduceMI, stroke, and revascularisation	Merck & Co.
PROSPER	11/02	Lancet	Pravachol	5,804	3.2 yrs	19	Reduce coronary heart disease death and non-fatal myocardial infarction risk	Bristol-Myers Squibb
ALLHAT- LLT	12/02	JAMA	Pravachol**	10,355	4.8 yrs	14	Reduce neither mortality nor CHD significantly compared with ususal care	Pfizer
ASCOT- LLA	05/03	Lancet	Lipitor	10,305	3.3 yrs	11	Reduce cardiovascular morbidty	Pfizer
ALERT	06/03	Lancet	Lescol	2,102	5.1 yrs	24	Reduce Cardiac Death and non-fatal MI	Novartis
REVERSAL	03/04	JAMA	Lipitor*, Pravachol	2,163	1.5 yrs	13	Reduce coronary atherosclerosis	Pfizer Inc.
PROVE IT -TIMI	04/04	NEJM	Lipitor*, Pravachol	4,162	2 yrs	10	Protect against death or cardiovascular events	Bristol-Myers Squibb
ALLIANCE	06/04	JACC	Lipitor	2,422	4.3 yrs	12	Reduce the risk of first cardiovascular event	Pfizer
CARDS	08/04	Lancet	Lipitor	2,838	3.9 yrs	15	Lower incidence of coronary events, stroke, and coronary revascularization procedures	Pfizer
A to Z	09/04	JAMA	Zocor	4,498	2 yrs	10	Reduce cardiovascular events.	Merck & Co.

* Both the comparative studies show superior efficacy of Lipitor over Pravachol.

** ALLHAT-LLT shows unfavorable results to Pravachol.

Table 5: Summary Statistics on No. of Prescriptions and Promotional-mix

Sample period: March 1993 to December 2004

	# of Months	Detailing (CAD)				Journal Advertising (Pages)				# of Prescriptions			
		Mean	Std. Dev.	Min.	Max.	Mean	Std. Dev.	Min.	Max.	Mean	Std. Dev.	Min.	Max.
Mevacor	142	26,201	38,998	0	133,017	4,204	7,832	0	27,000	65,214	23,639	30,014	100,181
Zocor	142	105,990	70,110	0	343,295	19,034	12,749	0	48,000	154,288	87,246	33,084	315,666
Pravachol	142	89,346	67,169	0	260,511	10,794	11,734	0	46,000	118,210	45,582	35,382	176,228
Lescol	130	69,539	66,541	0	256,938	7,669	10,623	0	39,000	23,894	7,809	310	37,486
Lipitor	94	259,015	78,993	83,879	514,180	33,506	9,415	0	60,000	415,147	265,539	0	925,001
Baycol	41	217,767	72,537	105,539	494,145	22,030	10,979	0	44,000	36,873	26,869	846	93,001
Crestor	23	300,349	75,784	182,725	495,986	35,304	16,230	0	59,000	101,096	68,685	0	195,514

Sample period: July 1988 to February 1993

	# of Months	Detailing (CAD)				Journal Advertising (Pages)			
		Mean	Std. Dev.	Min.	Max.	Mean	Std. Dev.	Min.	Max.
Mevacor	56	101,536	34,844	6,000	226,000	22,483	19,618	0	109,224
Zocor	31	124,355	61,107	0	316,000	34,096	21,415	0	100,862
Pravachol	29	165,759	56,214	74,000	261,000	27,809	19,289	7,155	80,172

Table 6: Market Expansion Results

Variables	(1)		(2)		(3)		(4)		(5)	
	Coef.	S.E.	Coef.	S.E.	Coef.	S.E.	Coef.	S.E.	Coef.	S.E.
Inclusive Value$_t$	**0.5571**	0.0218	**0.5530**	0.0095	**0.6502**	0.0075	**0.5508**	0.0080		
STK_lc^s_t	0.3286	0.2218	0.3452	0.2259			**0.2752**	0.1016	0.7062	0.4458
STK_rh^s_t	-0.0963	0.3147	-0.1149	0.3407					0.3635	0.7190
STK_se^s_t	0.4574	0.5127	0.4381	0.5387			0.3594	0.4488	**-4.6654**	0.8439
Number of Statins$_t$	-0.0035	0.0173							**0.3715**	0.0191
Carryover (Publicity)	**0.9820**	0.0203	**0.9810**	0.0211			**0.9849**	0.0164	**0.9772**	0.0115
Intercept	**-4.3971**	0.0384	**-4.3968**	0.0356	**-4.7612**	0.0381	**-4.3896**	0.0319	**-3.9021**	0.0860
Standard Deviation of ν_t	**0.0616**	0.0037	**0.0616**	0.0037	**0.1123**	0.0068	**0.0616**	0.0037	**0.1466**	0.0086
Log-Likelihood	194.3015		194.2779		108.9953		194.2063		71.1315	
DW	1.6389		1.6346		0.5024		1.6324		0.4834	

Estimates shown in bold are significant at 5% level.

