# Ask Your Doctor? Direct-to-Consumer Advertising of Pharmaceuticals

Michael Sinkinson
*The Wharton School, University of Pennsylvania*
Amanda Starc*
*The Wharton School, University of Pennsylvania and NBER*

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**Abstract**

We measure the impact of direct-to-consumer television advertising by statin manufacturers. Our identification strategy exploits shocks to local advertising markets generated by idiosyncrasies of the political advertising cycle. We find that a 10% increase in the quantity of a firm's advertising leads to a 0.76% increase in revenue, while the same increase in rival advertising leads to a 0.55% decrease in firm revenue. Results also indicate that a 10% increase in category advertising produces a 0.2% revenue increase for non-advertised drugs. Both the business-stealing and spillover effects would not be detected through OLS. Decomposition using micro data comfirms that the effect is due mostly to new customers as opposed to switching among current customers. Simulations show that an outright ban on DTCA would have modest effects on the sales of advertised drugs as well as on non-advertised drugs.

*The Wharton School, University of Pennsylvania, 3620 Locust Walk, Philadelphia PA 19104 (e-mail: msink@wharton.upenn.edu; astarc@wharton.upenn.edu). The authors would like to acknowledge the valuable advice and suggestions provided by Jason Abaluck, Patricia Danzon, Liran Einav, Brett Gordon, Wes Hartmann, Gunter Hitsch, JF Houde, Ginger Jin, Marc Meredith, Andrew Sfekas, Brad Shapiro, Ashley Swanson, and Bob Town. We would further like to thank workshop participants at Wharton, the Columbia Commuter Strategy Conference, Marketing Dynamics, IIOC, QME, and the Econometric Society for their comments and feedback. Starc gratefully acknowledges funding from the Leonard Davis Institute.

# 1 Introduction

This paper provides new causal estimates of the impact of advertising on consumers and firms using a novel identification strategy. While advertising is a ubiquitous part of life, economic theory offers few conclusions on its welfare effects (Bagwell 2007). The impact and consequences of advertising are empirical questions. Furthermore, estimation is a challenge due to endogeneity, issues in measurement, and heterogeneity across consumers. Yet advertising spending continues to grow, especially as firms expand to new platforms.[1]

Pharmaceutical companies are known for aggressively advertising their products directly to both physicians and consumers. Direct-to-consumer advertising (DTCA) of drugs accounted for over $3 billion in spending in 2012. DTCA has been controversial since the Federal Drug Administration (FDA) loosened restrictions in 1997. While the Federal Trade Commission has encouraged DTCA due to its perceived informational qualities, some in the industry are skeptical, noting that it can effectively create a wasteful arms race among competitors selling similar products. Anecdotal evidence indicates that strategic interaction among firms is an important component of direct-to-consumer advertising, with advertising often being purchased to "blunt the impact of ... competitors' ads."[2]

We identify the effectiveness of TV advertising for anti-cholesterol drugs known as statins.[3] Statins are an excellent market to examine the impact of DTCA for a number of reasons. First, there are a small number of advertised drugs - four during our sample period - allowing us to explore the importance of competitive interaction between firms. Second, the products, whether advertised or not, are close substitutes, and idiosyncratic consumer preferences are less important in this setting. Third, the products are considered effective with few side-effects. Fourth, unique variation allows us to identify the effect of both own and rival advertising. Finally, the category is economically important, generating $34 billion in sales in 2007, with substantial ad spending.

Estimating returns to advertising is challenging because firm advertising decisions are endogenous: they depend both on unobserved market characteristics and actions of rival firms. First, firms are more likely to advertise in markets where advertising is likely to be most effective, due to either a transitory or permanent demand shock. Interaction between firms also has major implications for measurement and estimation: if advertising is largely business stealing, firms be trapped in a prisoner's dilemma, where all would prefer to pre-commit to lower levels of advertising. By contrast, if advertising is characterized by large spillovers, firms may have an incentive to under-

[1] Growth in online video advertising is strong, for example. See Hoelzel (2014).

[2] Ian Spatz, formerly of Merck, has been especially critical (Spatz (2011)).

[3] This paper focuses on television advertising only, but evidence is presented that the results are not contaminated by spending in other channels, such as print or radio. Television is the primary medium for advertising in the data, accounting for over twice the spending in any other channel.

advertise. Both issues highlight the need for exogenous variation in advertising levels to measure effectiveness.

Our identification strategy exploits novel variation in advertising due to political campaigning during the 2008 national election. Idiosyncrasies of the US political process meant that in January of 2008, voters in New Hampshire, Iowa, and South Carolina saw large quantities of political ads, while in May of 2008, political advertising was concentrated in Indiana, Pennsylvania and North Carolina. In the months leading up to the general election, advertising was heaviest in "swing states" in the presidential contest, and where House and Senate races were most competitive.[4] We show that political advertising displaces pharmaceutical advertising. Our first-stage estimates imply that the thousands of political ads aired through the election cycle had a significant, negative effect on the level of DTCA.

Graphical analyses show that political primaries are associated with statistically significant reductions in drug sales using market-month-drug level usage data from Truven Medstat. Regression results show an own-advertising elasticity of revenue with respect to the quantity of ads of .0764 for a sample of privately insurer consumers. We also provide estimates of revenue elasticities with respect to rival advertising: here, we estimate an elasticity of -.0548. We separately estimate the impact on non-advertised branded and generic drugs and estimate an elasticity with respect to branded advertising of 0.02. Therefore, advertising has both a business-stealing effect among branded, advertised drugs, but a spillover effect to non-advertised drugs.

Elasticities are similar in a sample of Medicare Part D beneficiaries, and we cannot reject that our elasticities are the same across samples. We also examine heterogeneity across different subsets of consumers in the Part D sample. When we restrict the sample to first-time consumers, we estimate much larger elasticities: the effectiveness of advertising is largest for new consumers who have no history of statin use. Both data sets tell a consistent story: DTCA has an economically important impact on drug sales. Competitive interaction between rivals is an important feature of the market, and rival advertising can have a significant business-stealing effect among some drugs, while having a beneficial effect on others.

We use our estimates in a number of policy simulations. First, we show that the estimated business-stealing effect is economically meaningful: revenue for branded advertised drugs would 21-24% higher absent the effect of rival advertising. Second, banning DTCA harms sales unadvertised drugs. For advertised drugs, the net effect of eliminating both the positive and negative effects of advertising is a modest 2.6% reduction in quantity for Lipitor and only a 1% reduction for Crestor.

[4]While the list of swing states varies from election to election and there is no clear definition, Politico determined that the 2008 presidential race was most competitive in Colorado, Florida, Indiana, Missouri, Nevada, New Hampshire, New Mexico, North Carolina, Ohio, Pennsylvania, and Virginia. Source: http://www.politico.com/convention/swing state.html

While we believe our paper is the first to exploit this form of political advertising as an instrument, we build on a substantial literature examining the impact of DTCA.[5] Previous researchers have found significant evidence for the market-expanding or spillover effects of DTCA on outcomes such as doctors visits, drug sales, and drug adherence (Berndt 2005, Jin and Iizuka 2005, Wosinska 2002, Wosinska 2005, Rosenthal et al. 2003, Berndt et al. 1995). The paper closest to our study is Shapiro (2014), which estimates economically significant spillover effects in the anti-depressant market using a cross-border strategy and structural model of demand. Our paper is consistent with these previous studies, while finding an additional, economically important role for business stealing in the statin market. This paper also contributes to a literature that attempts to measure the causal impact of advertising. Recent work (Lewis and Rao 2013, Blake, Nosko and Tadelis 2013) has utilized randomized experiments on online platforms. Similar to these studies and work by Ackerberg 2001, our natural experiment finds heterogeneity in the effect of advertising in a setting with plausibly exogenous variation in advertising levels. While our focus is on the statin market, the identification strategy we propose is likely to be useful in many other product markets.

The paper is organized as follows. Section 2 describes the market and setting. Section 3 presents of model of strategic interaction and simulation results. Section 4 describes the data and empirical strategy, while Section 5 presents results. We perform robustness checks and explore heterogeneity in the main results in Section 6. Section 7 details simulations, and Section 8 concludes.

# 2 Setting

Cholesterol is a waxy substance that is both created by the body and found in food. Low-density lipoprotein (LDL, or "bad" cholesterol) is associated with a higher risk of heart attack and stroke. While cholesterol can usually be well controlled with diet and exercise, drug therapy can also be effective. A large class of drugs - statins - work by preventing the synthesis of cholesterol in the liver. Statins are big business: each year during our sample period, Lipitor and Crestor alone had nearly $15 billion in combined sales. The first statin on the market was Mevacor, which was introduced in 1987 by Merck. Mevacor was followed by a large number of “me-too” drugs: similar, but chemically distinct, compounds with the same mechanism of action. Zocor was introduced by Merck in 1991, as was Pravachol.

Between 2007 and 2008, four branded anti-cholesterol medications were being advertised. The two largest advertisers were Lipitor (Pfizer), approved in 1997, and Crestor (AstraZeneca),

[5] A recent literature has examined the effect of political advertising in political campaigns and explore supply side competition. (see Gordon and Hartmann 2013 and Gordon and Hartmann 2014).

approved in 2003. According to trade press and news, the introduction of Lipitor heralded an increase in the "'arms race' of drug marketing."[6] The FDA clarified its stance on DTCA in 1997; Pfizer marketed Lipitor to consumers aggressively beginning in 2001. Zocor's patent expired in 2006, and heavy generic competition began shortly thereafter. This hurt the sales of not only Zocor, but also Crestor and, to a lesser extent, Lipitor, as cheaper generic substitutes flooded the market and Zocor gave aggressive rebates to insurers to keep consumers taking their product. Prescription drugs without patent protection are rarely advertised by their manufacturers.[7] Lipitor's patent expired at the end of November 2011 and Crestor's is scheduled to expire in 2016.

Manufacturer strategies for differentiating their products often rely on results from clinical trials showing efficacy. Zocor marked an early use of clinical trials in marketing drugs (largely to physicians): Merck showed in the Scandinavian Simvastatin Survival Study (4S) that Zocor prevented additional heart attacks among patients who had already suffered a heart attack. In April 2008, Merck released the results of the ECLIPSE trial, which favored Crestor relative to Lipitor for some sub-populations of patients,[8] corresponding to the increase in Crestor marketing.

Two issues affected the marketing of statins during our sample period. The ENHANCE trial results led to the end of advertising of Vytorin and Zetia in 2008.[9] The study, completed in 2006[10], showed that Vytorin (Zetia and Zocor combined) was no better than Zocor alone. The American Academy of Cardiologists recommended that doctors no longer prescribe Vytorin and strongly discouraging the use of Zetia.[11] The effect on Vytorin's market share was dramatic, falling 10% immediately and 40% over the course of 2008 in our sample data.[12] The second came in April of 2008, when Lipitor halted its advertising campaign featuring Dr. Robert Jarvik (developer of the Jarvik artificial heart). Many, including Congress, had concluded that the advertisements were misleading.[13] As a result, Crestor was the only statin airing DTC TV spots from April 2008 until August 2008. In 2008, Lipitor's sales fell by 2% and Crestor's sales rose by nearly 29%.[14]

Statins are widely covered by insurance plans. Most consumers with employer-sponsored

[6] For a more complete historical narrative, see Jack (2009) and **?**. While initially, Pfizer priced aggressively and detailed heavily, they eventually turned to DTCA as a way to expand the market and gain market share.

[7] This is in contrast to over-the-counter medications, which are often advertised even through an exact molecular substitute is available. See Bronnenberg et al. (2014) for details.

[8] They use the results of this trial in marketing. See, for example, http://www.crestor.com/c/about-crestor/crestor-clinical-studies.aspx, and Faergeman et al. (2008) for the clinical trial results.

[9] Congress specifically sent a letter to the FDA to challenge marketing of Vytorin (Mathews (2008)).

[10] Greenland and Lloyd-Jones (2008)

[11] Davidson and Robinson (2007)

[12] By contrast, a recent, much larger study (18,000 subjects vs. just 750) found Vytorin to be more effective than Simvastatin alone. See Kolata (2014) for news coverage and Blazing et al. (2014) for study design. We do not take a strong stand on the role of these studies except to point out that the findings are often referenced in DTCA and this advertising, in addition to the information content of the studies themselves, may affect demand.

[13] Dr. Jarvik was not a licensed cardiologist and was replaced by a stunt double in some of the TV spots.

