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

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Abstract

We measure the impact of direct-to-consumer television advertising (DTCA) by drug manufacturers. Our identification strategy exploits shocks to local advertising markets generated by the political advertising cycle and a regulatory intervention affecting a single product. We find evidence of significant business stealing effects among branded, advertised drugs. In addition, we show positive spillovers from drug advertisements to non-advertised competitors in the same class. We decompose the effect and show it is primarily due to new customers. Finally, we provide evidence that DTCA is cost-effective from a societal standpoint in our setting.

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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, as ads can provide valuable information for consumers or, alternatively, create "spurious product differentiation" (Bagwell (2007)). The impact and consequences of advertising are empirical questions, although estimation is challenging due to endogeneity, issues in measurement, and heterogeneity across consumers. This paper credibly shows that advertising can generate both positive spillovers within a product category and business-stealing effects among rivals. This provides evidence of the impact of advertising and, therefore, strategic incentives facing firms. In addition, our estimates focus on a policy-relevant product class: prescription drugs.

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. Industry insiders suggest 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."[1]

We identify the effectiveness of TV advertising for anti-cholesterol drugs known as statins.[2] 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 that combines regulatory action and displacement from political advertising 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

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

[2] 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.

a transitory or permanent demand shock. Interaction between firms also has major implications for measurement and estimation: if advertising is largely business stealing, firms may 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 positive spillovers, firms may have an incentive to reduce advertising. We exploit exogenous variation in advertising levels to measure effectiveness.

Our identification strategy exploits novel variation in advertising due to political campaigning in the lead-up to 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.[3] Our first-stage estimates imply that the thousands of political ads aired through the election cycle had a significant displacement effect on DTCA. However, this shock affected all products. To separately estimate the impact of own and rival advertising, we interact political advertising with a regulatory action that temporarily halted a Lipitor campaign for part of 2008.

We highlight our identification strategy using four sets of complementary reduced form analyses. First, graphical analyses show that political primaries are associated with statistically significant reductions in drug sales using market-month-drug level usage data from Truven MarketScan. Second, a difference-in-differences analysis shows the impact of political advertising on Crestor and Lipitor sales during and outside of the regulatory action period. Third, we present a saturated fixed-effect OLS model which indirectly exploits the political advertising shocks by including product-month fixed effects; conditional on those controls, we argue that advertising is as good as randomly assigned. Finally, our IV regression results show an own-advertising elasticity of revenue with respect to the quantity of ads of 0.0761 for a sample of privately insured consumers. We also provide estimates of revenue elasticities with respect to rival advertising: here, we estimate an elasticity of -0.0547. We separately estimate the impact on non-advertised branded and generic drugs and estimate an elasticity with respect to branded advertising of 0.0188. Therefore, advertising has a business-stealing effect among branded, advertised drugs, but a positive 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. We estimate much larger elasticities for new consumers who have no history of statin use. Both data sets tell a consistent story: DTCA has an economically

[3] While the list of swing states varies from election to election and there is no clear definition, the 2008 presidential race was most competitive in Colorado, Florida, Indiana, Missouri, Nevada, New Hampshire, New Mexico, North Carolina, Ohio, Pennsylvania, and Virginia.

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.

By examining multiple datasets, we can document heterogeneity in the effects of advertising across drugs and different types of consumers. In order to synthesize our results and simulate the effects of a ban on DTCA – the key policy counterfactual of interest – we specify a model of consumer demand that builds on the reduced form estimates and identification strategy. The model is flexible enough to allow the impact of ads to vary across drugs and new and existing consumers. The results show that banning DTCA harms sales of all statins including non-advertised (often generic) drugs. We combine our estimates with external information on the value of statin consumption to provide evidence that advertising is cost effective from a social perspective, as the market expansion effects outweigh the socially wasteful business stealing aspect of advertising in this category of highly effective drugs. This finding should caution policymakers and physicians interested in banning or curtailing DTCA.

While we believe our paper is the first to exploit this form of political advertising as an instrument for drug advertising, we build on a substantial literature examining the impact of DTCA.[4] 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), Wosinska (2002), Wosinska (2005), Rosenthal et al. (2003), Berndt et al. (1995), Liu and Gupta (2011)). We also contribute to the literature on the economics of the prescription drug market (see Scott Morton and Kyle (2012) for a survey) and how firms deploy DTCA (Ling, Berndt and Kyle (2002)). The papers closest to our study are Jin and Iizuka (2005), which finds positive effects of advertising on doctor visits across a large set of drug classes and demographic groups, and Shapiro (2016), 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 and exploiting a time-varying shock to advertising levels.

This paper also contributes to a literature that attempts to measure the causal impact of advertising. Recent papers such as Lewis and Reiley (2014), Lewis and Rao (2015) and Blake, Nosko and Tadelis (2015) have utilized randomized experiments on online platforms while others such as Hartmann and Klepper (2015) and Stephens-Davidowitz, Varian and Smith (2015) use plausibly exogenous cross-sectional shocks to ad viewership due to Super Bowl ratings. 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. Our results are

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

highly relevant to policy makers in the United States and abroad, where DTCA remains a contentious issue, especially in connection with the literature on the effects of patent expiry (Kyle and McGahan (2012), Scott Morton (1999)).

The paper is organized as follows. Section 2 describes the market and setting and estimation bias. Section 3 describes the data and empirical strategy, while Section 4 presents results and robustness checks. Section 5 details the demand model and counterfactual analyses, and Section 6 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 1992.