Variable Definitions

Variable	Definition
Inclusive Value$_t$	Inclusive value from the second stage estimation
STK_lc^s_t	Monthly stock of publicity for statin general in the dimension of lowering cholesterol levels
STK_rh^s_t	Monthly stock of publicity for statin general in the dimension of risks of reducing heart disease
STK_se^s_t	Monthly stock of publicity for statin general in the dimension of side-effects
Number of Statins$_t$	Number of statins available in the market

Table 7: Brand Choice Results

Variables	Erorr terms are AR(1).									
	(1)		(2)		(3)		(4)		(5)	
	Coef.	S.E.	Coef.	S.E.	Coef.	S.E.	Coef.	S.E.	Coef.	S.E.
STK_detail_{jt}	**1.4800**	0.0993	**1.4385**	0.2526			**1.9001**	0.1798	**1.1785**	0.2081
$STK\_journal_{jt}$	**0.0597**	0.0065	**0.0487**	0.0077			**0.0580**	0.0070	**0.0433**	0.0066
$STK\_clinical_{jt}$*STK_detail_{jt}	-0.0012	0.0011	**0.0048**	0.0016			**0.0052**	0.0015	**0.0047**	0.0015
$LC\_quality_j$*STK_detail_{jt}	**-0.1034**	0.0153	0.0286	0.0278			**-0.0721**	0.0210	**0.0495**	0.0252
STK_c_{jt}							**-1.4593**	0.3110		
STK_nc_{jt}							**0.5999**	0.0363		
$STK\_lc\_c_{jt}$			-0.5536	0.8009	-1.0290	0.7320			-0.6998	0.6072
$STK\_lc\_nc_{jt}$			**0.8015**	0.0824	**0.4604**	0.0477			**0.6667**	0.0450
$STK\_rh\_c_{jt}$			-0.9594	0.7515	0.1746	0.6571			-0.1018	0.7321
$STK\_rh\_nc_{jt}$			**-0.4761**	0.2314	-0.3335	0.2172			**-0.4378**	0.1823
$STK\_se\_c_{jt}$			**-2.7596**	0.8402	**2.7174**	1.0431				
$STK\_se\_nc_{jt}$			**2.8796**	0.7097	**4.2183**	0.7037				
$STK\_se\_p_{jt}$									**2.3791**	0.4363
$STK\_se\_n_{jt}$									1.6591	0.9009
STK_debate_{jt}			-0.0011	0.0019	0.0013	0.0037	-0.0030	0.0019	-0.0109	0.0082
STK_c_{jt}*STK_detail_{jt}							**0.8802**	0.2097		
STK_nc_{jt}*STK_detail_{jt}							**-0.3621**	0.0274		
$STK\_lc\_c_{jt}$*STK_detail_{jt}			-0.3888	0.6756					-0.0281	0.5204
$STK\_lc\_nc_{jt}$*STK_detail_{jt}			**-0.6461**	0.0670					**-0.4749**	0.0373
$STK\_rh\_c_{jt}$*STK_detail_{jt}			**1.3692**	0.5735					0.3675	0.4950
$STK\_rh\_nc_{jt}$*STK_detail_{jt}			**1.0172**	0.2492					**0.8697**	0.1845
$STK\_se\_c_{jt}$*STK_detail_{jt}			**3.2682**	1.0053						
$STK\_se\_nc_{jt}$*STK_detail_{jt}			**-1.8871**	0.8237						
$STK\_se\_p_{jt}$*STK_detail_{jt}									**-1.4290**	0.4561
$STK\_se\_n_{jt}$*STK_detail_{jt}									-1.5542	0.8659
Carryover (Detail/JournalAd)	**1.0035**	0.0006	**0.9953**	0.0010			**0.9978**	0.0009	**0.9971**	0.0008
Carryover (Publicity)			**0.9929**	0.0011	**0.9894**	0.0014	**0.9933**	0.0012	**0.9937**	0.0009
Lipitor	**2.3796**	0.1076	**2.3124**	0.1630	**2.4831**	0.1359	**2.4700**	0.2048	**2.3624**	0.1663
Baycol	**1.2030**	0.1204	**1.3255**	0.1771	**1.3908**	0.1564	**1.5278**	0.2300	**1.4027**	0.1804
Lescol	**-0.3924**	0.1066	**0.5027**	0.1460	**0.3874**	0.1248	0.2923	0.1597	**0.5749**	0.1484
pravachol	-0.1014	0.0620	**0.7935**	0.1212	**1.2255**	0.1016	**0.5499**	0.1000	**0.8566**	0.1191
Crestor	**3.5790**	0.1828	**2.7684**	0.2217	**3.0607**	0.2230	**3.1932**	0.3027	**2.9102**	0.2173
Zocor	**-0.2396**	0.0279	**0.7169**	0.1134	**1.3259**	0.0996	**0.4884**	0.0911	**0.7576**	0.1098
Standard Deviation of ξ_{jt}-$\xi_{Mevacor,t}$	**0.0581**	0.0022	**0.0513**	0.0015	**0.0786**	0.0025	**0.0526**	0.0015	**0.0508**	0.0016
Log-Likelihood	804.3670		875.3492		634.3291		860.9666		880.0030	
D-W Test	1.3118		1.3191		1.0743		1.2768		1.3413	