[14] See **?**.

health insurance have prescription drug coverage as part of their benefits package.[15] Insurance coverage is usually generous, and consumers will face only a fraction of a branded statin's $3/day price tag. Consumers in employer-sponsored insurance tend to have a limited number of choices (Dafny, Ho and Varela (2013)) and are unlikely to select into insurance plans based on their coverage or cost sharing for particular drugs. By contrast, most seniors obtain their drug coverage through the Medicare Part D program. Consumers in Medicare Part D face a very non-linear insurance contract: there is an initial deductible, followed by (an average of) 25% co-payment rates up to an initial coverage limit. Once a consumer hits the initial coverage limit, they must pay for all of their expenditure in the "donut hole" or coverage gap until they meet a catastrophic cap. The donut hole is now closing due to the Patient Protection and Affordable Care Act (ACA), but this basic structure would have been in plan during our sample period. There are many plans available to most consumers and these plans are likely to vary substantially in terms of their formularies, that is, the specific drugs covered by the plan. A savvy consumer will choose a plan based on their expected drug demand over the course of the year. Meanwhile, insurers have incentives to steer consumers to lower cost drugs and manufacturers provide rebates to plans in exchange for preferred positioning on formularies. This has led to lower prices for branded drugs (Duggan and Morton (2010)). Therefore, plan selection and copay structure are more likely to be a concern in the Medicare Part D setting.

Finally, to obtain a statin, a patient must have a prescription. Manufacturers advertise their products to physicians, through detailing, as well as directly to consumers. Physicians and consumers may disagree about the best course of treatment, and asymmetric information creates the potential for physician agency to be an important feature of prescription drug markets. Prescription drug manufacturers, aware of the influence of physicians, engage in substantial detailing at the doctor level in addition to DTCA (known as "push" techniques, as opposed to "pull" techniques that target the consumer). Both plan selection and physician agency are outside the scope of this paper. While they influence the market, their effects are likely to remain fixed over our short time period, allowing us to focus on measuring the impact of DTCA.

# 3 Firm Advertising Decisions and Estimation Bias

The direction of bias in OLS estimates is not obvious in the context of firm advertising decisions. Consider a static, simultaneous move advertising game among two single-product firms with demand for drugs $j \in 1,2$ given by

$$D_j(a_j, a_{-j}, \xi),$$

[15] This insurance coverage may be provided by the consumer's health insurer of by a pharmacy benefits manager.

where $a_j$ is firm $j$'s advertising level and $a_{-j}$ is rival advertising. While we assume this game takes place across many markets, we will supress market notation. The vector $\xi$ is a set of shocks to demand for each good, $\xi = \{\xi_1, \xi_2\}$. The per-unit cost of advertising is $c$, and profit per unit sold is $\rho$.

In equilibrium, firms choose $a_j$ such that the marginal benefit of advertising equals its marginal cost. Firms observe their demand shock when choosing their advertising. The econometrician observes the realized $D_j$ and the chosen $a_j$ for all firms across many markets and over time, but never the vector $\xi$.[16]

The econometrician observes many outcomes from this game and estimates the demand elasticity of own and rival advertising, using a specification such as

$$ln(D_j) = \alpha + \beta_1 ln(1 + a_j) + \beta_2 ln(1 + a_{-j}) + \varepsilon_j. \quad (1)$$

Because the demand shock $\xi$ is unobserved to the econometrician, OLS estimates suffer from omitted variables bias.[17]

Advertising levels depend on consumer responsiveness to ads, which will depend on the functional form and parameters of the demand system.[18] In the case of a single firm advertising (so that $a_{-j} = 0$ for that firm), optimal advertising choices that create a positive correlation between demand shocks and advertising lead to overstated impacts in OLS estimates. By contrast, a negative correlation between demand shocks and advertising leads to downward bias in OLS estimates.

In the case of multiple firms advertising, the levels of $a$ are equilibrium objects of a game, where a firm's best response to rival advertising may be to either increase or decrease its own advertising. Consider an example: Lipitor has a positive demand shock in a market, which increases their return to advertising. Lipitor's heightened advertising increases Crestor's return from advertising, so both firms advertise at high levels. This would create positive correlation between Crestor ads and positive demand shocks for Lipitor. Such correlation would lead the econometrician to conclude that Crestor advertising has a spillover effect on Lipitor, when that is not the case. The strategic interactions among firms can lead to correlations between advertising levels and unobservables that result in upward or downward bias in OLS estimates.

From a welfare perspective, understanding the forces that shape equilibrium outcomes is crit-

[16] Appendix A lists regularity assumptions for the analysis that follows.

[17] It is common to think of the shock as positive in the sense that $\frac{\partial D_j}{\partial \xi_j} > 0$ and rival shocks as negative $\frac{\partial D_j}{\partial \xi_j} < 0$ , as we will do here.In the Labor literature, it is also typically the case that this heterogeneity is positively correlated with the input of interest, e.g. $\frac{\partial a_j}{\xi_j} > 0$, such as in the returns to schooling literature, although this need not be the case in general.

[18] Returns to advertising need not be linear and may depend on relative market shares. For example, in the empirical application in Dubé, Hitsch and Manchanda 2005, the authors assume thresholds and diminishing returns to advertising.

ical. If advertising generates spillovers, we would expect it to be undersupplied in equilibrium relative to the social optimum: the advertising firm cannot capture all of the surplus generated. Similarly, if advertising is about business-stealing, it would be oversupplied, as private firms do not account for the negative effect it has on rivals. This latter case is an example of a prisoner's dilemma where both firms would prefer to commit to lower levels of advertising, while in the former case both firms would do best to have a joint marketing agreement.[19]

## 3.1 Model Simulation

We simulate a Logit formulation of the above setting to explore estimation bias. Our formulation has the following utility functions in each simulated market $m$

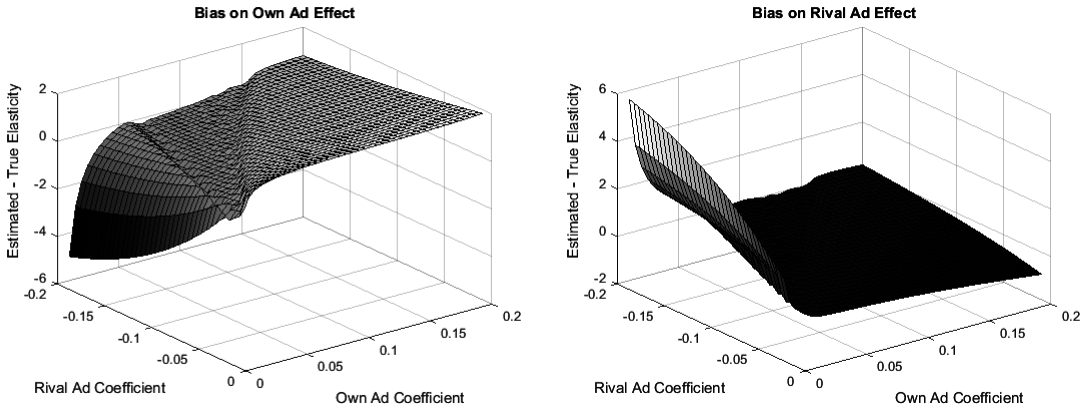
$$\begin{aligned} u_{ijm} &= \alpha_j + \beta_1 ln(1+a_{jm}) + \beta_2 ln(1+a_{\_jm}) + \xi_{jm} + \varepsilon_{ijm} \\ u_{i0m} &= \varepsilon_{i0m}, \end{aligned}$$

where $u_{i0}$ denotes the utility of the outside good. Assuming $\varepsilon_{ijm}$ is i.i.d. type I extreme value, market shares $D_{jm}$ can be computed given parameters and advertising levels using the standard Logit formula. Firm profits in this model are given by $\pi_{jm} = \rho D_{jm} - c a_{jm}$. We solve for advertising levels in each market such that both firms' first-order conditions are satisfied and create a dataset containing demand and advertising data. We then estimate equation 1, and compare the estimated elasticity with respect to own and rival advertising with analytic values (full details are in Appendix A).

We simulate 200 markets and optimal advertising decisions for both firms at a range of parameter values for $\beta_1$ and $\beta_2$. Plots below show the difference between estimated and analytic elasticities. The level of the surface indicates the bias in different areas of the parameter space: it is apparent that there can be upward (greater than zero) or downward (less than zero) bias in both own and rival advertising elasticities. In no simulation were own and rival elasticities both estimated with less than 5% bias. Table 11 shows estimates and standard errors for a particular set of parameter values.

[19] This is nicely illustrated in the market for antidepressants by Shapiro 2014.

**Figure 1:** Simulations of OLS Estimate Bias




# 4 Data and Empirical Strategy

## 4.1 Identification Strategy

We exploit shocks from political advertising in markets over time. These shocks are a result of the staggered nature of the party nomination processes and variation in competitiveness of different races in the general election. The United States holds quadrennial general elections for the presidency, which coincide with elections for all seats of the House of Representatives, numerous state governors, and approximately one-third of seats in the Senate. The election is held on the Tuesday following the first Monday of the month of November in the election year. Presidential campaigns begin well over a year before the general election as candidates seek their party's nomination, which is conferred by delegates voting at each party's national convention. Individual states and state political parties determine the timing and format of the contest to determine the state's delegation to each party's national convention, with the majority of states using government-run primary elections, and the remainder using party-run caucuses. The staggered nature of the primaries increases the national attention on and importance of early contests in Iowa and New Hampshire, as well as South Carolina, Florida and Nevada.[20] In 2008, the Democratic party contest between Hillary Clinton and Barack Obama extended into June, while John McCain secured the Republican nomination by March of 2008. Figure 2 highlights the staggered nature of the process by showing political ad concentrations for January to June, 2008.

During the general election, the "winner take all" nature of the Electoral College means that

[20]New Hampshire law stipulates that no other state can have a primary earlier: "The presidential primary election shall be held on the second Tuesday in March or on a date selected by the secretary of state which is seven days or more immediately preceding the date on which any other state shall hold a similar election, whichever is earlier, of each year when a president of the United States is to be elected or the year previous." NH RSA 653:9

political advertising in swing states is likely to be far more valuable than in "safe states", leading to large variations in the numbers of ads different markets are exposed to (Gordon and Hartmann, 2013). For example, in October of 2008, New York, NY had 0 television ads for presidential candidates (547 for Governor/House/Senate candidates), while Cleveland, OH had 8,073 television ads for presidential candidates (and another 2,439 for Governor/House/Senate candidates). Political campaigns and outside influence groups often purchase premium advertising slots that can pre-empt previously purchased advertising.[21]

While political advertising provides useful variation that allows us to identify the effect of advertising, we are interested in both the effect of the focal firm's advertising and their rival's advertising. To separately identify the two effects, we use an additional shock specific to the statin market. As discussed above, Pfizer was forced to halt its consumer advertising in mid-2008. In order to separately identify the effect of own and rival advertising, we interact the political advertising instrument with the timing of this regulatory action. We assume that the relative impact of this shock across markets is uncorrelated with drug demand.

## 4.2 Data

We combine two sources of advertising data. First, data from Kantar Media contain both the number of ads and the level of spending for 2007-2008 at the month-drug level for every DMA in the United States. We also have a record of every political ad (presidential, senate, house, and gubernatorial) aired during the 2007-2008 election cycle in every DMA from the Wisconsin Advertising Project, which we normalize to a 30-second length and aggregate into monthly figures.

The number of political ads in a market-month varies widely during the Jan 2007-Nov 2008 time period: half of the month-market observations during this period have zero ads, while some markets have over 20,000 political ads in a month (i.e. Denver, CO in October of 2008). Figure 2 shows the progression of the political ad shocks for the first six months of 2008, where each DMA is represented by a circle sized proportionally to the number of political ads. The mean number of monthly ads by market from Jan 2007 to Nov 2008 is 535, with a standard deviation of 1600. By contrast, there are fewer drug ads in general: when combining national ads with local ads, the average number of statin ads aired in a market during a month is 98 with a standard deviation of 59. (National and local refer to the level of the ad buy, not the content.) Figure 3 shows the levels of monthly national ads for the advertised statins during our sample period, while Figure 4 shows the highest number of monthly local ads for each of the drugs (the minimum is always zero). Local

[21] See the discussion in Gordon and Hartmann (2014) regarding how political campaigns purchase advertisements. As they discuss, campaigns and issue advocacy groups purchase premium "non-preemptible" advertising slots, which can displace advertising previously ordered at the lowest unit rate. This is in spite of laws that guarantee low rates to political campaigns. This is also explored in Moshary (2014), whose author examines differential pricing among political action committees (PACs). She further argues that LUR regulation may lead stations to withhold some slots.

advertising can be a substantial portion of a firm's total advertising. While some markets receive no additional advertising, the maximum amount of local advertising is often higher than the national advertising, indicating that a substantial proportion of advertising comes from local ads.