Figure 1 provides a timeline of major events in this market. In 1996, Pfizer began marketing Lipitor, and in 2003, AstraZeneca introduced Crestor. Pfizer marketed Lipitor to consumers aggressively beginning in 2001. According to trade press and news, this heralded an increase in the "arms race of drug marketing."[5] Merck introduced Zetia, which blocks absorption of dietary cholesterol, in 2002, and Vytorin, which combines the active ingredients in Zetia and Zocor, in 2004. Zocor's patent expired in 2006, and heavy generic competition began shortly thereafter. This hurt the sales of not only branded Zocor, but also Crestor and 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.[6] Lipitor's patent expired at the end of November 2011 and Crestor's expired 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

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

[6] See Ellison and Ellison (2011) for a discussion of strategic behavior around patent expiration. This is in contrast to over-the-counter medications, which are often advertised even though an exact molecular substitute is available. See Bronnenberg et al. (2014) for details.

physicians): in 1994, 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, AstraZeneca released the results of the ECLIPSE trial, which favored Crestor relative to Lipitor for some sub-populations of patients,[7] corresponding to the increase in Crestor marketing. During our sample period, the ENHANCE trial results led to the end of advertising of Vytorin and Zetia in 2008.[8] The study showed that Vytorin (Zetia and Zocor combined) was no better than Zocor alone.[9] The American Academy of Cardiologists recommended that doctors no longer prescribe Vytorin and strongly discouraged the use of Zetia.[10] The effect on Vytorin's market share was dramatic, falling 10% immediately and 40% over the course of 2008 in our sample data; Zetia sales fell by 5% immediately.[11]

During 2007 and 2008, four branded anti-cholesterol medications were being advertised. The two largest products by both advertising and sales were Lipitor and Crestor, while Vytorin and Zetia were also marketed to consumers during this time period. Lipitor halted its advertising campaign featuring Dr. Robert Jarvik (developer of the Jarvik artificial heart) in April of 2008. Many, including Congress, had concluded that the advertisements were misleading.[12] As a result, Crestor was the only statin airing TV spots from April 2008 until August 2008. In 2008, Lipitor's sales fell by 2% and Crestor's sales rose by nearly 29%; Appendix Figure 12 plots time trends for Crestor and Lipitor during our sample period, which we flexibly control for in our empirical analysis.

2.1 Demand

Statins are widely covered by insurance plans. Most consumers with employer-sponsored health insurance have prescription drug coverage as part of their benefits package.[13] Insurance coverage for these drugs is usually generous, and consumers will face only a small 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.

[7] 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.

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

[9] The study was completed in 2006. See Greenland and Lloyd-Jones (2008)

[10] Davidson and Robinson (2007)

[11] By contrast, a recent, much larger study (18,000 subjects vs. just 750) found Vytorin to be more effective than simvastatin (Zocor) 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.

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

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

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: during our sample period there was an initial deductible, followed by (an average of) 25% co-payment rates up to an initial coverage limit. Once a consumer hit the initial coverage limit, they had to pay for all of their expenditure in the "donut hole" or coverage gap until they met a catastrophic cap. 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, and consumer price sensitivity will be a function of complex plan features (Dalton, Gowrisankaran and Town (2014); Einav, Finkelstein and Schrimpf (2014); Abaluck, Gruber and Swanson (2015)). 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 Scott Morton (2010)). Therefore, plan selection and copay structure are more likely to be a concern in the Medicare Part D setting; to preview our results, we estimate similar elasticities among the employer sponsored and Medicare populations, reducing the concern that plan selection may bias our results.

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 (detailing is known as a "push" technique, as opposed to "pull" techniques that target the consumer). Appendix C shows that payments to physicians are unaffected by political advertising shocks using data from 2014 and so is therefore likely to remain constant during our sample period, allowing us to focus on measuring the impact of DTCA. Both plan selection and physician agency are outside the scope of this paper.

2.2 Firm Advertising Decisions and Estimation Bias

The direction of bias in OLS estimates is ambiguous in the context of firm advertising decisions. Advertising levels depend on consumer responsiveness to ads, which will in turn depend on the functional form and parameters of the demand system.[14] In the case of a single firm advertising, optimal advertising choices that create a positive correlation between demand shocks and advertising lead to upward bias in OLS estimates. By contrast, a negative correlation between demand

[14]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.

shocks and advertising leads to downward bias in OLS estimates.

In the case of multiple firms advertising, the levels of advertising 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 positive spillover effect on Lipitor, when that is not the case. The strategic interactions among firms can therefore lead to correlations between advertising levels and unobservables that result in upward or downward bias in OLS estimates. Appendix A.1 graphically shows the ambiguous bias in simulated datasets of a Logit formulation of the above setting.

Understanding the forces that shape equilibrium outcomes is critical for policy makers. If advertising generates spillovers, we would expect it to be under-supplied in equilibrium relative to the social optimum: the advertising firm cannot capture all of the surplus generated. Similarly, if advertising is 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.[15]

3 Data and Empirical Strategy

3.1 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; the majority of states use government-run primary elections, and the remainder use party-run caucuses. The staggered nature of the primaries

[15] This is nicely illustrated in the market for antidepressants by Shapiro (2016).

increases the national attention on and importance of early contests in Iowa and New Hampshire, as well as South Carolina, Florida and Nevada.[16] In 2008, there was no incumbent candidate for either party; the Democratic party contest between Hillary Clinton and Barack Obama extended into June, while John McCain secured the Republican nomination by March of 2008.

During the general election, the "winner take all" nature of the Electoral College means that political advertising in swing states is far more valuable than in "safe states", leading to large variations in the numbers of ads different markets across markets (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 have preferential rules for buying advertising and both they and outside influence groups often purchase premium advertising slots that can pre-empt previously purchased advertising.[17] The 2008 election cycle was notable for breaking records for spending by candidates, with Barack Obama alone spending more than the total spent by both presidential candidates in 2004. The lengthy primary process and the rejection of public funding both contributed to the vast amounts of money spent during the campaign cycle.