Estimates shown in bold are significant at 5% level.

Variable Definitions

Variable	Definition
STK_detail_{jt}	Monthly stock of detailing cost for drug *j*
$STK\_journal_{jt}$	Monthly stock of journal advertising page for drug *j*
$LC\_quality_j$*STK_detail_{jt}	LC_quality * Monthly stock of detailing cost for drug *j*
$STK\_clinical_{jt}$*STK_detail_{jt}	Monthly stock of landmark clinical trials * Monthly stock of detailing cost for drug *j*
STK_nc_{jt}	Monthly stock of number of non-comparsion articles favoring drug *j*
STK_c_{jt}	Monthly stock of number of comparsion articles favoring drug *j*
$STK\_lc\_nc_{jt}$	Monthly stock of number of non-comparsion articles favoring drug *j* in the dimension of lowering cholesterol levels
$STK\_lc\_c_{jt}$	Monthly stock of number of comparsion articles favoring drug *j* in the dimension of lowering cholesterol levels
$STK\_rh\_nc_{jt}$	Monthly stock of number of non-comparsion articles favoring drug *j* in the dimension of reducing risks of heart disease
$STK\_rh\_c_{jt}$	Monthly stock of number of comparsion articles favoring drug *j* in the dimension of reducing risks of heart disease
$STK\_se\_nc_{jt}$	Monthly stock of number of non-comparsion articles favoring drug *j* in the dimension of side effects
$STK\_se\_c_{jt}$	Monthly stock of number of comparsion articles favoring drug *j* in the dimension of side effects
$STK\_se\_p_{jt}$	Monthly stock of number of articles favoring drug *j* in the dimension of side effects
$STK\_se\_n_{jt}$	Monthly stock of number of articles not favoring drug *j* in the dimension of side effects
STK_debate_{jt}	Monthly stock of number of debatable articles for Crestor's side effects

Table 8: Average Detailing Elasticity

	Mevacor	Zocor	Pravachol	Lescol	Lipitor	Baycol	Crestor
No Publicity (spec. 1)	1.019	1.004	1.377	0.819	0.361	0.280	0.169
Multi-dimension publicity (spec. 2)	0.114	0.715	1.118	0.840	0.203	0.740	0.407
Single dimension publicity (spec. 4)	0.271	0.651	1.021	0.934	0.176	0.573	0.348

Note: The table reports average detailing elasticity of each statin in the brand choice stage specs (1), (2) & (4).