We combine this advertising data with prescription drug usage and revenue data from two sources. First, we used Truven MarketScan data, which draws from a convenience sample of large, self-insured firms. These data represent individuals enrolled in traditional, employer-sponsored insurance, and are likely to be the primary target market of statin advertisers. Our sample consists of market-level aggregated revenues, quantities, and covered lives.[22] Summary statistics for the data sources are shown in Table 1. We utilize data covering 189 DMAs and 17 months, spanning July of 2007-November of 2008. The sample is younger than the population on the whole, and a relatively small proportion of this population takes statins. The largest branded drug captures just less than 5% of the total market.

We supplement this data with data from the Medicare Part D program. Our data represent a 10% random sample of all Medicare Part D beneficiaries. This data allows for tracking of individual consumers. However, many consumers who ever take a statin begin before they reach Medicare eligibility, and plan selection and copay structure are larger concerns. We restrict our sample to the same 189 DMAs, 17 months, and four drugs in the Truven data. We then aggregate the data to the product-month-DMA level and perform a parallel analysis. The combination of data sets allows us to explore heterogeneity in the effectiveness of DTCA and provides additional confidence in the magnitude of our empirical results.

To test for covariate balance, we utilize the Part D data. For simplicity, we split the sample into markets that experience more or less than the median level of political ads during our entire sample period. Table 2 provides summary statistics; the unit of observation is the DMA. We consider age, gender, and race as well as mortality rates (a crude measure of health) and dual eligible status (a crude measure of poverty). None of the differences between the two groups are statistically different with the exception of % dual eligible. Consumers in markets with less political ads seem to be slightly poorer; if anything, income effects would only increase drug demand in above median markets assuming prescription drugs are a normal good.

# 5 Results

## 5.1 First-Stage Results

Political advertising is plausibly exogenous: the political primary and caucus schedule is set independently of any prescription drug market factors and the competitiveness of specific races is

[22] We aggregate MSAs to DMAs to arrive at our analysis data set.

unlikely to be correlated with the market for statins. We next demonstrate that the level of political advertising predicts drug advertising. Figure 5 shows a binned scatter plot highlighting the relationship between political advertising and statin advertising, where observations are de-meaned by market and drug-year-month, and then binned to create a scatter plot of the data. There is a strong negative correlation between the two series.

Table 3 presents a regression of the log of the number of statin advertisements for a drug in a market on the log of the number of political advertisements (in 1000s). The level of observation is a DMA-month for January 2007 until November 2008. We include a variety of fixed effects across different specifications. The OLS results show that a 10% increase in political advertising leads to a 1.2% decrease in statin advertising. The effect is slightly larger if you do not account for month fixed effects. To account for the fact that drug ads cannot be negative, the last columns of Table 3 estimates a Tobit model. We find a significantly larger effect: the elasticity of an individual drug's ads with respect to political ads in a market is -0.2598 in our preferred specification, implying that a 10% increase in political ads decreases each drug's ads by 2.6%. Appendix Table 14 shows the analogous results using levels instead of logs, with all results strongly negative and significant.

We address four possible concerns about this strategy. First, since the political cycle is known in advance, firms could have substituted ads to months before or after a market received a large number of political ads. In Table, 13 we show that leads and lags of political advertising are not predictive of drug ads in the current month, indicating that there was not substitution to earlier or later months. Second, firms may substitute from TV advertising to other local media (radio, newspaper) when political ads displace television advertising. In Table 12, we show that total local spending is not affected by political ads once local TV ads are controlled for.[23] Third, firms may modify their detailing plans due to the displacement of their local TV ads by political ads. While we do not have data to directly test for this, discussions with industry managers led us to conclude that this is infeasible, as detailing plans are set at the annual level and cannot be quickly scaled up or down at the market level. Finally, we do not believe that drug firms are responding to political advertising shocks by buying more advertising in less desirable time slots, which would create measurement error in the number of ads in our data. The relationship between political advertising and drug advertising is largely driven by availability and pre-emption as opposed to prices. The Communications Act of 1934 limits media outlets from raising prices for political campaigns, and Table 15 shows that the price per impression is not changed significantly due to political advertising. If firms were shifting their ad buys to lower-priced slots with lower ratings, we would expect a negative relationship.

[23]The results appear to show that local TV ads and other local media are complements, not substitutes, and is consistent with the political cycle being a shock to all forms of media in a local market.

## 5.2 Graphical Evidence

First, we present a number of simple graphical analyses. We initially focus on unadvertised drugs, for which there is only one causal effect to estimate. During the time leading up to a primary, consumers are exposed to fewer ads for Crestor, Lipitor, Vytorin, or Zetia. If these ads have spillover effects on unadvertised (often generic) drugs, we would expect a drop in sales at the time of the primaries. The timing of primaries is staggered, giving a simple test of the effect. Figure 6 shows the effect of primaries on market share growth for unadvertised drugs. While sales are stable in the months before the primary, there is a statistically significant reduction in sales growth concurrent to the primary. We argue that the natural mechanism for this reduction is a drop in statin advertising. Appendix Figure 10 shows a placebo test where we artificially move primaries to 2009 and find no effect.

We are also interested in the effect on branded drugs. Here, the competitive interaction makes interpretation more difficult. While the political process displaces Lipitor ads, it displaces Crestor ads as well, and we will only be able to measure the net effect without additional variation or assumptions. However, some primaries take place during the months in which Lipitor was not advertising due to regulation. Given this additional fact, we would expect the direct effect of the primary to be larger for Crestor than for Lipitor. That is exactly what we see in Figure 7; the magnitude of the effect of primaries on Crestor sales is nearly twice as large as the effect on Lipitor sales. During this time period, Crestor and Lipitor are the primary advertisers. The overall effect is negative: the effect of a firm's advertising is not outweighed by its rival's advertising. Furthermore, these results imply that the absence of DTCA would lead to a drop in overall drug sales.

## 5.3 Regression Results

We utilize the identifying variation in a regression framework to estimate elasticities. We estimate the following equation:

$$log(revenue_{jtm}) = \beta_0 + \beta_1 log(1 + ad_{jtm}) + \beta_2 log(1 + \sum_{k \neq j} ad_{ktm}) + \beta_3 X + \varepsilon_{jtm},$$

where $X$ represents a vector of covariates. In all specifications, we include product and market fixed effects. We control for time trends in product demand in two ways: drug-year fixed effects and a drug-specific time trend. Figure 9 shows that there are important time trends during our sample period: Lipitor's market share is steadily decreasing over the entire sample period. There does not seem to be a distinct, national break at the time that Pfizer pulled the Jarvik ads. At the same time, Crestor's market share is increasing, though not as dramatically. Because we will eventually utilize the regulatory shock to Lipitor advertising, we cannot allow for finer (monthly- or

quarterly-) product-specific fixed effects. However, we can can allow for a linear, product-specific time trend that approximates the data reasonably well. In 18, we show that higher order, drug-specific time trends have a negliable effect on the estimates. Because the specification is log-log, we can interpret the coefficients as elasticities.

Table 4 shows the results of OLS specifications for advertised drugs. The first pair of columns use contemporaneous ads and revenues; the next pair regresses this month's revenue on the averages of this month's and the previous month's advertising levels; the final pair average the previous three months' advertising levels. Previous research has shown that advertising can be cumulative and/or have a lagged effect (Dubé, Hitsch and Manchanda 2005), but that the effects of DTCA can depreciate quickly (Jin and Iizuka (2005)). In each regression, the level of analysis is the DMA-month-drug. We include each of the drugs advertised during our sample period from July 2007 through November 2008 that are classified in the same in Truven Redbook class 059: Lipitor, Crestor, Vytorin, and Zetia. The dependent variable is logged drug revenue per insured individual in the market. Regardless of controls, the OLS regressions consistently show a small, but statistically significant and positive effect of DTCA on sales. The specifications that allow for a product specific time trend are typically smaller in magnitude.

We document the causal impact of advertising in Table 5. We instrument own and rival advertising levels using (i) the level of political ads, as well as second- and third-order polynomials of political ads, (ii) a dummy for the congressional action that halted Lipitor advertising, and (iii) an interaction of this dummy with the polynomials of political advertising. Our instruments are remarkably strong predictors of own and rival advertising. The F-statistic for a test of joint signification of the excluded instruments in the first stage of our main specifications is 493.66 for own advertising and 67.30 for rival advertising.

Based on the results in the previous table, the OLS analysis underestimates the effects of own and rival advertising. The own advertising effect in column 4 (.0064) is less than 10% of the effect measured in the IV specification (.0764). Similarly, we find substantial evidence of business stealing in the IV specifications that is absent from the OLS results. As discussed in Section 3, the direction of OLS bias is ambiguous, but in this case it appears that the strategic interaction between firms leads to the effect of own advertising being biased downward, while the effect of rival advertising is biased upward.[24]

Unsurprisingly, we find that the effects are attenuated as we look at a broader window. The effect of contemporaneous advertising in the drug-year fixed effects regression is the largest (0.0808), while the two-month (0.0764) and three-month (0.0536) moving averages are smaller. Despite this

[24]One other possible explanation for the bias we find is that measurement error could be attenuating the OLS estimates. Alternatively, we measure a local average treatment effect that captures the short run elasticity of sales with respect to advertising expenditures and the long run elasticity may be smaller in magnitude.

attenuation, the results are stable across specifications. While the estimates that allow for product-specific time trends are smaller in magnitude, we cannot reject that they are statistically the same. We focus on the two-month trailing average specification with drug specific time trends moving forward. Our preferred own-revenue elasticity estimates for advertised drugs is 0.0764, from column 5. This implies that a 10% increase in advertising would yield a 0.76% increase in revenue. Our preferred cross-revenue elasticity estimate is -0.055. Appendix Table 18 shows similar results if the outcome of interest is quantity (market share) instead of revenue. These results are consistent with a model in which advertising is business-stealing and may create an arms race.

Table 6 estimates the spillover effects for unadvertised drugs. Columns 3 and 4 replicate the last set of specifications in Table 5 in which two-month moving averages of drug advertising are the independent variables of interest. However, columns 1 and 2 present OLS and IV specifications in which logged revenue of unadvertised antihyperlipidemia drugs (as classified by Redbook) is the dependent variable. In the OLS specifications, we find no effect of rival advertising. Once we instrument for advertising, we find evidence that advertising has a small, but significant spillover effect. A 10% increase in advertising for the class leads to a 0.23% increase in sales of unadvertised drugs. Our results support a model in which advertising has largely persuasive or business-stealing effects, but also spillovers to unadvertised drugs, consistent with informational effects.

# 6 Robustness Checks and Heterogeneity

## 6.1 Robustness Checks

We perform a number of additional analyses and robustness checks. We present three key sets of specifications here. First, in Table 17, we explore the direction of the bias in OLS results. We argue that strategic interaction is an important determinant of returns to advertising. To test this, we run two specifications in which we omit the effect of rival ads. The results are in Columns 1 and 3. These specifications explicitly violate our exclusion restriction: shocks to political advertising affect drug sales not only through changes in my own advertising, but changes in my rival's advertising as well. Therefore, we do not interpret these estimates as causal. When we do not control for rival advertising, the estimated own-advertising elasticity is much smaller (0.0163), less than 15% of the effect measured in columns 2. Both my advertising and my rivals' advertising are endogenous and the outcome of dynamic game; our identification strategy allows us to capture both effects.

Table 18 shows that our results are robust to the different assumptions about the timing of advertising effectiveness. Column 2 presents a specification where advertising is measured with a one-month lag, rather than a two-month trailing average. The estimates are quite similar, though

larger in magnitude and closer to the contemporaneous estimates in column 2 of Table 5. The specification in column 1 controls for the fact that advertising stock might also have an effect on drug revenues by including a one-year lag of advertising as a control. We obtain statistically indistinguishable estimates as compared to our preferred specification.