The Bipartisan Campaign Reform Act of 2002 established several regulations over the purchasing of advertising by political campaigns during our time period. In particular, in the 45 days leading to a primary or 60 days leading to a general election, broadcast outlets can only charge qualified political campaigns their "lowest unit rate" (LUR) for a given class of advertising (e.g. non-preemptible, preemptible with notice, or "run-of-schedule"). They are further required to offer "reasonable access" to federal office candidates, and "equal opportunity" for candidates in non-federal races. More recently, advertising agencies are actively warning clients about "heavy pre-emption of existing advertising schedules"[18] due to the steady increase in election spending and the 2010 *Citizens United* Supreme Court ruling.[19]

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 rivals' 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

[16] 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

[17] See the discussion in Gordon and Hartmann (2014) regarding how political campaigns purchase advertisements.

[18] "Navigating Media Through Political Season", Mark Buchele, Gragg Advertising. Archived at: https://web.archive.org/web/20150906080219/http://www.graggadv.com/navigating-media-political-season/

[19] 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.

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 regulatory shock on displacement from political advertising across markets is uncorrelated with drug demand; this allows us to compare the effect of political advertising shocks in markets with substantial Crestor advertising but no Lipitor advertising to markets where political advertising displaces both Lipitor and Crestor advertising. We do not need to exclude the direct effect of the regulatory action from the second stage of the regression; we can simply use the interaction of political advertising with the timing of the regulatory action.

3.2 Data

We combine two sources of advertising data. First, data from Kantar Media contain both the number of drug ads and the level of spending for 2007-2008 at the month-drug level for every designated market area (DMA) in the United States. We also have a record of every political ad (house, presidential, senatorial, 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 DMA-month varies widely during the Jan 2007-Nov 2008 time period: half of the DMA-month observations during this period have zero ads, while some DMA-months have over 20,000 political ads in a month (e.g. 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.[20] The mean number of monthly political ads by DMA 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 DMA 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 total number 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).[21] Local advertising can be a substantial portion of a firm's total advertising. While some DMAs 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 and that there is substantial geographic variation, as highlighted in Appendix Figure 14.

[20] Maps for the remaining months of 2008 are in Appendix Figure 13.

[21] National advertising levels are driven by a number of factors, including the release of clinical trial data that may impact demand. For example, Vytorin and Zetia quit advertising after the release of the ENHANCE trial (Greenland and Lloyd-Jones (2008)) and Crestor increased advertising after the release of the ECLIPSE study (Faergeman et al. (2008)).

We combine this advertising data with prescription drug usage and revenue data from two sources. First, we use Truven MarketScan data, which draws from a convenience sample of large, self-insured firms. These data represent individuals enrolled in traditional, employer-sponsored insurance. 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 186 DMAs and 17 months, spanning July of 2007-November of 2008.[23] The sample is younger than the population on the whole, and a relatively small proportion of this population takes statins.

We supplement this data with data from the Medicare Part D program, where we have individual demographic information. Our data represent a 10% random sample of all Medicare Part D beneficiaries. This data allows for tracking of individual consumer behavior. We restrict our sample to the same 186 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 DMAs that experience more or less than the median level of political advertising 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 DMAs with fewer political ads seem to be slightly poorer; if anything, income effects would only increase drug demand in above median DMAs and thus bias our results toward zero, assuming prescription drugs are a normal good.

4 Results

4.1 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 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 scatter plot highlighting the relationship between political advertising and statin advertising, where observations are de-meaned by DMA

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

[23] There are 210 DMAs in the United States. We drop those that do not have any political ads or where Marketscan did not report data due to an insufficient number of observations.

and drug-year-month, and then binned to create a scatter plot of the data. This highlights the negative relationship between political advertising and drug advertising looking across DMAs within a drug-month pair. For example, there were 5120 political ads aired in Iowa in January of 2008 and 126 statin ads, as compared to an average of 19 political ads and 133 statin ads in all other DMAs. This figure shows that an increase in political ads in Iowa in January of 2008 implies lower statin advertising as compared to the level of statin advertising in other DMAs in the same month.[24]

In order to separately estimate the impact of own and rival ads, we need an additional source of identifying variation. Appendix Figure 15 describes how regulatory action combines with political advertising to give us sufficient advertising variation to identify both effects. The right-hand panel shows the relationship between political and drug advertising for all drugs except Lipitor. There is a strong negative correlation between the two series during our entire sample period. By contrast, the left-hand panel shows the relationship between political advertising and Lipitor advertising. During the time period excluding the regulatory action months, the effect of political advertising is still strong and negative. However, during the regulatory action period, Lipitor runs no ads for plausibly exogenous reasons, and Crestor could not react to this change in the short-run. While this type of variation is less likely to be available in other settings, limiting the generalizability of our strategy, is it critical to have two independent sources of variation to separately identify own and rival advertising effects.

Table 3 presents a regression of the number of statin advertisements for a drug in a DMA on the number of political advertisements (in 1000s). The level of observation is a DMA-month for January 2007 until November 2008. We include drug-year-month fixed effects so that we can exploit solely cross-sectional variation. The OLS results show that a 1000 political ads displace 0.1583 ads. To account for the fact that drug ads cannot be negative, the last columns of Table 3 estimate a Tobit model. We find a significantly larger effect: 1000 political ads displace 0.6941 ads. Appendix Table 13 shows the analogous results using logs instead of levels, with all results strongly negative and significant.