Figure 1: Monthly Number of Prescriptions for Statins

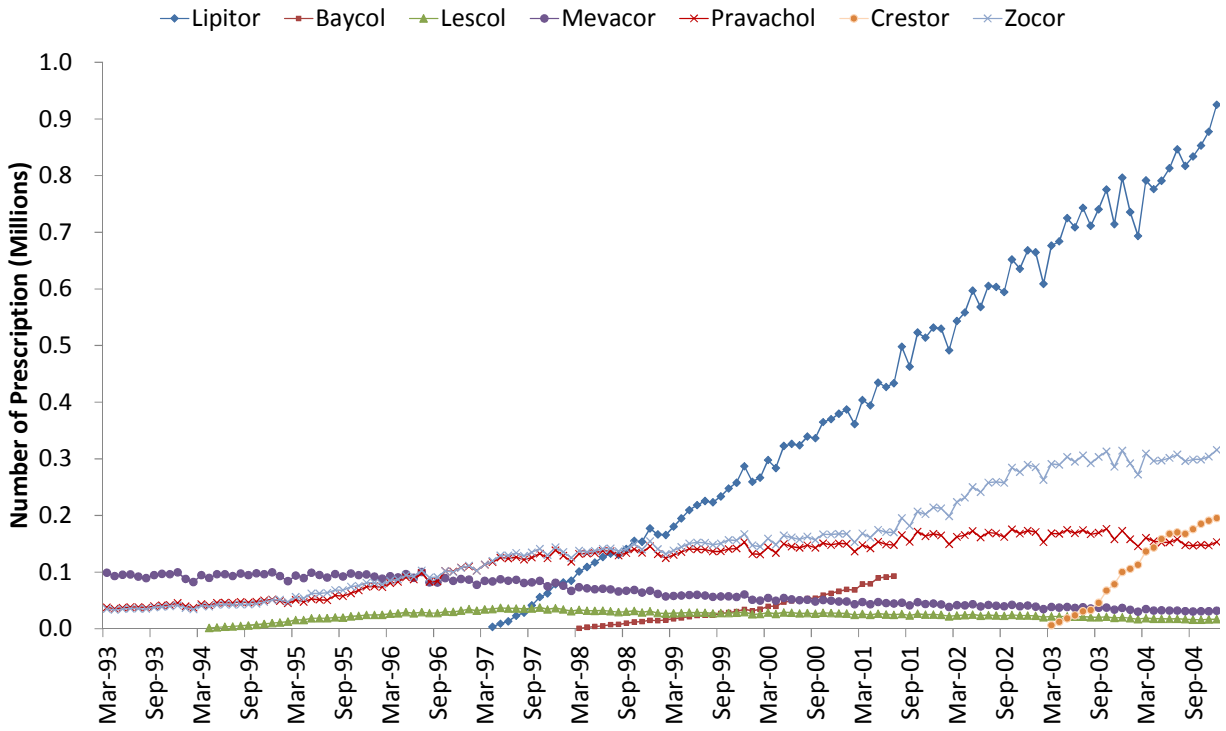


Figure 2: Monthly Detailing Flow for Four Leading Statins

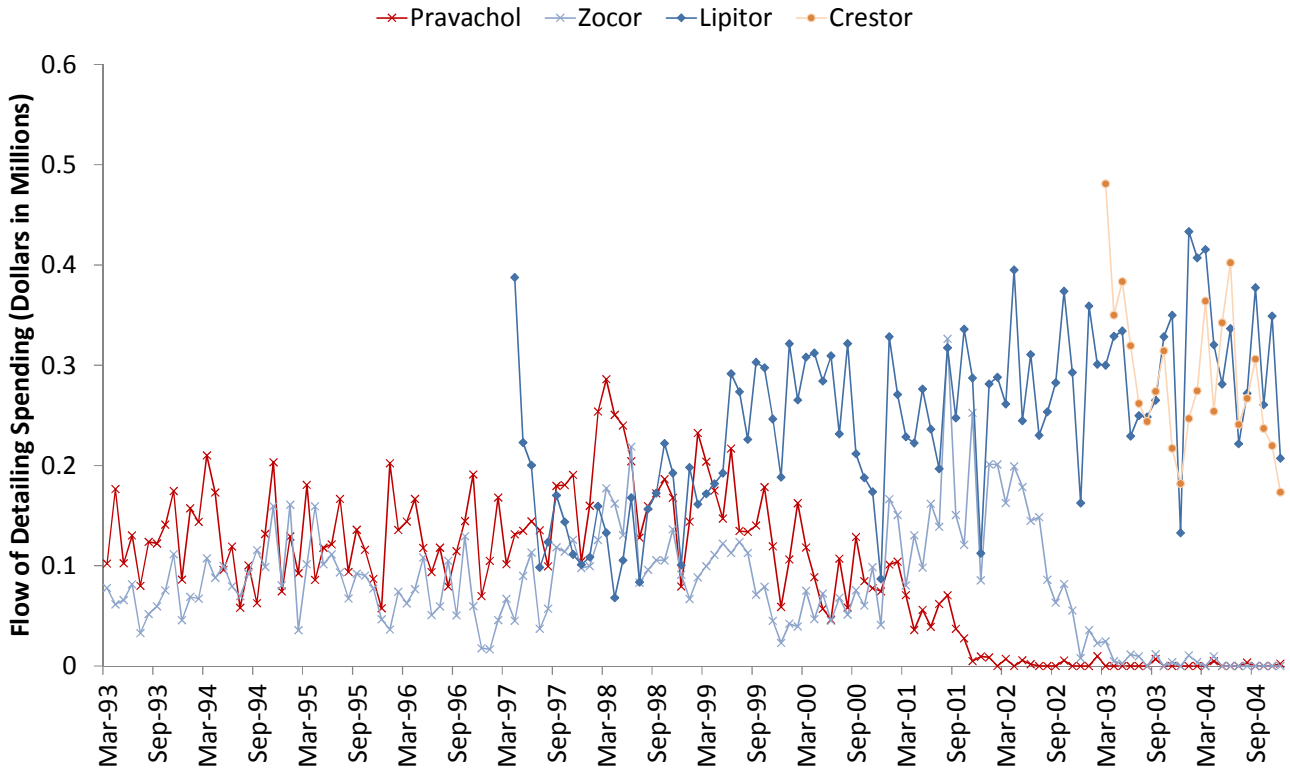