Finally, our identification strategy exploits both the timing of the political process and the pulling of Lipitor ads featuring Dr. Robert Jarvik. We have more confidence in the first source of identification; it is possible that the pulling of Lipitor ads also led to numerous news stories and this publicity, while it contained no content about the quality of the drug itself, may have had an impact on sales. However, in the third column of 18, we still interact the regulatory action with the level of political advertising and utilize the "intensity of treatment" across areas as a second instrument, while omitting the main effect from both stages. We are comparing those states where a primary would have had a large impact on Lipitor ads if not for the regulatory action with those states where a primary affects all drugs more equally. We also run an additional specification that includes the main effect of the Jarvik regulatory action and interactions in both stages of the regression and present the results in column 4 Table 18. The estimates are noisier, but confirm our basic story. The own advertising elasticity in both of these specifications is larger in magnitude than our main results, but not statistically different.

## 6.2 Part D Sample

These results are compelling, but the Truven MarketScan data represent only a fraction of the potential statin market. While there is no reason to believe the consumers in the Truven MarketScan are not representative of employees of large, self-insured firms, the sample is not representative of the population as a whole. In order to further explore the effect of DTCA, we utilize Medicare Part D claims data. Medicare Part D covers a population that is significantly older and sicker than the Truven MarketScan data. Furthermore, the contractual features of plans do more to alter utilization or steer consumers towards particular drugs. This analysis gives us an opportunity to compare elasticities across settings and explore additional heterogeneity in the data.

In all our specifications, we aggregate the Part D claims data, which are individual-prescription fill level observations, to the DMA-product-month level. We keep only those markets for which we have Truven data, leaving us with the same number of observations in each specification and identical first stage regressions. Any differences in the estimates are due to differences in relative sales across the two samples.

Table 16 shows the results of OLS specifications for advertised drugs. The results are remarkably similar in magnitude to the estimates in Table 4, though slightly larger. The differences between the estimates are rarely statistically significant. In the IV regressions in Table 7, the own

advertising elasticities range from 0.054-0.147. while the estimates from the employer-sponsored sample ranged from 0.076-0.125. In both samples, we see significant evidence of business stealing effects, though the (negative) effect of rival advertising is smaller in magnitude than the (positive) effect of own advertising. The estimates for the Part D sample are more precise; we cannot reject that the estimated elasticities are the same. Replicating our main results in this sample provides additional confidence in both the qualitative pattern and empirical magnitudes.

The Part D data also allows us to explore heterogeneity in the effect of DTCA across different demographic groups, utilization patterns, and insurance regimes. Of primary interest is whether these effects are driven by new consumers, with no history of statin use, or by switchers, who may be more likely to try an alternative statin after seeing an ad. In order to quantify the separate effects on consumers without a history of statin use, we focus on revenue from new prescriptions. We restrict the claims data to first time prescriptions, defined by the first fill of Crestor, Lipitor, Vytorin, or Zetia. We then collapse the data to the market-month-product level and replicate the same analysis. We have slightly fewer observations as we do not observe "new" prescriptions in every DMA-month-product cell. Otherwise, the specifications are the same as previous specifications but utilize a different dependent variable.

The results are presented in Table 8. We report specifications with product specific time trends. There are two key observations. First, the own advertising elasticity is five times as large in magnitude for new consumers (0.288 versus 0.054 for the entire sample). Second, the rival elasticities are larger in magnitude among new consumers as well (0.149 vs. 0.0401 for the entire sample). We conclude that the effect is largely being driven by new consumers, rather than switchers. This has important implications for firm strategy, which we hope to explore in future research.

We also explore additional dimensions of heterogeneity in the appendix. Medicare Part D has four phases, corresponding to total spending. What phase a consumer ends the year in reflects both the marginal cost of drugs to the consumers and their relative utilization. We show that advertising does not have a significant effect on consumption for the sickest consumers - those who end the year in the "catastrophic coverage" phase of Medicare Part D.[25] Finally, consumers can choose between two alternative types of insurance plans under the Part D program. They can enroll in a stand-alone Part D plan, or a comprehensive Medicare Advantage (MA) plan. The MA plans tend to have more supply-side restrictions, and advertising has a smaller impact among these enrollees.

[25]The results do not seem to depend on effective marginal prices: the results for the initial coverage phase, in which the consumer has a relatively small co-pay, and the donut hole, where they have effectively no coverage, are statistically the same. This is consistent with previous research that argues that consumers tend to respond to the spot price rather than the true marginal price (Aron-Dine et al. (2014),Abaluck, Gruber and Swanson (2013), Dalton, Gowrisankaran and Town (2014), Einav, Finkelstein and Schrimpf (2014)).

# 7 Implications and Discussion

A back-of-envelope calculation shows that our estimates are quite sensible.[26] Lipitor spent $175M on DTCA in 2009, or $15M a month. US revenue was approximately $490M/month, and their financial statements indicate that costs were 25% of revenue. Our elasticity estimates are 0.0764 and 0.0543 for the Truven and Part D samples, respectively. This implies that a 1% increase in advertising ($150,000) increases revenues net of costs by $200,000-$281,000. While this does not exactly equate marginal costs and marginal revenues, it does hold fixed rival advertising, and so is a partial elasticity. Furthermore, the OLS estimates would imply an increase of revenue net of costs by $75,000 assuming our *largest* estimates. The OLS estimates imply marginal revenue far below marginal cost, or that firms are not maximizing profits.

## 7.1 Simulations

Our results can be used to quantify the magnitudes of business-stealing and spillovers in this market. In all simulations below, we bootstrap by re-sampling the data set 100 times (with replacement), re-estimate our main specifications, and then compute a simulated object such as the change in revenue or quantity. We report the mean of the bootstrapped results, as well as the 95% confidence interval.

First, we calculate sales of advertised drugs in the absence of a business-stealing effect of competitor advertising. To do this, we set the coefficient on rival ads equal to zero in the main specification (column 4 of 5) and calculate the percentage change in revenue. We do not alter the level of the ads themselves. This is important for two reasons. First, firms still benefit from the content of their own advertising. Second, we are not measuring an equilibrium outcome; firms may choose higher or lower levels of advertising absent a business-stealing effect.

Table 9 presents the results. Panel A shows that business-stealing has a sizable impact on revenues. Absent the negative impact of rival ads, sales would be 23% higher for Lipitor and 21% higher for Crestor over the sample period.[27] To the extent that business-stealing is less likely to be seen as welfare-enhancing, this has important implications for policy. This also suggests that DTCA creates a prisoners' dilemma, where an individual firm has a strong incentive to advertise, but in equilibrium, all are spending more on advertising and seeing minimal effects. Panel B performs the same simulation for non-advertised drugs, which effectively eliminates spillovers from other drug advertising. In the absence of such spillovers, revenues for unadvertised drugs

[26] In this calculation, we assume that prices are constant, so that an increase in spending is equivalent to an increase in the number of ads.

[27] This is a substantial increase, but not unreasonable given our estimates and the data. At $90 for a month's supply, this amounts to approximately 250 more monthly prescriptions in the average market.

would fall by 9.7%. This indicates a potentially large role for welfare-enhancing spillovers in drug advertising.

We can also quantify the impact that the political process's shock had on drug firm revenues. We first predict what advertising levels would have been in the absence of any political ads, and then use our main results to predict revenues in the absence of political ads. Panel A shows that if the political process had not displaced drug advertising, revenues for Crestor and Lipitor would have been roughly two percent higher over the study period.

Finally, we analyze the impact of changes in the regulatory environment: a ban on DTCA. This eliminates both the effect of a firm's own ads and their rival's ads. The FDA is unlikely to be concerned about firm revenues, and so the outcome of interest is the quantity (share) of consumers taking a particular drug. We proceed with our simulations based on the specification from Appendix Table 18. Table 10 shows that all firms see fewer customers under this scenario, although the effect is not identical across drugs. Figure 8 shows the distribution of the percent change from each simulation for Lipitor, Crestor, and non-advertised drugs. The results show that in the absence of DTCA, Lipitor is significantly harmed, while Crestor is harmed to a lesser degree. In general, Lipitor advertises more than Crestor during this time period. For non-advertised drugs, we see a more dispersed but still negative effect, as these drugs benefited from rival advertising.

Based on these calculations, we conclude that DTCA is primarily characterized by a business-stealing effect among branded competitors, with a small spillover to unadvertised drugs. Significantly, DTCA increases the number of patients taking all drugs in the category, advertised or not. We recognize that the statin market has a small number of players that are very close substitutes with few side-effects, and so the empirical effects may differ in other drug classes with a larger number players or where the "match" of a patient to a drug is more important.

## 7.2 Discussion

While our results present a consistent story, there are a number of caveats. First, these are short-run elasticities. Though they are much larger for new consumers, the long-run impact is unclear. Second, we do not consider selection into insurance plans or explore the role of physician agency. Given that we are looking at such a short time period, we do not believe these factors bias our results. Third, all of our results take the decision to advertise at all as given. This decision in non-random, and our treatment effects need not generalize.

Similarly, some of our results are likely to be specific to the market we study, with a limited number of advertisers who are close clinical substitutes. For example, much of the literature has examined the antidepressant market, which is similarly characterized by spillovers, but finds little evidence of business stealing effects (Avery, Eisenberg and Simon (2012); Donohue and Berndt

(2004); Narayanan, Desiraju and Chintagunta (2004) and, most recently Shapiro (2014)). Our results are consistent with these studies; for example, Shapiro (2014) finds that a cooperative advertising campaign that internalized spillovers would generate five times as many ads and increase category size by 13.7%. Our simulations are different in flavor and eliminate ads completely, but find a 5% reduction in the sales on unadvertised drugs, which comprise the bulk of the market. Here, we argue that substantial advertising expenditure is also defensive and may not provide a great deal of value from a social perspective, but that eliminating DTCA would significantly reduce the number of patients taking an effective, safe drug. Our identification strategy is likely to be useful in a number of product markets, including other drug classes. However, additional variation will be necessary to separately identify the impact of rival advertising.

A final caution is that these are only partial equilibrium calculations. Firms may alter their pricing or detailing strategies in response to changes in the competitive environment. Future work should further explore firm decisions to advertise in an equilibrium model. building on the intuition in Section 3, we would like to explore a model of advertising competition that can be estimated and used for additional counterfactual calculations. This model should be both tractable and dynamic to capture firm incentives.

# 8 Conclusion

This paper provides causal estimates of the impact of DTCA. The estimation strategy utilizes exogenous variation in the level of advertising generated by the political cycle. OLS estimates are biased due to firms strategically advertising in response to both consumer demand and competitor actions. We find significant returns to advertising in the statin market: our estimates indicate that a 10% increase in advertising leads to a 0.76% increase in revenues, holding rival actions constant. We estimate the effect in two samples: among the privately insured and among Medicare beneficiaries. In the Medicare sample, we show that our effect is primarily driven by new prescriptions. We find both business-stealing and spillover effects of advertising in the statin market.

The impact of DTCA is an empirical question of critical policy importance. Our simulations highlight the role of advertising competition and the potential for an advertising ban to reduce wasteful advertising spending. While sales of unadvertised drugs fall by nearly 5%, the savings from eliminating television advertising are substantial. Our results help quantify the tradeoffs that policy makers may face when regulating pharmaceutical firms. Furthermore, the impact of advertising is a fundamental empirical challenge and our identification strategy provides useful evidence on this important question.