In order to understand the magnitude of these effects, we plot a histogram of the size of political shocks by market in Figure 6. In each market, we calculate the maximum number of political ads within a market across all months. All markets receive shocks from the political process. For Lipitor, in the median market by level of political advertising in the month of the shock, 16% of drug ads (9 ads) are displaced, while at the 90th percentile of markets, 30% of ads (18 ads) are displaced in the month of the political shock. The maximum of political ads (national and local) across months and markets in our data is 25,381. In this market-month, our estimates imply a

[24] In our main specifications, the endogenous regressor is the total level of advertising, rather than local advertising alone. Local and national advertising are positively correlated (the correlation coefficient is 0.4). However, the variation we exploit is primarily from the local advertising shocks, as we control for product fixed effects and allow for flexible product time trends.

displacement of 31 Lipitor ads, or 54% of the total. For Crestor by level of political advertising in the month of the shock, in the median market, political ads displace 12% of ads (2-3 ads).

We address three 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 Appendix Table 14, 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 Appendix Table 15, we show that total local drug ad spending is not affected by political ads once local TV ads are controlled for.[25] Third, firms may modify their detailing plans due to the displacement of their local TV ads by political ads. In Appendix C, we show that payments to physicians are not responsive to the political cycle using data from 2014. Furthermore, 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.[26]

We believe that pre-emption – as opposed to prices – drives our first stage results for several reasons. The 2008 election was "surprising" in a number of key ways. First, the strength of Obama's campaign was unexpected, which led to a longer than expected Democratic primary. Second, the political environment and historic nature of Obama's campaign expanded the number of battleground states; for example, Obama advertised heavily in, and eventually won, Virginia and North Carolina, both of which were won by George W. Bush in 2004. Finally, the level of spending in 2008 was much greater than expected: Obama rejected public funds (despite an earlier campaign promise), raising $745 million dollars for his campaign, over twice McCain's fundraising total (West (2013)). It would be difficult for pharmaceutical manufacturers to adapt to these evolving market conditions, especially since most airtime is sold in an upfront market in May of each year (Phillips (2012)).[27]

[25] 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.

[26] Another concern is that Lipitor detailing may have increased during the regulatory action period. This could potentially bias our estimates of the effect of own advertising toward zero. However, our results are robust to including both a regulatory action dummy and its interaction with product fixed effects in both stages of the model. In addition, 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 effective ads in our data.

[27] We also note that the exclusion restriction is valid even if firms adjust along other margins; political advertising still represents a shock to the cost of advertising. To the extent that detailing or other forms of advertising are complements or substitutes to DTC ads, the shocks shift firms toward a less desirable advertising strategy, and our results still highlight a large business stealing component of pharmaceutical marketing.

4.2 Graphical Evidence

We now present a number of simple graphical analyses. We initially focus on non-advertised 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, and Zetia. If these ads have spillover effects on non-advertised (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 7 shows the effect of primaries on overall market share growth for non-advertised 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 16 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 8; the magnitude of the effect of primaries on Crestor sales is nearly twice as large as the effect on Lipitor sales. During the sample 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 suggest that the absence of DTCA would lead to a drop in overall drug sales.

4.3 Difference-in-Difference Estimates

We observe local political advertising in 1,434 of our 3,200 DMA-month combinations. Because political advertising displaces drug advertising, we expect prescription fills to be lower in markets with local political advertising, just as we expect lower sales in primary months. In the first column of Table 4, we show that DMA-months with political ads have 3% lower sales of both Crestor and Lipitor. Furthermore, because no Lipitor ads ran during the regulatory action period discussed in the previous section, we can illustrate our identification strategy using a difference-in-differences specification. In general, local political activity should reduce Lipitor sales by reducing the number of Lipitor ads. Similarly, the regulatory action should reduce Lipitor sales due to either negative publicity or a lack of advertising or both. We can distinguish between business stealing and spillover effects by measuring the impact of local political ads during the regulatory action period. Under business stealing, the sign of the interaction (political advertising during the regula-

tory action period) should be positive: there are fewer Crestor ads aired in markets with political advertising and fewer Crestor ads imply higher Lipitor sales. If there are spillovers, the opposite should be true.

Therefore, we estimate the following difference-in-differences specification in a sample limited to Lipitor:

$$Y_{tm} = \beta_1 + \beta_2 * P_{tm} + \beta_3 * I_t + \beta_4 * P_{tm} * I_t + u_m + \varepsilon_{tm},$$

where Y_{tm} is sales in DMA m and month t, P_{tm} is an indicator for any political ads in DMA m and month t, I_t is an indicator for the regulatory action months that prevented Lipitor from advertising, and u_m is a DMA fixed-effect. The main effect of the regulatory action, β_3 will capture the effect of both negative publicity and the absence of Lipitor ads. The interaction, β_4, captures the effect of the reduction in Crestor ads on Lipitor sales. In all specifications, we include product and DMA fixed effects and, following Bertrand, Mullainathan and Duflo (2004), cluster at the DMA level to address serial correlation. The results are in the second column of Table 4. The coefficients indicate that Lipitor sales are lower on average by 9.5% in DMA-months with political advertising outside the regulatory action. In addition, Lipitor sales are 8.7% lower during the regulatory action period, consistent with a lack of advertising and potentially negative publicity. Consistent with business stealing, β_4 is positive and statistically significant: by displacing Crestor ads, political advertising increases Lipitor sales during the regulatory action period.

4.4 Regression Results

We utilize the identifying variation generated by political advertising in a regression framework to obtain elasticities by estimating the following equation:

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

where X represents a vector of covariates as described in the regression tables and ad_{jtm} is the number of ads by firm j in month t and DMA m. In all specifications, we include product and DMA fixed effects and cluster at the DMA level. Because the specification is log-log, we can interpret the coefficients as elasticities. The level of analysis is the DMA-month-drug and we include each of the drugs advertised during our sample period from July 2007 through November 2008 in the relevant drug class: Lipitor, Crestor, Vytorin, and Zetia. The dependent variable is logged drug revenue per insured individual in the market.