Figure 3: Monthly Medical Journal Advertising Flow for Four Leading Statins

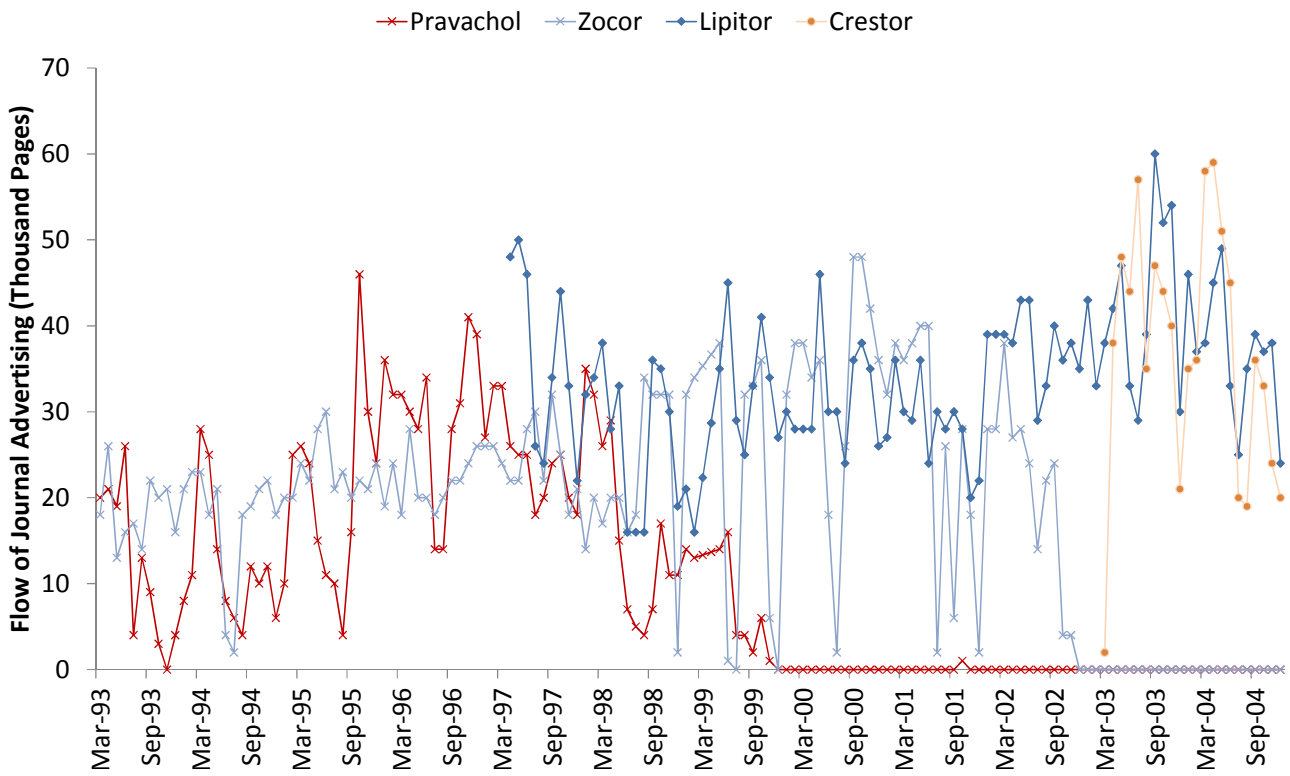
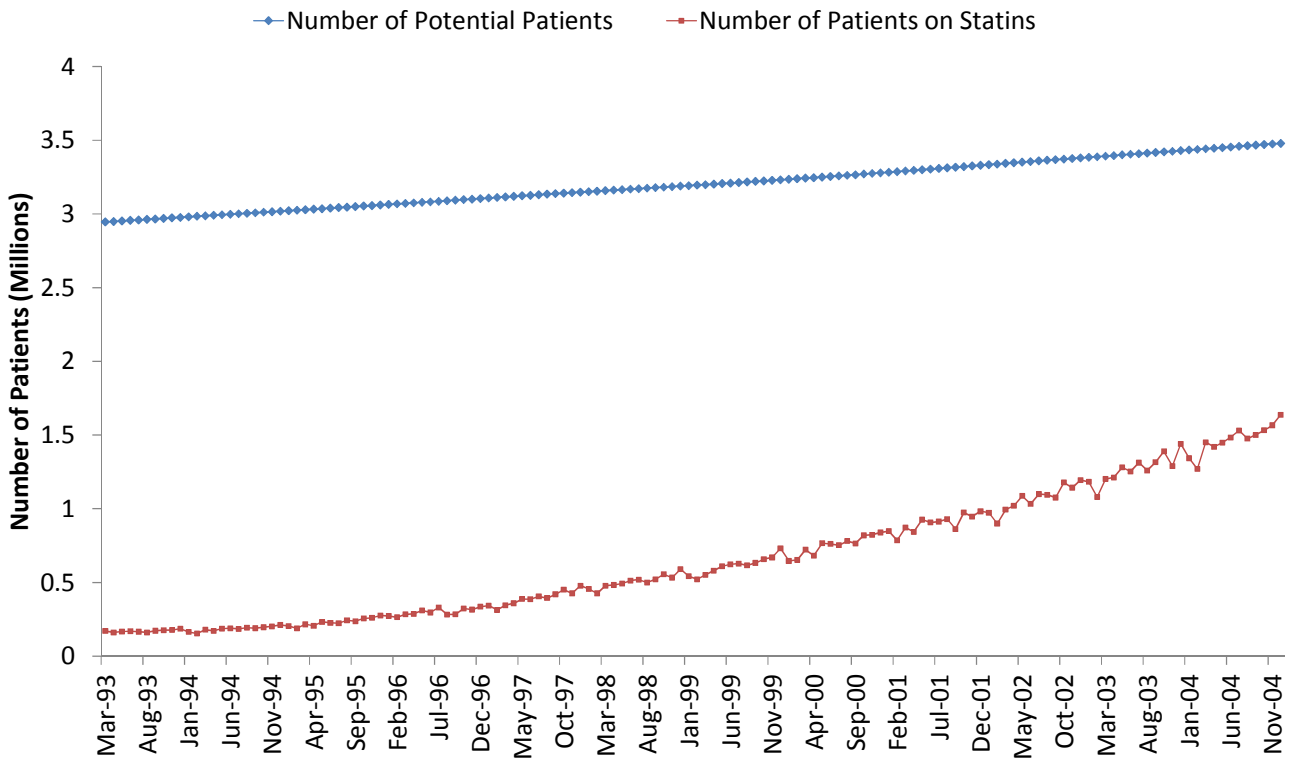