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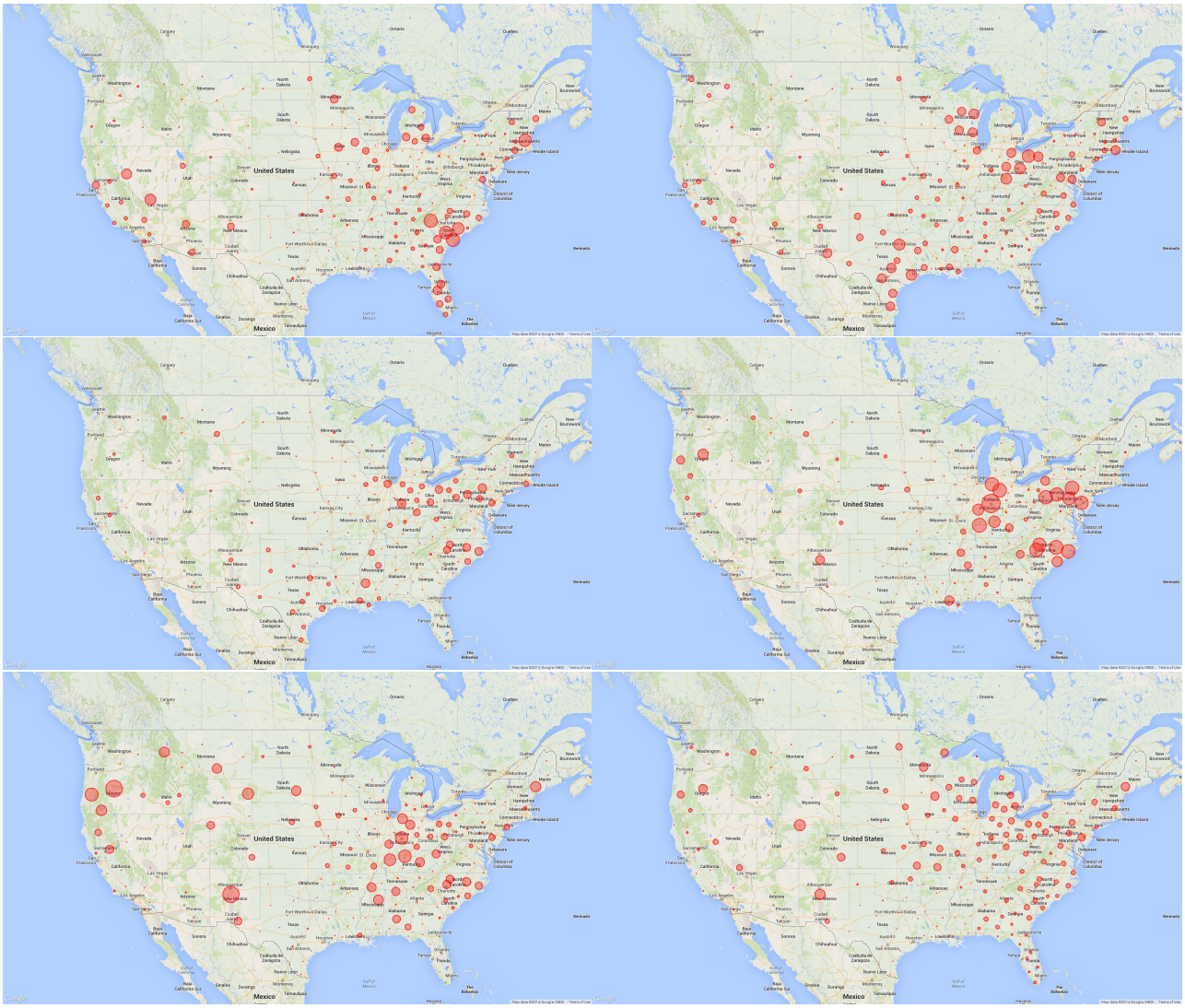
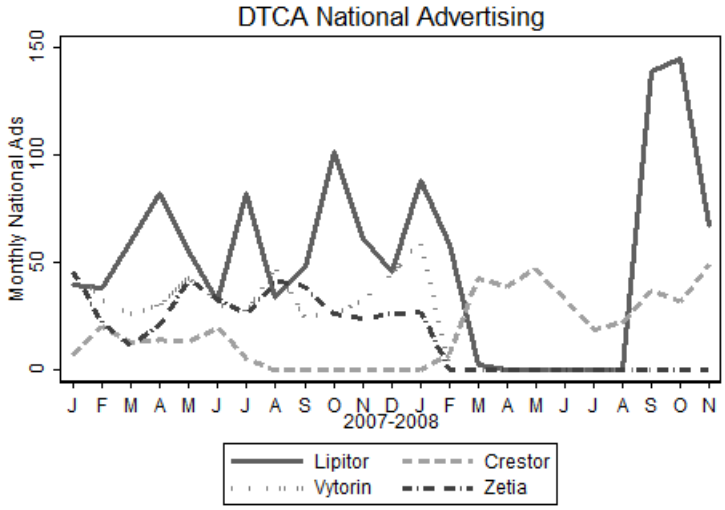
# Figures

**Figure 2:** Political Ad Levels, January-June 2008



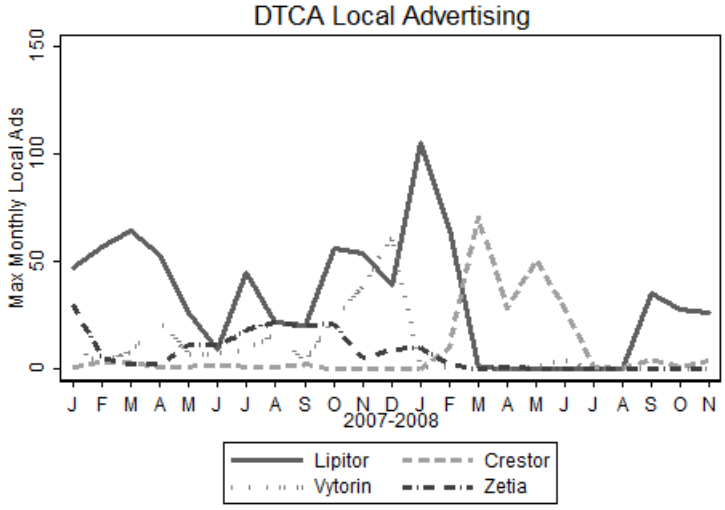

Notes: The above maps show a dot for each DMA in the USA. The diameter of each dot is proportional to the number of political ads aired in that market, in that month, for all races (Presidential, Senatorial, House, Gubernatiorial). The first row are January and February; second row are March and April, and third row are June and July.

**Figure 3:** National Pharmaceutical Ad Levels for Statins



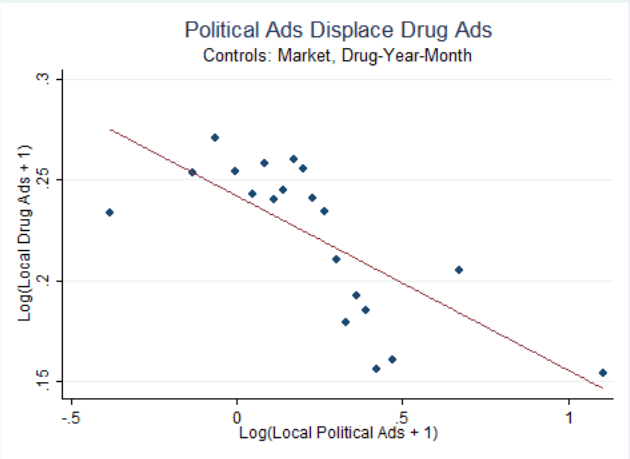

Notes: The above graphic plots spending on national advertising buys from the Kantar data. Data spans January 2007-November 2008.

**Figure 4:** National Pharmaceutical Ad Levels for Statins



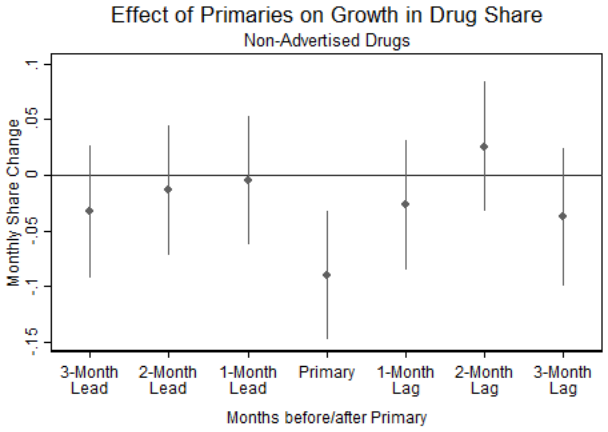

Notes: The above graphic plots spending on local advertising buys from the Kantar data. Data spans January 2007-November 2008. The axes are the same as the previous figure.

**Figure 5:** Political Ads Displace Local Drug Ads, Binned Scatter plot



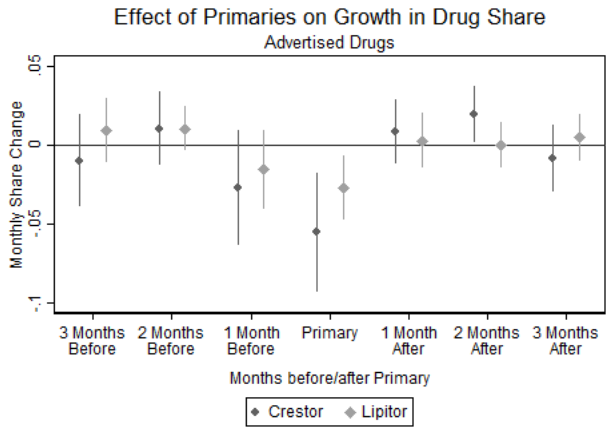

Notes: The above plots bins of observations from January 2007 to November 2008 at the market-month level after residualizing by market and year-month fixed effects, and adding back the sample mean. Twenty bins are used. The fitted line is based on a regression of all underlying data, not only the binned values.

**Figure 6:** Effect of Primaries on Growth in Market Share of Non-Advertised Statins



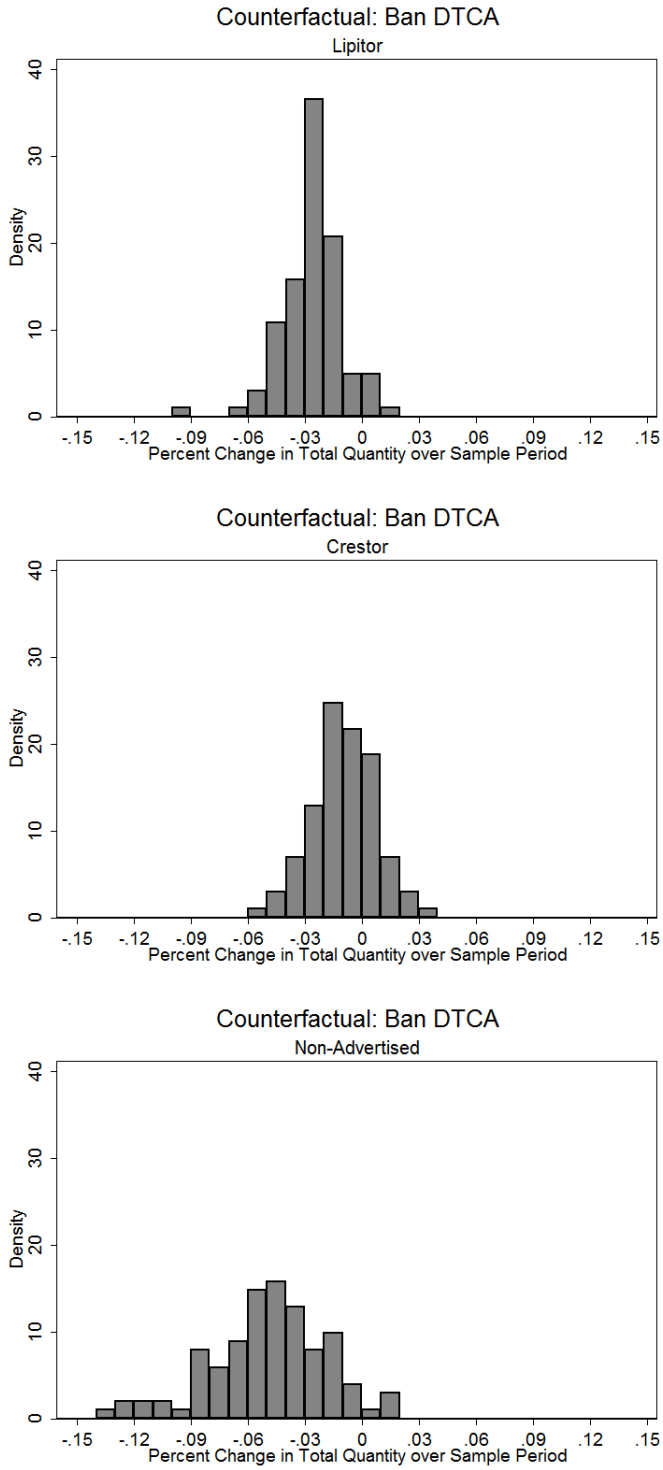

Note: The above plots estimated coefficients for timing dummies relative to a market's primary month. The dependent variable is the (one-month) change in market share, defined as the percentage of the population taking a non-advertised statin.

**Figure 7:** Effect of Primaries on Growth in Market Share of Crestor and Lipitor




Note: The above plots estimated coefficients for timing dummies relative to a market's primary month. The dependent variable is the (one-month) change in market share, defined as the percentage of the population taking Lipitor or Crestor.