Our measure of advertising is the average of this month's and the previous month's ad 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 (Iizuka and Jin (2007)). Furthermore, the need to obtain a prescription from a doctor is likely to delay sales after ad impressions. We control for product specific time trends in a number of different ways, including product-year fixed effects, product specific time trends, and flexible dummy variables constructed as all possible interactions between product dummies and dummies for before, during, and after what we describe as the "regulatory action period," which encompasses April through August of 2008.[28]

Table 5 shows the results of OLS specifications for advertised drugs. The first three regressions consistently show a small, but statistically significant and positive effect of DTCA on sales. Finally, in the fourth column, we include product-month-year fixed effects. Conditional on these fixed effects, which partial out the effect of national ads, variation in advertising is largely determined by local shocks driven by the political process. Therefore, ad levels are almost randomly assigned in this specification. The results are qualitatively different. The coefficients show a large elasticity of own advertising (.32) and significant business stealing (an elasticity of -.18).

We then turn to our IV estimates. In the first two columns of Table 6, we instrument for total (local and national) own and rival advertising levels using (i) the level of total (local and national) political ads, as well as second- and third-order polynomials of political ads, (ii) a dummy for the regulatory action that halted Lipitor advertising, and (iii) an interaction of this dummy with the polynomials of political advertising. We see that the OLS analysis underestimates the effects of own and rival advertising. The OLS own advertising effect in column 1 of Table 5 (.024) is less than 20% of the effect measured in the equivalent IV specification (.147). Similarly, we find substantial evidence of business stealing in the IV specifications that is absent from the OLS results. As discussed in Section 2.2, the direction of OLS bias is ambiguous; in this case the strategic interaction between firms leads to the effect of own advertising being biased downward, while the effect of rival advertising is biased upward.[29] In the second specification, we show that our results are robust to including more flexible time trends in drug sales. The results are similar in magnitude and we cannot reject that they are statistically the same.[30]

[28] We can also include a drug-specific linear trend as Appendix Figure 12 shows that there are important time trends during our sample period. 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 do not need to assume that the regulatory ban only affects drug sales through ads, and can allow for drug fixed effects that vary before, during, and after the regulatory action period.

[29] 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. However, our exogenous shocks are modest in size: for Lipitor, in the median market in terms of political ads, 16% of drug ads (9 ads) are displaced, while at the 90th percentile of markets, 30% of ads (18 ads) are displaced in the month of the largest shock. Given diminishing returns to adverting, we expect the effect of the marginal ads to be smaller than the impact of inframarginal ones.

[30] These results are larger than, but not statistically different from, our preferred IV specification. The appendix

In the final specification, we account for the end of the advertising campaign featuring Dr. Robert Jarvik and its potential direct effect on drug sales using fixed effects. It is possible that the pulling of these ads 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.[31] After Pfizer pulled their ads in response to regulatory scrutiny, they did not air any ads until a new campaign was ready in September 2008, as it took time and investment to develop a new campaign. According to officials within Pfizer quoted in the trade press, the delay was due to "creative brainstorming and testing" (Koroneos (2008)), rather than strategic concerns. In the last two columns of Table 6, we include the main effect of the regulatory action in both stages of the regression, but still interact the regulatory action with the level of political advertising and utilize the intensity of treatment across areas as a second source of variation. 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.[32]

In this specification, we allow for a flexible set of fixed effects constructed as all possible interactions between product dummies and dummies for before, during, and after what we describe as the "regulatory action period," which encompasses April through August of 2008. This flexible set of fixed effects partials out average sales of Lipitor (and the three other drugs) during each of July 2007-March 2008, April-August 2008, and September-November 2008. In addition to controlling for different time trends across drugs, this specification includes the effect of the regulatory action in both stages of the regression. Therefore, the regulatory action is allowed to have a differential effect on the sales of each drug directly.

We note that our instruments are still strong predictors of own and rival advertising, despite the fact that we no longer rely on the strong relationship between the regulatory action dummy and Lipitor ad levels.[33] The partial F-stat for the excluded instruments of our final, preferred specification is 51.6 for own advertising and 28.9 for rival advertising. In this specification, we find a positive and significant effect of own ads and a negative and significant effect of rival ads. The own-advertising elasticity estimate is 0.1395, which implies that a 10% increase in advertising

shows that the bias in the naive OLS is ambiguous.

[31] Furthermore, the results of the ECLIPSE trial, released concurrently, could have had a direct impact on Crestor and Lipitor sales in addition to increasing Crestor advertising.

[32] The partial F-statistics indicate that we still have a great deal of power. In columns 5 and 6, the main effect of political advertising is positive due to correlation in the time trends of Crestor sales and political advertising. If we extend the sample period through 2009, the main effect in the first stage of this specification is again negative. Market-specific deviations from the time trend in political advertising remain valid instruments for drug advertising.

[33] Despite this, we are not concerned about weak instruments (Rossi (2014)). The critical values for testing the hypothesis of joint weak instruments from Stock and Yogo (2005) are for models with i.i.d. standard errors, while we believe clustered standard errors are essential in our settings. Nonetheless, in unreported regressions using only first-order levels of political ads by product as instruments, we obtain a Cragg-Donald Wald F statistic of 15.505 with unclustered standard errors.

would yield a 1.4% increase in revenue. The rival ad elasticity is -0.1155. All of our specifications indicate that a firm's own advertising has a significant positive impact on sales, while rival advertising has a smaller negative effect on sales.