Figure 4: Potential Market Size and Total Demand for Statins



A Hypothesis Development (More Formal Analysis)

Suppose that physicians are uncertain about an attribute for drug j (the attribute could be *lc*, *rh* or *se*). Let Q_{jt} and σ^2_{jt} be the mean value and variance of physicians' consensus distribution of an attribute for drug j at time t after obtaining information from different sources. Let $Q^{D,k}_{jt}$ and σ^2_{dt} be the information signals and variance of the detailing source (k indexes the signals) and $Q^{P,l}_{jt}$ and σ^2_{pt} be the information signals and variance of the publicity source (l indexes the signals). Let n_{djt} and n_{pjt} be the amount of detailing and the amount of publicity in time t, respectively.

To capture publicity's potential role as corroborative evidence, we allow σ^2_{dt} to be a function of n_{pjt}.

$$\sigma^2_{dt} = \frac{\sigma^2_{d0}}{1+\phi(n_{pjt})} \simeq \frac{\sigma^2_{d0}}{1+\gamma_1 \cdot n_{pjt}}. \tag{A.1}$$

The theory of corroborative evidence (Godden, 2010) implies that $\partial\phi(.)/\partial n_{pjt} \geq 0$, and $\phi(n_{pjt}) \geq 0$. As a first order 'local' approximation, we use $\phi(n_{pjt}) \simeq \gamma_1 \cdot n_{pjt}$, and expect that $\gamma_1 \geq 0$. This approximation will simplify the notation.