**Figure 8:** Simulation Results: Eliminating DTCA




Note: The above plots are histograms of the change in quantity for each drug (or drug group) from bootstrapped simulations that eliminate DTCA from the market over the sample period. See section XX for an extended discussion of the methodology.

# Tables

**Table 1:** Summary Statistics

| Drug | | Drug Usage (Truven Analysis Data set) | |
|---|---|---|---|
| Number of Markets | 189 | Average Branded Share | 0.829% |
| Number of Months | 17 | Range, Branded Share | (0.000%, 4.71%) |
| Advertised Statins | 4 | Average Generic Share | 3.05% |
| | | Range, Generic Share | (0.000%, 7.62%) |
| Political Ads | | Drug Ads | |
| Average | 774 | Average Local Ads by Drug | 1.56 |
| Standard Deviation | 1,897 | Range, Local Ads | (0, 105) |
| Minimum | 0 | Average National Ads by Drug | 19.71 |
| Maximum | 22,636 | Range, National Ads | (0, 145) |

Notes: The Truven analysis data set limits the sample to months that are most active in political advertising, July 2007-November 2008. Average Branded Share is by drug, not aggregate for all brands.

**Table 2:** Covariate Balance, Part D Data

| | Below Median Markets | Above Median Markets | Difference |
|---|---|---|---|
| Average Age | 71.109 | 71.309 | −0.1994 |
| % Female | 0.5489 | 0.5519 | −0.0030 |
| % White | 0.8536 | 0.8727 | −0.0190 |
| % Black | 0.0849 | 0.0933 | −0.0083 |
| % Hispanic | 0.0147 | 0.0088 | 0.0058 |
| Mortality Rate | 0.0423 | 0.0425 | −0.0002 |
| % LIS | 0.6874 | 0.6657 | 0.0217** |

Notes: We split the Part D beneficiary summary sample into two groups. We take the sum of political advertising over the 2008 calendar year and compare demographics for markets above and below the median. We compare demographics across the two groups. Statistical significance at the 10%, 5% and 1% levels are denoted by *, **, and ***.

**Table 3:** Political Ads Displace Drug Ads

| | Dependent Variable: Log(Local Statin Ads + 1), Drug-Market-Year-Month Level | | | | | |
|---|---|---|---|---|---|---|
| Model: | OLS | OLS | OLS | Tobit | Tobit | Tobit |
| Log(Political Ads in 1000s + 1) | $-0.1895^{***}$ | $-0.1208^{***}$ | $-0.1208^{***}$ | $-0.8479^{***}$ | $-0.3097^{***}$ | $-0.2598^{***}$ |
| | (0.0098) | (0.0116) | (0.0116) | (0.0494) | (0.0613) | (0.0103) |
| Controls: | | | | | | |
| Market FEs | X | X | X | X | X | X |
| Year-Month FEs | | X | X | | X | X |
| Drug FEs | X | X | X | X | X | X |
| Drug-Year-Month FEs | | | X | | | X |
| $N$ | 24,150 | 24,150 | 24,150 | 24,150 | 24,150 | 24,150 |
| $R^2$ | 0.314 | 0.364 | 0.478 | 0.305 | 0.402 | 0.552 |

Notes: Unit of observation is the drug-market-month level. There are 5 advertised drugs, 210 markets and 23 months of data. OLS and Tobit standard errors clustered at the market-year-month level. Statistical significance at the 10%, 5% and 1% levels are denoted by $^{*}$, $^{**}$, and $^{***}$. Reported $R^2$ is adjusted for OLS, pseudo for Tobit.

**Table 4:** OLS Revenue Regressions for Advertised Drugs

| Dependent Variable: Log(Revenue per Insured) Drug-Market-Year-Month Level | | | | | | |
|---|---|---|---|---|---|---|
| | This Month | | Two Month Trailing Average | | Three Month Trailing Average | |
| Own Ads | 0.0194*** | 0.0064*** | 0.0239*** | 0.0066*** | 0.0316*** | 0.0086*** |
| | (0.0022) | (0.0020) | (0.0021) | (0.0021) | (0.0020) | (0.0023) |
| Rival Ads | 0.0042* | 0.0056** | 0.0016 | 0.0036 | 0.0008 | 0.0028 |
| | (0.0025) | (0.0023) | (0.0027) | (0.0026) | (0.0029) | (0.0029) |
| Controls: | | | | | | |
| Market FEs | X | X | X | X | X | X |
| Year FEs | X | X | X | X | X | X |
| Drug FEs | X | X | X | X | X | X |
| Drug FE*Time Trend | | X | | X | | X |
| Drug-Year FEs | X | | X | | X | |
| $N$ | 11,551 | 11,551 | 11,550 | 11,550 | 10,875 | 10,875 |
| $R^2$ | 0.842 | 0.847 | 0.843 | 0.847 | 0.845 | 0.849 |

Notes: Regressions are based on the Truven data. Standard errors clustered at the market-year-month level. Includes revenues for July 2007 until November 2008. Advertised drugs are Crestor, Lipitor, Vytorin and Zetia during this period. "Own Ads" and "Rival Ads" are constructed as Log(1+X). "Two Month Trailing Average" indicates that the independent variables are constructed as the average of the revenue month and one month before. The "Three Month Trailing Average" is constructed accordingly using three months. First stage excluded instruments are political advertising, its square and cube, and interactions with a dummy that takes on a one during April 2008-August 2008. Statistical significance at the 10%, 5% and 1% levels are denoted by *, **, and ***.

**Table 5:** IV Revenue Regressions for Advertised Drugs

| Dependent Variable: Log(Revenue per Insured) Drug-Market-Year-Month Level | | | | | | |
|---|---|---|---|---|---|---|
| | This Month | | Two Month Trailing Average | | Three Month Trailing Average | |
| Own Ads | $0.1559^{***}$ | $0.0808^{**}$ | $0.1252^{***}$ | $0.0764^{***}$ | $0.1048^{***}$ | $0.0536^{***}$ |
| | (0.0251) | (0.0344) | (0.0136) | (0.0258) | (0.0099) | (0.0235) |
| Rival Ads | $-0.1064^{***}$ | $-0.0492^{**}$ | $-0.0966^{***}$ | $-0.0548^{***}$ | $-0.0908^{***}$ | $-0.0407^{*}$ |
| | (0.0179) | (0.0247) | (0.0112) | (0.0212) | (0.0095) | (0.0230) |
| Controls: | | | | | | |
| Market FEs | X | X | X | X | X | X |
| Year FEs | X | X | X | X | X | X |
| Drug FEs | X | X | X | X | X | X |
| Drug FE*Time Trend | | X | | X | | X |
| Drug-Year FEs | X | | X | | X | |
| $N$ | 11,551 | 11,551 | 11,550 | 11,550 | 10,875 | 10,875 |
| $R^2$ | 0.755 | 0.819 | 0.788 | 0.824 | 0.810 | 0.840 |

Notes: Regressions are based on the Truven data. Standard errors clustered at the market-year-month level. Includes revenues for July 2007 until November 2008. Advertised drugs are Crestor, Lipitor, Vytorin and Zetia during this period. "Own Ads" and "Rival Ads" are constructed as Log(1+X). "Two Month Trailing Average" indicates that the independent variables are constructed as the average of the revenue month and one month before. The "Three Month Trailing Average" is constructed accordingly using three months. First stage excluded instruments are political advertising, its square and cube, and interactions with a dummy that takes on a one during April 2008-August 2008. Statistical significance at the 10%, 5% and 1% levels are denoted by $^{*}$, $^{**}$, and $^{***}$.

**Table 6:** Revenue Effect Decomposition

| Dependent Variable: | Log(Revenue per Insured), | | Log(Revenue per Insured), | |
|---|---|---|---|---|
| | Non-Advertised Drugs | | Advertised Drugs | |
| Model: | OLS | IV | OLS | IV |
| Own Ads | - | - | $0.0239^{***}$ | $0.0764^{***}$ |
| | | | $(0.0021)$ | $(0.0258)$ |
| Rival Ads | 0.0018 | $0.0233^{***}$ | 0.0016 | $-0.0548^{***}$ |
| | $(0.0037)$ | $(0.0089)$ | $(0.0027)$ | $(0.0212)$ |
| Controls: | | | | |
| Market FEs | X | X | X | X |
| Drug FEs and Time Trends | X | X | X | X |
| $N$ | 3,146 | 3,146 | 11,500 | 11,500 |
| $R^2$ | 0.875 | 0.874 | 0.843 | 0.824 |

Notes: Regressions are based on the Truven data. OLS and IV standard errors clustered at the market-year-month level. Revenue data are for July 2007 until November 2008. "Own Ads" and "Rival Ads" are constructed as Log(1+X), where X is the two-month trailing average of the number of ads. First stage excluded instruments are political advertising, its square and cube, and interactions with a dummy that takes on a one during April 2008-August 2008. Statistical significance at the 10%, 5% and 1% levels are denoted by $^{*}$, $^{**}$, and $^{***}$.

**Table 7:** IV Revenue Regressions for Advertised Drugs, Part D Data

| | Dependent Variable: Log(Revenue per Insured) Drug-Market-Year-Month Level | | | | | |
|---|---|---|---|---|---|---|
| | This Month | | Two Month Trailing Average | | Three Month Trailing Average | |
| Own Ads | $0.124^{***}$ | 0.0317 | $0.147^{***}$ | $0.0543^{***}$ | $0.128^{***}$ | $0.0428^{**}$ |
| | (0.0194) | (0.0242) | (0.0121) | (0.0180) | (0.00924) | (0.0184) |
| Rival Ads | $-0.0867^{***}$ | $-0.0162$ | $-0.119^{***}$ | $-0.0401^{***}$ | $-0.118^{***}$ | $-0.0350^{**}$ |
| | (0.0140) | (0.0177) | (0.0101) | (0.0151) | (0.00892) | (0.0177) |
| Controls: | | | | | | |
| Market FEs | X | X | X | X | X | X |
| Year FEs | X | X | X | X | X | X |
| Drug FEs | X | X | X | X | X | X |
| Drug FE*Time Trend | | X | | X | | X |
| Drug-Year FEs | X | | X | | X | |
| $N$ | 11,551 | 11,551 | 11,550 | 11,550 | 10,875 | 10,875 |
| $R^2$ | 0.819 | 0.874 | 0.792 | 0.866 | 0.814 | 0.872 |

Notes: Data created by collapsing Medicare Part D event data to the market-month-product level. Standard errors clustered at the market-year-month level. Includes revenues for July 2007 until November 2008. Advertised drugs are Crestor, Lipitor, Vytorin and Zetia during this period. "Own Ads" and "Rival Ads" are constructed as Log(1+X). "Two Month Trailing Average" indicates that the independent variables are constructed as the average of the revenue month and one month before. The "Three Month Trailing Average" is constructed accordingly using three months. First stage excluded instruments are political advertising, its square and cube, and interactions with a dummy that takes on a one during April 2008-August 2008. Statistical significance at the 10%, 5% and 1% levels are denoted by $^{*}$, $^{**}$, and $^{***}$.

**Table 8:** IV Revenue Regression for First Prescriptions Only, Part D Data

Dependent Variable: Log(Revenue per Insured) Drug-Market-Year-Month Level

| | This Month | | Two Month Trailing Average | | Three Month Trailing Average | |
|---|---|---|---|---|---|---|
| Model: | OLS | IV | OLS | IV | OLS | IV |
| Own Ads | 0.0496*** | 0.361*** | 0.0469*** | 0.288*** | 0.0472*** | 0.255*** |
| | (0.00408) | (0.0973) | (0.00418) | (0.0769) | (0.00467) | (0.0800) |
| Rival Ads | 0.0528*** | −0.173** | 0.0511*** | −0.149** | 0.0497*** | −0.134* |
| | (0.00531) | (0.0743) | (0.00577) | (0.0667) | (0.00631) | (0.0788) |
| Controls: | | | | | | |
| Market FEs | X | X | X | X | X | X |
| Year FEs | X | X | X | X | X | X |
| Drug FEs | X | X | X | X | X | X |
| Drug FE*Time Trend | X | X | X | X | X | X |
| $N$ | 10,789 | 10,789 | 10,788 | 10,788 | 10,125 | 10,125 |
| $R^2$ | 0.594 | 0.291 | 0.593 | 0.421 | 0.591 | 0.480 |

Notes: Data created by restricting event data to first prescriptions only and collapsing to the market-month-product level. Standard errors clustered at the market-year-month level. Includes revenues for July 2007 until November 2008. Advertised drugs are Crestor, Lipitor, Vytorin and Zetia during this period. "Own Ads" and "Rival Ads" are constructed as Log(1+X). "Two Month Trailing Average" indicates that the independent variables are constructed as the average of the revenue month and one month before. The "Three Month Trailing Average" is constructed accordingly using three months. First stage excluded instruments are political advertising, its square and cube. Statistical significance at the 10%, 5% and 1% levels are denoted by *, **, and ***.