Our specifications thus far have pooled across drugs. However, elasticities may vary across firms. In order to explore potential heterogeneity in the effect, we separately estimate the effect for Crestor and Lipitor in Table 7. We note that in these specifications, "rival" ads still include Vytorin and Zetia by construction; own and rival ads are not collinear. In column 2, the results indicate larger elasticities (for both own- and rival- advertising) for these two drugs relative to the pooled specification in Table 6. Though baseline market shares differ, column 3 shows that the own elasticity is particularly large for Crestor, while the rival elasticity is particularly large for Lipitor; we cannot reject the hypothesis that the elasticities are the same. Our results indicate that the business stealing nature of advertising has a significant effect on sales. Absent the effect of rival advertising but holding all else constant, Crestor sales would likely rise by approximately a third; the effect for Lipitor would be even larger. In Section 5, we will explore this heterogeneity further by adding structure that allows us to think about differential effects of ads on consumer behavior, focusing on Crestor and Lipitor while collapsing Vytorin and Zetia into the composite good.

Table 8 estimates the spillover effects for non-advertised drugs. Column 2 replicates the last specification in Table 6 in which two-month moving averages of drug advertising are the independent variables of interest. Column 1 presents the equivalent OLS specification. In the OLS specifications, we find no effect of rival advertising. Once we instrument for advertising, we find evidence that advertising has a positive spillover effect. A 10% increase in advertising for the class leads to a 0.19% increase in sales of non-advertised drugs. Columns 3 and 4 show the net effect of all category advertising on category sales. Overall, advertising expands the statin market: a 10% increase in advertising for the class leads to a 0.16% increase in category sales. Our results support a model in which advertising has business-stealing effects, but also positive spillovers to non-advertised drugs.

4.5 Robustness Checks

We believe our preferred specification flexibly controls for trends in drug demand over time, and allows for a sufficient lag between advertising impressions and the realization of demand, as consumers must obtain a prescription before purchasing a statin. Appendix Table 16 explores this timing assumption. The results show a similar pattern for contemporaneous, 2-month trailing average, and 3-month trailing average specifications, with some attenuation as the window expands. We also show a similar effect using lagged advertising values and when including a stock of adver-

tising as a control. In addition, we show that our estimates are stable when adding product-DMA fixed effects or if the outcome of interest is quantity (market share) instead of revenue. In all specifications, we cluster at the DMA level.

In Appendix 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 hypothesis, 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 than the causal effect measured in columns 2 and 4. Both own advertising and rival advertising are endogenous and the outcome of dynamic game; our identification strategy allows us to capture both effects.

Finally, several robustness checks in Appendix Table 18 allow us to show that the effect is similar even if we restrict to markets and time periods that are less likely to have a predictably high amount of political advertising. In column 2, we exclude December 2007 through February 2008. If one thought that both primaries would conclude after "Super Tuesday," when a number of large states hold contests, then this excludes the predictable portion of the primary period; the results are quite similar to our preferred specification. In column 3, we exclude the traditional swing states of Ohio and Florida, which were pivotal in the 2004 and 2000 elections, respectively. Again, we obtain similar results.

4.6 Part D Sample

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 the second column of Table 9, the own advertising elasticity is 0.1310 for the two-month trailing average, while the estimate from the employer-sponsored sample was 0.1395. 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. 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 DMA-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 the last two columns of Table 9. There are two key observations. First, the own advertising elasticity is nearly three times as large in magnitude for new consumers (0.3710 versus 0.1310 for the entire sample). Second, the rival elasticities are larger in magnitude among new consumers as well (0.211 vs. 0.0234 for the entire sample).[34] We conclude that the effect is largely being driven by new consumers, rather than switchers; we account for this feature of advertising in the demand model and policy counterfactuals in the next section.

5 Consumer Demand and Counterfactual Analysis

Our reduced form results show that among advertised drugs, a firm's advertising causally increases its own sales, while rival advertising decreases sales. We also find evidence of positive spillovers to non-advertised drugs from such advertising. Micro-level data demonstrate that these effects are concentrated among new consumers. However, there are several margins on which consumers may respond to advertising. They could be more likely to see a physician (Jin and Iizuka (2005)), which may increase prescriptions of all drugs. Consumers may also ask for specific drugs by name, increasing the probability of receiving the advertised drug and simultaneously driving our business stealing results. Finally, ads could affect adherence to existing prescription drug regimens (Wosinska (2005)), though our reduced form results indicate this effect is likely to be small in magnitude.[35]

In this section, we examine the effect of a ban on DTCA using a discrete choice model of drug consumption. Our model builds on existing models in the literature that explore state dependence in markets for consumer packaged goods (i.e. Dubé, Hitsch and Rossi (2010)) and, more recently,

[34] While we compare elasticities, we note that the levels are very different: the sample of "new customers" is relatively small.

[35] These results have important implications for both firms and policymakers. Firms can increase sales through advertising, but must consider the actions of rival firms as well. Furthermore, the existence of positive spillovers implies that there may be substantial social benefits of advertising. However, the presence of business stealing indicates that some ads may be socially wasteful. This tension is echoed in the policy debate surrounding DTCA among both physicians and policymakers. The American Medical Association (AMA) has called for a ban on DTCA, while the Hillary Clinton presidential campaign has advocated eliminating favorable tax status for marketing expenditures.

financial products such as health insurance (Handel (2013); Polyakova (2015); Abaluck and Gruber (2016)). The model is meant to capture demand patterns rather than welfare-relevant primitives of the consumer or physician utility function. It is flexible enough to allow past behavior to alter the effect of advertising in addition to allowing the affect of advertising to vary by drug. Estimation relies on the same plausibly exogenous variation utilized in our reduced form approach. We combine data from Medstat, which measures the impact among the privately insured, and Medicare Part D, which allows us to disentangle the effects on new and existing consumers. The model is tractable, yet flexible; by utilizing a nesting structure, we can capture the similarity among statin regimens (as opposed to the outside good) and branded statins (as opposed to generics). Importantly, the model estimates can be used to simulate the effects of a DTCA ban on new statin prescriptions and across drugs. After measuring the impact of a ban on sales, we quantify the health impacts and social value of advertising in our setting.