To capture the potential role of rational inattention, we let $\hat{n}_{djt}$ be the amount of detailing to which physicians pay attention, and assume that $\hat{n}_{djt}$ is a function of (n_{djt}, n_{pjt}). We expect that the partial derivative of $\hat{n}_{djt}$ w.r.t. n_{djt} is positive, and w.r.t. n_{pjt} is negative. This is to capture the idea that if physicians learn sufficient information from the publicity source, the marginal gain of paying attention to sales representatives will be small. Therefore, the higher the publicity volume, the less likely that physicians would incur the costs of paying attention to detailing.[33] The idea that there is a cost of paying attention is borrowed from Reis (2006a,b). Here, we assume that the cost of obtaining information from publicity is zero (or close to zero) because patients can inform physicians about what they read in the news during their office visits. Since physicians are serving patients, it is likely that

[33]This does not necessarily mean that physicians do not meet with sales representatives. But during the conversation, when the content is about *lc*, physicians may choose not to pay attention. This is similar to some students who attend classes, but not listen to the lecture because paying attention requires mental resource. For another example of rational inattention in the brand choice literature, see Ching et al. (2014).

they would pay attention to what patients have to say. More specifically, we write,

$$\hat{n}_{djt} = n_{djt}(1 - \psi(n_{pjt})) \simeq n_{djt}(1 - \gamma_2 \cdot n_{pjt}). \tag{A.2}$$

Note that $\partial\psi(.)/\partial n_{pjt} \geq 0$, and $0 < \psi(n_{pjt}) < 1$. This implies that $\partial\hat{n}_{djt}/\partial n_{pjt} \leq 0$. Again, we use a first order 'local' approximation: $\psi(n_{pjt}) \simeq \gamma_2 \cdot n_{pjt}$, and expect that $\gamma_2 \geq 0$.

To simplify the notation further, we will drop the t subscripts in the following formulas (and keep in mind that all variables are measured in the same period). We modify the consensus distribution formulas in Winkler (1981) as follows to illustrate our argument.[34] For simplicity, we assume that information sources are independent.

$$Q_j = \frac{\hat{n}_{dj} \cdot \sigma_p^2}{\hat{n}_{dj} \cdot \sigma_p^2 + n_{pj} \cdot \sigma_d^2} \sum_k Q_j^{D,k} + \frac{n_{pj} \cdot \sigma_d^2}{\hat{n}_{dj} \cdot \sigma_p^2 + n_{pj} \cdot \sigma_d^2} \sum_l Q_j^{P,l}, \tag{A.3}$$

$$\sigma_j^2 = \frac{1}{\frac{n_{pj}}{\sigma_p^2} + \frac{\hat{n}_{dj}}{\sigma_d^2}}. \tag{A.4}$$

By substituting equation (A.1) into equation (A.4), the consensus variance term can be rewritten as:

$$\begin{aligned} \sigma_j^2 &= \frac{1}{\frac{n_{pj}}{\sigma_p^2} + \frac{\hat{n}_{dj} \cdot (1 + \gamma_1 \cdot n_{pj})}{\sigma_{d0}^2}} \\ &= \frac{\sigma_p^2 \cdot \sigma_{d0}^2}{n_{pj} \cdot \sigma_{d0}^2 + \hat{n}_{dj} \cdot (1 + \gamma_1 \cdot n_{pj}) \cdot \sigma_p^2} \\ &= \frac{\sigma_p^2 \cdot \sigma_{d0}^2}{n_{pj} \cdot \sigma_{d0}^2 + \hat{n}_{dj} \cdot \sigma_p^2 + \hat{n}_{dj} \cdot n_{pj} \cdot \gamma_1 \cdot \sigma_p^2}, \end{aligned} \tag{A.5}$$

or, the precision of the consensus distribution (i.e., $\pi_j \equiv \frac{1}{\sigma_j^2}$) can be written as:

$$\pi_j = \frac{n_{pj}}{\sigma_p^2} + \frac{\hat{n}_{dj}}{\sigma_{d0}^2} + \frac{\gamma_1 \cdot \hat{n}_{dj} \cdot n_{pj}}{\sigma_{d0}^2}. \tag{A.6}$$

By substituting equation (A.2) into equation (A.6), we have

$$\pi_j = \frac{n_{pj}}{\sigma_p^2} + \frac{n_{dj}}{\sigma_{d0}^2} - \frac{\gamma_2 \cdot n_{dj} \cdot n_{pj}}{\sigma_{d0}^2} + \frac{\gamma_1 \cdot n_{dj} \cdot n_{pj}}{\sigma_{d0}^2} - \frac{\gamma_1 \cdot \gamma_2 \cdot n_{dj} \cdot n_{pj}^2}{\sigma_{d0}^2}. \tag{A.7}$$

[34] An implicit assumption behind these formulas is that agents have a very diffuse initial prior before obtaining any information signals.

For information types that are relatively simple (e.g., *lc* and *se*), the publicity source can deliver the information fairly precisely. Given that the publicity source is practically free for physicians to access, when the information about the simple type of information is abundant (which is the case for news on *lc*), we expect that physicians are able to resolve most of the uncertainty about this attribute from the publicity source. This makes the marginal gain of paying attention to sales reps about *lc* small. Therefore, we expect that $\gamma_2 > 0$. On the other hand, when most of the uncertainty can be resolved from publicity, we also expect that there would be little room for publicity to play the role of corroborative evidence. Hence, $\gamma_1 \simeq 0$. Roughly speaking, we expect that for simple types of information (*lc* or *se*), the last two terms of equation (A.7) will be approximately zero, and the only interaction term remaining is $-\frac{\gamma_2}{\sigma_{d0}^2} \cdot (n_{dj} \cdot n_{pj})$, which is negative.