**Table 9:** Revenue Simulations

Panel A: Simulations for Advertised Drugs

| | % Change in Revenue: | Crestor | Lipitor |
|---|---|---|---|
| (1) | Eliminating Business-Stealing | 0.2107 | 0.2327 |
| | Confidence Interval | (0.0047, 0.5639) | (0.0054, 0.6082) |
| (2) | Eliminate Political Ads | 0.0207 | 0.0163 |
| | Confidence Interval | (0.0039, 0.0689) | (0.0038, 0.0484) |

Panel B: Simulations for Non-Advertised Drugs

| | % Change in Revenue: | Unadvertised Drugs |
|---|---|---|
| (3) | Eliminate Spillovers | −0.0974 |
| | Confidence Interval | (−0.1539, −0.0262) |
| (4) | Eliminate Political Ads | −0.0030 |
| | Confidence Interval | (−0.0049, −0.0008) |

Notes: Estimates from "Two Month Trailing Average" and drug-specific time trend specifications are used in all simulations. (1) sets the coefficient on rival advertising in column 4 of Table 5 equal to zero. (2) estimates the number of drug ads in the absence of political ads, and then estimates sales at those levels of advertising. In Panel B, (3) sets the coefficient on rival advertising in column 2 of Table 6 equal to zero. (4) estimates the number of drug ads in the absence of political ads, and then estimates sales for non-advertised drugs at those levels of advertising. Estimates are bootstrapped by re-sampling the data set, re-estimating the primary specifications, and re-computing the counterfactual exercise on the observed data. We use 100 bootstrap replications and report the 2.5%-97.5% confidence interval as well as then mean.

**Table 10:** Quantity Simulations

| | % Change in Quantity | Crestor | Lipitor | Unadvertised |
|---|---|---|---|---|
| (1) | Ban All Advertising | −0.0094 | −0.0255 | −0.0491 |
| | Confidence Interval | (−0.0444, 0.0248) | (−0.0614, 0.0070) | (−0.1230, 0.0107) |

Notes: Estimates from "Two Month Trailing Average" and drug-specific time trend specifications are used in all simulations. The dependent variable is the log of the market share of a product. The simulation sets the coefficient on own and rival advertising equal to zero. Estimates are bootstrapped by re-sampling the data set, re-estimating the primary specifications, and re-computing the counterfactual exercise on the observed data. We use 100 bootstrap replications and report the 2.5%-97.5% confidence interval as well as then mean. Figure 8 shows the distributions of the simulated outcomes.

# Appendix

## Supplemental Appendix For Online Publication

# A Model Assumptions and Simulation Details

**Assumption 1.** *Function $D_j$ is smooth and continuous in all its arguments; first- and second-derivatives are defined everywhere. Function $D_j$ is concave in all arguments.*

Note that Logit demand satisfies this assumption, as do many other standard demand formulations. Concavity gives the result that rival advertising lowers the return to own advertising under spillovers, and raises it under business-stealing.

**Assumption 2.** *The following conditions hold:* $\frac{\partial D_j}{\partial a_j} > 0$ *and* $\left.\frac{\partial D_j}{\partial a_j}_{a_j=0}\right| > \frac{c}{\rho}$ .

Assumption 2 guaratees there is an incentive to advertise. If a firm's advertising creates spillovers for rivals, that implies that $\frac{\partial D_j}{\partial a_{-j}} > 0$ in our notation, while business-stealing implies $\frac{\partial D_j}{\partial a_{-j}} < 0$. When we say that the effectiveness of advertising is diminishing in the level of drug demand, we mean that $\frac{\partial D_j^2}{\partial a_j \partial \xi_j} < 0$, while if it is complementary to the level of drug demand we have $\frac{\partial D_j^2}{\partial a_j \partial \xi_j} > 0$. A firm's first-order condition for advertising is satisfied when $\frac{\partial D_j}{\partial a_j} = \frac{c}{\rho}$.

Parameters were set to the following values: $\alpha_1 = \alpha_2 = 0$, $c = 1$, $\rho = 1000$. Matlab's FSOLVE function was used to set a system of first-order conditions to zero. We use 200 markets and we draw values of $\xi$ for each firm in each market where $\xi \sim N(0, 0.25)$.

Analytic values of own and rival advertising elasticities are calculated as the mean over all observations of

$$\begin{aligned} \eta_{own} &= \beta_1(1-s_j) - \beta_2 s_{-j} \\ \eta_{rival} &= \beta_2(1-s_j) - \beta_1 s_{-j} \end{aligned}$$

We drop any simulations where Matlab's FSOLVE function failed to converge to a solution for firm first-order conditions for advertising levels. The full space of simulations covered $\beta_1 \in [0.01, 0.3]$ and $\beta_2 \in [-0.4, -0.01]$, both in increments of 0.005. The share of simulations where the bias in estimating own advertising elasticity was less than 5%, was only 1.54% of simulations, and 1.20% for rival advertising elasticity. The table below shows for one particular set of parameter values the OLS bias in estimating elasticities of own and rival ads.

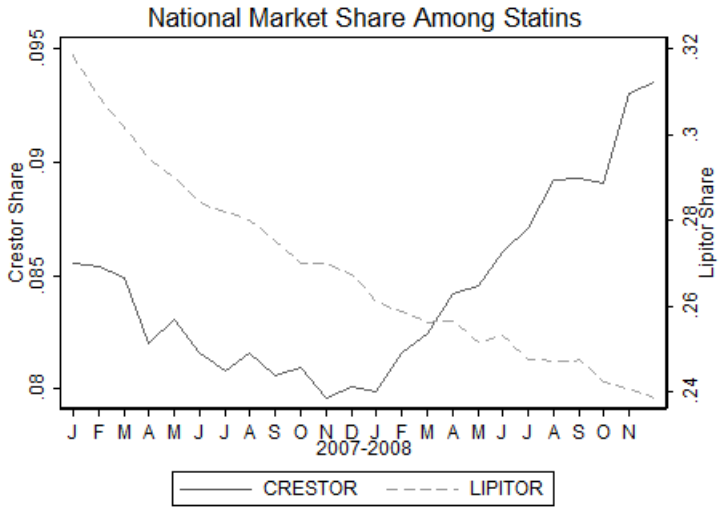
**Table 11:** Sample Model Simulation Results

| Dependent Variable: | Log(Revenue) | | |
|---|---|---|---|
| Specification: | Naive | With $\xi$ | Analytic Values |
| | (1) | (2) | (3) |
| Log(1+Own Ads) | $0.0945^{***}$ | $0.1457^{***}$ | 0.1497 |
| | (0.0263) | (0.0020) | |
| Log(1+Rival Ads) | $-0.0286$ | $-0.2641^{***}$ | $-0.2516$ |
| | (0.0238) | (0.0017) | |
| Control: $\xi$ | | X | |
| $N$ | 200 | 200 | |
| $R^2$ | 0.267 | 0.998 | |

Notes: Parameter values for these results were $\beta_1 = 0.1$ and $\beta_2 = -0.3$. Firm optimal advertising levels were solved for using Matlab's FSOLVE routine and first-order conditions for profit maximization. Estimates are for equation 1, with $\xi_j$ and $\xi_{-j}$ as additional controls in the second column.

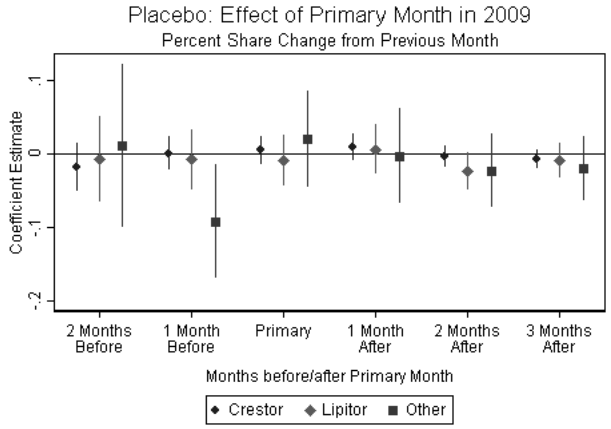
# B Additional Robustness Checks

**Figure 9:** Time Trends




Notes: The above graphic plots the share of Lipitor and Crestor from the Truven data as a percentage of total statin sales over the period of January 2007-November 2008. Note different axes.

**Figure 10:** Effect of Placebo Primaries on Shares of Non-Advertised Sales




Note: The above plots estimated coefficients for timing dummies relative to a market's primary month, with the "timing" of the primary shifted 12 months forward. The dependent variable is the (one-month) change in market share, defined as the percentage of the population taking a non-advertised statin.

**Table 12:** Robustness: No Substitution to Other Media

| | Dependent Variable: Local Non-TV Advertising Spending, Product-Market-Year-Month Level | | |
|---|---|---|---|
| Model: | OLS | OLS | OLS |
| Political Ads (1000s) | $-0.4474^{**}$ | $-0.2477$ | $-0.2802$ |
| | (0.1830) | (0.1843) | (0.1849) |
| Local TV Drug Ads | | $1.0554^{***}$ | $1.1303^{***}$ |
| | | (0.1357) | (0.1379) |
| National TV Drug Ads | | | $-0.0867^{***}$ |
| | | | (0.0125) |
| Controls: | | | |
| Market FEs | X | X | X |
| Year-Month FEs | X | X | X |
| Drug FEs | X | X | X |
| $N$ | 20,087 | 20,087 | 20,087 |
| $R^2$ | 0.080 | 0.100 | 0.101 |

Notes: Regressions combine the Wisconsin and Kantar data sets. OLS standard errors clustered at the market-year-month level. Results differ from Table 3 as this is at the individual drug level. Statistical significance at the 10%, 5% and 1% levels are denoted by $^{*}$, $^{**}$, and $^{***}$.

**Table 13:** Robustness: No Substitution to Earlier/Later Months

| | Dependent Variable: Local Drug Ads, Product-Market-Year-Month Level | | | |
|---|---|---|---|---|
| Model: | OLS | OLS | OLS | OLS |
| Political Ads (1000s) | $-0.0819^{***}$ | | $-0.0632^{**}$ | |
| | (0.0263) | | (0.0304) | |
| One Month Lag | 0.0265 | 0.0012 | | |
| | (0.0284) | (0.0299) | | |
| One Month Lead | | | $-0.0239$ | -0.0405 |
| | | | (0.0301) | (0.0294) |
| Controls: | | | | |
| Market FEs | X | X | X | X |
| Year-Month FEs | X | X | X | X |
| Drug FEs | X | X | X | X |
| Drug National Ads | X | X | X | X |
| $N$ | 8,925 | 8,925 | 8,120 | 8,120 |
| $R^2$ | 0.225 | 0.225 | 0.219 | 0.218 |

Notes: Regressions combine the Wisconsin and Kantar data sets. OLS standard errors clustered at the market-year-month level. Results differ from Table 3 as this is at the individual drug level. Statistical significance at the 10%, 5% and 1% levels are denoted by $^{*}$, $^{**}$, and $^{***}$.

**Table 17:** Effect of Business Stealing (IV Results)

| Dependent Variable: Log(Revenue per Insured) | | | | |
|---|---|---|---|---|
| Exposure: | 2-Month | | 3-Month | |
| | (1) | (2) | (3) | (4) |
| Log Own Ads | $0.0121^{***}$ | $0.0764^{***}$ | $0.0119^{***}$ | $0.0536^{***}$ |
| | (0.0022) | (0.0258) | (0.0026) | (0.0235) |
| Log Rival Ads | | $-0.0548^{***}$ | | $-0.0407^{*}$ |
| | | (0.0212) | | (0.0230) |
| Controls | | | | |
| Market FE | X | X | X | X |
| Drug FEs and Time Trends | X | X | X | X |
| N | 11,550 | 11,550 | 10,875 | 10,875 |
| $R^2$ | 0.847 | 0.824 | 0.849 | 0.840 |

Notes: Standard errors clustered at the market-year-month level. Revenue data are for July 2007 until November 2008. "Own Ads" and "Rival Ads" are constructed as Log(1+X), where X is a trailing average of the number of ads. T-statistics in parentheses. First stage excluded instruments are political advertising, its square and cube, and interactions with a dummy that takes on a one during April 2008-August 2008. Statistical significance at the 10%, 5% and 1% levels are denoted by $^{*}$, $^{**}$, and $^{***}$.