5.1 Model

Consider a set of consumers who make a consumption decision at the beginning of each month. For each consumer, last period's consumption choice j directly affects this period's choice k. The consumer chooses among four consumption states; they can choose not to begin or continue a statin regimen (the outside good, denoted by o). Among the set of inside goods (denoted by d), the consumer can choose a non-advertised statin (all non-advertised statins are collapsed into a single composite good, denoted n) or among the set of advertised drugs (denoted by a); among advertised drugs, the consumer can choose Crestor (c) or Lipitor (l).[36] Following Table 7, we separately estimate advertising effects for Crestor and Lipitor and include Vytorin and Zetia in the non-advertised composite good in these specifications for simplicity; we allow the advertising of these goods to affect choice probabilities by including their ads in our measure of rival advertising. As shown in Figure 3, Vytorin and Zetia dramatically scale back advertising in February of 2008 and cease advertising entirely by June 2008, so they are not central to this exercise. Because parameters vary with previous consumption, we write out separate probabilities as a function of lagged choice. Choice probabilities are estimated using a nested logit model with these two levels of nests: the inside and outside good, and among the inside good, advertised and non-advertised drugs.

Choice probabilities in the nested logit model are parameterized as functions of δ corresponding to each possible choice. We form δ_{jkt} for each product k in period t conditional on previous consumption of product j in period $t-1$ as a function of flexible fixed effects and, for some tran-

[36] We include Vytorin and Zetia among the non-advertised statins given their small market share; they represent less than ten percent of such sales in 2008.

sition probabilities, own and rival advertising:

$$\delta_{jkt} = \beta_0^{jk} + \beta_1^{jk} log\,(1 + ad_{kt}) + \beta_2^{jk} log\,(1 + ad_{-kt}) + \xi_{kt}.$$

We normalize $\delta_{jo} = 0$ for the outside good for all j, and estimate fixed effects β_0^{jk} for the remaining probabilities. This specification is flexible enough to allow ads to affect both the probability that a new consumer chooses the advertised statin (through the effect of own advertising) and the probability that the new consumer chooses a different statin (through the effect of rival advertising). This parameterization, described fully in Table 10, allows the effect of advertising to vary by drug and flexibly captures substitution patterns across drugs.[37] Since this is a discrete choice framework, any positive effect of a firm's own advertising mechanically implies a lower probability of consuming a rival drug; therefore, the parameters on rival advertising for branded drugs could be zero or even positive while still implying business-stealing.

We construct a share transition matrix described in Table 10 by calculating choice probabilities conditional on each of the previous consumption states. Consequently, each row of this matrix represents a nested logit discrete choice model for the choice of consumers in that particular consumption state in the previous period. Each column is used to construct the market share of each choice in the current period by summing across the previous consumption states and their transition probabilities. That is, if market share for good j in time t is given by s_{jt} and the probability of switching from good j to k is P_{jk}, then $s_{j,t+1} = \Sigma_{i\in\{o,n,c,l\}} P_{ij} s_{it}$. Following Train (2009), letting d subscripts represent the inside goods and suppressing time and lagged choice notation, the probability of choosing the outside good is given by:

$$\frac{1}{1 + e^{\lambda_d I_d}},$$

where λ_d determines the correlation of unobserved shocks in the inside nest and the inclusive value of the inside good is given by $I_d = ln\left(e^{\delta_n/\lambda_d} + e^{I_a/\lambda_d}\right)$.

We let a subscripts represent the nest of advertised drugs. The probability of choosing an non-advertised drug is the product of choosing the inside good multiplied by the conditional probability of choosing a non-advertised drug, or $P_d \cdot P_{n|d}$:

$$\left(1 - \frac{1}{1 + e^{\lambda_d I_d}}\right) \cdot \frac{e^{\delta_n/\lambda_d}}{e^{\delta_n/\lambda_d} + e^{\lambda_a I_a/\lambda_d}},$$

where λ_a determines the correlation of unobserved shocks among the alternatives in the adver-

[37] We do not estimate all advertising parameters in all probabilities: we allow for own and rival effects on the extensive margin and for adherence effects of own and rival advertising among consumers currently taking a statin.

tised nest and $I_a = ln\left(\sum_{k\in\{c,l\}} e^{\delta_k/\lambda_a}\right)$. The probability of choosing Crestor or Lipitor is similarly written as the product of conditional probabilities , $P_d \cdot P_{a|d} \cdot P_{j|a}$, where

$$P_{j|a} = \frac{e^{\delta_j/\lambda_a}}{\sum_{k\in\{c,l\}} e^{\delta_k/\lambda_a}}.$$

Therefore, the probability of choosing Crestor or Lipitor ($k = c$ and $k = l$ respectively) is given by:

$$\left(1 - \frac{1}{1+e^{\lambda_d I_d}}\right) \cdot \left(1 - \frac{e^{\delta_n/\lambda_d}}{e^{\delta_n/\lambda_d} + e^{\lambda_a I_a/\lambda_d}}\right) \cdot \frac{e^{\delta_j/\lambda_a}}{\sum_{k\in\{c,l\}} e^{\delta_k/\lambda_a}}.$$

These probabilities are consistent with a myopic consumer i who consumed j last period solving, in period t,

$$\max_k \delta_{jkt} + \varepsilon_{ijkt}.$$

As λ_d approaches 1, there is less substitution between statins, and as λ_a approaches 1, there is less substitution between advertised drugs; these parameters govern the correlation in the nested logit error term, ε_{ijkt} (distributed generalized extreme value). We note three key features of this specification. First, advertising affects choice probabilities in a way that mirrors the reduced form specification, but is allowed to vary across drugs. Second, by allowing fixed effects to vary across both initial consumption states and drugs, we can separately capture the effect of adherence and the propensity to begin a statin regimen. Finally, we allow these fixed effects to capture any price effects, as we do not observe product-specific price variation over time or across markets.