For complicated types of information (e.g., *rh*), we expect that information sources are noisier and this is particularly the case for the publicity source due to the time and space constraints in the news media. Hence, it seems likely that even after accessing the information from the publicity source, there is still a lot of uncertainty remaining. This makes the marginal gain of paying attention to sales reps regarding *rh* fairly large. Thus, we expect that $\gamma_2 \simeq 0$. The messages from the detailing source can also be noisy. This gives room for the information from publicity to play the role of corroborative evidence to bolster physicians' confidence in the messages from detailing, implying that $\gamma_1 > 0$. Roughly, we expect that for complicated types of information (*rh*), the third and last terms of equation (A.7) will be approximately zero, and the only interaction term remaining is $\frac{\gamma_1}{\sigma_{d0}^2} \cdot (n_{dj} \cdot n_{pj})$, which is positive.

Finally, suppose that physicians and patients care about π_j (intuitively, this means they care about how precise the consensus information is). If we replace n_p and n_d with their goodwill stocks, then this set up will roughly boil down to the reduced form demand model we present. In particular, γ_{int} in equation (2) corresponds to $\frac{\gamma_1}{\sigma_{d0}^2}$ or $-\frac{\gamma_2}{\sigma_{d0}^2}$ in equation (A.7); γ_{detail} and $\gamma_{publicity}$ in equation (2) correspond to $\frac{1}{\sigma_{d0}^2}$ and $\frac{1}{\sigma_p^2}$ in equation (A.7), respectively.

The discussion above gives us two hypotheses: (i) for simple types of information (lc or se), we should see a negative interaction between the publicity stock in lc or se, and the detailing stock; (ii) for complicated types of information (rh), we should see a positive interaction between the publicity stock in rh and the detailing stock.

We should make three remarks. First, we can also allow the physician's utility to depend on the expected value of the consensus distribution. But note that if the signals from each information source are distributed around the true value of the attribute, $\bar{Q}_j$, we have $E[Q_j] = \bar{Q}_j$ regardless of the weights used in equation (A.3). Our brand intercepts in the reduced form model capture this.

Second, the way we model the corroborative evidence effect is related to the Quasi-Bayesian learning model proposed by Camacho et al. (2011). They introduce the idea that some information signals can be more salient under special circumstances. More specifically, they argue that when a patient needs to switch a medication in period t, his/her physician would put more weight on the information feedback signal in that period. Here, our arguments imply that when a physician sees more corroborative evidence from publicity, he would put more weight on the information signals from the detailing source.

Third, Anderson (1981) proposes an Information Integration Theory, where he argues that human beings use cognitive algebra to combine information from different sources for decision making. The cognitive algebra rules could be additive, multiplicative, etc., depending on the circumstances. The way to test which rule applies is to regress decision outcome variable on measures of information signals from different sources. If the interaction term is statistically significant, then it provides evidence for the multiplicative rule. If only the variables that measure information signals are significant, then it provides evidence for the additive rule. However, Anderson (1981) did not provide theories about when a particular cognitive rule would apply. The theoretical argument provided above can be viewed as a potential theoretical foundation for using the multiplicative rule.

B An Example of Endpoints of Landmark Clinical Trials

Table 9: Clinical Endpoints of 4S

Fatal Events
Cardiovascular Events
All Coronary Events
Definite Acute MI
Probable Acute MI
Acute MI Not Confirmed*
Instantaneous Death
Death Within 1 h
Death Within 1-24 h
Death >24 h After Onset of Event
Non-witnessed Death
Intervention-associated
Cerebrovascular Events
Other Cardiovascular Events
Non-Cardiovascular Events
Cancer
Suicide
Trauma
Other
Nonfatal Cardiovascular Events*
Coronary Events*
Any Major Coronary Events
Definite Acute MI
Definite or Probable Acute MI
Silent MI
Resuscitated Cardiac Arrest
Acute MI, Intervention-associated
Non-MI acute CHD
Acute Non-CHD Cardiac
Cerebrovascular Events*
Any Cerebrovascular Events
Stroke, Non-embolic
Stroke, Embolic
Stroke, Haemorrhagic
Stroke, Unclassified
Stroke, Intervention-associated
Transient Ischaemic Attack
Other Cardiovascular Events

*The events were not used as endpoints.