**Table 14:** Political Ads Displace Drug Ads

| | Dependent Variable: Statin Ads, Drug-Market-Year-Month Level | | | | | |
|---|---|---|---|---|---|---|
| Model: | OLS | OLS | OLS | Tobit | Tobit | Tobit |
| Political Ads (1000s) | $-0.2903^{***}$ | $-0.2257^{***}$ | $-0.2257^{***}$ | $-1.5484^{***}$ | $-0.8438^{***}$ | $-0.9257^{***}$ |
| | (0.0212) | (0.0216) | (0.0217) | (0.1271) | (0.1084) | (0.0239) |
| Controls: | | | | | | |
| Market FEs | X | X | X | X | X | X |
| Year-Month FEs | | X | X | | X | X |
| Drug FEs | X | X | X | X | X | X |
| Drug-Year-Month FEs | | | X | | | X |
| $N$ | 24,150 | 24,150 | 24,150 | 24,150 | 24,150 | 24,150 |
| $R^2$ | 0.226 | 0.258 | 0.374 | 0.198 | 0.254 | 0.337 |

Notes: Regressions combine the Wisconsin and Kantar data sets. Unit of observation is the drug-market-month level. OLS and Tobit standard errors clustered at the market-year-month level. Statistical significance at the 10%, 5% and 1% levels are denoted by $^{*}$, $^{**}$, and $^{***}$.

**Table 15:** Political Ads and Ad Pricing

| | Dependent Variable: Cost per Unit of Drug Advertising | | | |
|---|---|---|---|---|
| Political Ads (1000s) | 0.0029 | 0.0037 | 0.0023 | 0.0012 |
| | (0.0059) | (0.0051) | (0.0064) | (0.0063) |
| Controls: | | | | |
| Market FEs | | X | X | X |
| Month FEs | | | X | X |
| Drug FEs | | | | X |
| $N$ | 2584 | 2584 | 2584 | 2584 |
| $R^2$ | 0.000 | 0.578 | 0.610 | 0.634 |

Notes: Regressions combine the Wisconsin and Kantar data sets. Unit of observation is the drug-market-month level. Standard errors clustered at the market-year-month level. Statistical significance at the 10%, 5% and 1% levels are denoted by $^{*}$, $^{**}$, and $^{***}$.

**Table 16:** OLS Revenue Regressions for Advertised Drugs, Part D Data

| | Dependent Variable: Log(Revenue per Insured) Drug-Market-Year-Month Level | | | | | |
|---|---|---|---|---|---|---|
| | This Month | | Two Month Trailing Average | | Three Month Trailing Average | |
| Own Ads | 0.0229*** | 0.00991*** | 0.0259*** | 0.00817*** | 0.0324*** | 0.00792*** |
| | (0.00215) | (0.00186) | (0.00197) | (0.00192) | (0.00192) | (0.00208) |
| Rival Ads | 0.00457** | 0.00692*** | 0.000423 | 0.00206 | −0.000383 | 0.000453 |
| | (0.00200) | (0.00187) | (0.00217) | (0.00209) | (0.00238) | (0.00235) |
| Controls: | | | | | | |
| Market FEs | X | X | X | X | X | X |
| Year FEs | X | X | X | X | X | X |
| Drug FEs | X | X | X | X | X | X |
| Drug FE*Time Trend | | X | | X | | X |
| Drug-Year FEs | X | | X | | X | |
| $N$ | 11,551 | 11,551 | 11,550 | 11,550 | 10,875 | 10,875 |
| $R^2$ | 0.871 | 0.877 | 0.871 | 0.877 | 0.873 | 0.878 |

Notes: Data created by collapsing Medicare Part D event data to the market-month-product level. OLS and IV standard errors clustered at the market-year-month level. Includes revenues for July 2007 until November 2008. Advertised drugs are Crestor, Lipitor, Vytorin and Zetia during this period. "Own Ads" and "Rival Ads" are constructed as Log(1+X). "Two Month Trailing Average" indicates that the independent variables are constructed as the average of the revenue month and one month before. The "Three Month Trailing Average" is constructed accordingly using three months. First stage excluded instruments are political advertising, its square and cube, and interactions with a dummy that takes on a one during April 2008-August 2008. Statistical significance at the 10%, 5% and 1% levels are denoted by *, **, and ***.

**Table 19:** Heterogeneity, Part D Data

| Dependent Variable: Log(Revenue per Insured) | | | | |
|---|---|---|---|---|
| | Two-Month Trailing Average | | Three-Month Trailing Average | |
| | OLS | IV | OLS | IV |
| Panel A: Beneficiaries Ending Year in Initial Coverage Phase | | | | |
| Log Own Ads | 0.0347*** | 0.221*** | 0.0437*** | 0.186*** |
| | (0.00219) | (0.0177) | (0.00215) | (0.0132) |
| Log Rival Ads | 0.00883*** | −0.166*** | 0.00908*** | −0.157*** |
| | (0.00275) | (0.0148) | (0.00300) | (0.0129) |
| $N$ | 11,550 | 11,550 | 10,875 | 10,875 |
| $R^2$ | 0.842 | 0.682 | 0.845 | 0.735 |
| Panel A: Beneficiaries Ending Year in Donut Hole | | | | |
| Log Own Ads | 0.0212*** | 0.203*** | 0.0273*** | 0.184*** |
| | (0.00238) | (0.0181) | (0.00235) | (0.0143) |
| Log Rival Ads | −0.00875*** | −0.186*** | −0.00922*** | −0.194*** |
| | (0.00287) | (0.0151) | (0.00312) | (0.0139) |
| $N$ | 11,547 | 11,547 | 10,872 | 10,872 |
| $R^2$ | 0.824 | 0.666 | 0.825 | 0.691 |
| Panel A: Beneficiaries Ending Year in the Catastrophic Phase | | | | |
| Log Own Ads | 0.0135*** | −0.0128 | 0.0149*** | 0.000359 |
| | (0.00260) | (0.0164) | (0.00260) | (0.0129) |
| Log Rival Ads | −0.00290 | 0.0100 | −0.00766** | −0.00157 |
| | (0.00323) | (0.0140) | (0.00351) | (0.0130) |
| $N$ | 11,491 | 11,491 | 10,819 | 10,819 |
| $R^2$ | 0.795 | 0.793 | 0.796 | 0.795 |

Notes: Data created by restricting event data to beneficiaries ending the year in each phase and collapsing to the market-month-product level. OLS and IV standard errors clustered at the market-year-month level. Includes revenues for July 2007 until November 2008. Advertised drugs are Crestor, Lipitor, Vytorin and Zetia during this period. "Own Ads" and "Rival Ads" are constructed as Log(1+X). "Two Month Trailing Average" indicates that the independent variables are constructed as the average of the revenue month and one month before. The "Three Month Trailing Average" is constructed accordingly using three months. First stage excluded instruments are political advertising, its square and cube. All regressions include market, year, and drug fixed effects and a drug specific time trend. Statistical significance at the 10%, 5% and 1% levels are denoted by *, **, and ***.

**Table 18:** Robustness Checks (IV Results)

| | Dependent Variable: Log(Revenue per Insured) | | | | | Dependent Variable: Log(Days Supply per Insured) | |
|---|---|---|---|---|---|---|---|
| Own Ads (2-Month Trailing) | 0.766*** | | 0.1306*** | 0.1189** | 0.0734*** | 0.1495*** | 0.0657*** |
| | (0.0258) | | (0.0492) | (0.0549) | (0.0270) | (0.0141) | (0.0255) |
| Rival Ads (2-Month Trailing) | −0.0550*** | | −0.0958** | −0.0915** | -0.0425** | −0.1202*** | −0.0488** |
| | (0.0212) | | (0.0383) | (0.0451) | (0.0215) | (0.0116) | (0.0209) |
| Jan.-Jun. '07 Ads | 0.0699** | | | | | | |
| | (0.0322) | | | | | | |
| 1-Month Lagged Own Ads | | 0.1444*** | | | | | |
| | | (0.0466) | | | | | |
| 1-Month Lagged Rival Ads | | −0.1041*** | | | | | |
| | | (0.0365) | | | | | |
| Market FEs | X | X | X | X | X | X | X |
| Year FEs | X | X | X | X | X | X | X |
| Drug FEs | X | X | X | X | X | X | X |
| Drug FE*Time Trend | X | X | X | X | X | | X |
| Drug FE*Time Trend^2 | | | | | X | | |
| Drug-Year FEs | | | | | | X | |
| 1(FDA), in first stage | X | X | | X | X | X | X |
| 1(FDA), in second stage | | | | X | | | |
| $N$ | 11,551 | 11,551 | 11,550 | 11,550 | 11,550 | 11,550 | 11,550 |
| $R^2$ | 0.871 | 0.877 | 0.776 | 0.787 | 0.832 | 0.745 | 0.822 |

Notes: Data created by collapsing Medicare Part D event data to the market-month-product level. Standard errors clustered at the market-year-month level. Includes revenues for July 2007 until November 2008. Advertised drugs are Crestor, Lipitor, Vytorin and Zetia during this period. "Own Ads" and "Rival Ads" are constructed as Log(1+X). "Two Month Trailing Average" indicates that the independent variables are constructed as the average of the revenue month and one month before. The "Three Month Trailing Average" is constructed accordingly using three months. First stage excluded instruments are political advertising, its square and cube, and interactions with a dummy that takes on a one during April 2008-August 2008. Statistical significance at the 10%, 5% and 1% levels are denoted by *, **, and ***.

**Table 20:** Heterogeneity II, Part D Data

| Dependent Variable: Log(Revenue per Insured) | | | | |
|---|---|---|---|---|
| | Two-Month Trailing Average | | Three-Month Trailing Average | |
| | OLS | IV | OLS | IV |
| Panel A: Beneficiaries in MA Plans | | | | |
| Log Own Ads | 0.0203**** | 0.106*** | 0.0271*** | 0.0963*** |
| | (0.00345) | (0.0219) | (0.00344) | (0.0172) |
| Log Rival Ads | −0.00876** | −0.0994*** | −0.0118** | −0.104*** |
| | (0.00445) | (0.0191) | (0.00482) | (0.0177) |
| $N$ | 10,996 | 10,996 | 10,364 | 10,364 |
| $R^2$ | 0.813 | 0.795 | 0.814 | 0.800 |
| Panel A: Beneficiaries in Stand-alone Part D Plans | | | | |
| Log Own Ads | 0.0267*** | 0.167*** | 0.0330*** | 0.143*** |
| | (0.00192) | (0.0128) | (0.00186) | (0.00975) |
| Log Rival Ads | 0.00295 | −0.134*** | 0.00251 | −0.130*** |
| | (0.00215) | (0.0107) | (0.00236) | (0.00935) |
| $N$ | 11,550 | 11,550 | 10,875 | 10,875 |
| $R^2$ | 0.893 | 0.800 | 0.894 | 0.826 |
| ...Panel C: LIS-Eligible Beneficiaries Only | | | | |
| Log Own Ads | 0.0207*** | 0.0348*** | 0.0241*** | 0.0379*** |
| | (0.00231) | (0.0121) | (0.00227) | (0.00907) |
| Log Rival Ads | 0.00252 | −0.0223** | −0.000817 | −0.0288*** |
| | (0.00260) | (0.0102) | (0.00287) | (0.00911) |
| $N$ | 11,538 | 11,538 | 10,863 | 10,863 |
| $R^2$ | 0.858 | 0.856 | 0.858 | 0.857 |

Notes: Data created by restricting event data to beneficiaries ending the year in each plan type and collapsing to the market-month-product level. OLS and IV standard errors clustered at the market-year-month level. Includes revenues for July 2007 until November 2008. Advertised drugs are Crestor, Lipitor, Vytorin and Zetia during this period. "Own Ads" and "Rival Ads" are constructed as Log(1+X). "Two Month Trailing Average" indicates that the independent variables are constructed as the average of the revenue month and one month before. The "Three Month Trailing Average" is constructed accordingly using three months. First stage excluded instruments are political advertising, its square and cube. All regressions include market, year, and drug fixed effects and a drug specific time trend. Statistical significance at the 10%, 5% and 1% levels are denoted by *, **, and ***.