5.2 Estimation

We estimate the parameters of interest via the generalized method of moments (GMM) using two sets of moments. First, we observe the market shares of all states for every DMA-month in our data. Therefore, we solve for a month-DMA vector of $\xi = [\xi_{Crestor}, \xi_{Lipitor}, \xi_{Other}]$ such that the observed shares in the subsequent month match the predicted values. We form moment conditions $E[\xi|\mathbf{z}] = 0$ for a vector of instruments $\mathbf{z}$, which include the same third-order polynomial of political advertising at the DMA-month level, as well as interactions with the regulatory action dummy variable, to primarily identify the advertising parameters. To identify the nesting parameters, we augment our set of instruments to also include (i) the number of primary care physicians per capita at the DMA level, and (ii) the rate of managed care penetration at the DMA level.[38] The first shifts the inside good share, as it affects the supply of physicians who can see patients and write

[38]Managed care penetration is measured as the market share of Medicare Advantage plans. Medicare Advantage plans are administered by private managed care organizations and provide coverage for a subset of Medicare enrollees. Managed care plans use supply side controls, including capitated payments to physicians, to control medical costs.

prescriptions. The second affects the branded drugs share, as physicians who see more managed care patients are likely to prescribe more generics, and this may spill over from those patients to other patients as well. The nesting parameters are constrained to be the same across each prior consumption state.

Second, we construct moments of the difference between actual annual switching rates among the four consumption states from the Medicare Part D micro-data and the annual switching rates that would be implied by our estimated monthly transition matrix for each market. This allows us to distinguish the level of switching that occurs among states; market shares alone could be justified by different levels of switching. We use annual switching rates to limit the noise from variation in the days supplied in any given prescription and to assure that we are capturing true transitions, and these moments allow us to pin down the β_0 constants in each transition probability with high precision. While the "micro-moments" are constructed at the DMA market-year level, all other moments are constructed at the DMA market-month level.

This estimation strategy is tailored to our empirical context and question. The estimates are not meant to represent primitive preferences, but to provide evidence on how statin demand is affected by advertising over time. We do not attempt to identify the source of any inertia in brand choice (Dubé, Hitsch and Manchanda, 2005), be it structural or spurious state dependence. Rather, we assume that these factors are invariant to the policy we study: a ban on DTCA. Furthermore, the outcomes we study – sales, health outcomes and costs – do not require us to take a stand on the welfare effects of advertising itself.

5.3 Results

Table 11 presents the model estimates. The first four panels describe estimates from each of the four nested logit models corresponding to a "from" state, while the fifth contains the nesting parameters. In the upper four panels, the first column reports the constant terms for each state (Crestor, Lipitor, non-advertised other statins, and the outside good). The estimates indicate a great deal of persistence in choice, as the fixed effects are largest within a panel for the current consumption state (i.e. when $j = k$). In the first three panels, the second column shows a negligible effect on adherence, as measured in the own and rival advertising parameters. Category level advertising also has a minimal impact on adherence within non-advertised drugs. The largest effects are on the extensive margin, as highlighted in the fourth panel: advertising has a big impact among "new" consumers previously consuming the outside good. Consistent with the reduced form results, we find that an increase in own advertising increases the probability that a new consumer begins taking a drug ($\beta_1^{oc} > 0, \beta_1^{ol} > 0$) .[39] In addition to this indirect business stealing effect, we also find

[39] By construction, this implies that own advertising decreases that probability that a new consumer begins taking a rival drug.

that rival advertising directly decreases the probability that a new consumer begins taking the focal advertised drug ($\beta_2^{oc} < 0$, $\beta_2^{ol} < 0$). Finally, $\beta_2^{on} > 0$ indicates that there are positive spillover effects on non-advertised drugs in addition to the business stealing effects among branded drugs. The bottom panel presents the nesting parameters, which indicate that consumers are more likely to substitute among statins than between statins and the outside good.

Our results are consistent with the reduced form estimates and also highlight additional features of demand for statins and the impact of advertising. First, consistent with our Medicare Part D results, we find that the vast majority of the quantity effect is driven by new consumers, with much smaller impacts on adherence rates. We note our estimates imply a great deal of persistence in drug demand, as measured by β_0^{jj} and shown in Appendix Table 19, though we do not take a stand on what drives this feature of the market.

Finally, the model also allows the effect of advertising to vary by drug. To put these estimates into context, we begin by showing the marginal effect of one additional ad for either Crestor or Lipitor in all DMAs in July 2007 on the number of consumers in the different consumption states throughout the remainder of our sample period. Figure 9 shows that a marginal ad in every DMA at the start of our sample period induces 40 (45) additional customers to initiate a Lipitor (Crestor) regimen across all DMAs and leads to 17 (12) fewer consumers initiating a Crestor (Lipitor) regimen. While the reduced form own ad revenue elasticity is larger for Crestor, the level effects are similar given Crestor's lower baseline sales. The positive effect for the advertiser dissipates as those new customers slowly switch to non-advertised drugs or discontinue their statin regimen. In addition, we see that ads are responsible for business stealing among advertisers; the magnitude of the business stealing results are similar across drugs. These results imply reasonable returns on advertising.[40]

5.4 The Effect of a Ban

We next consider a counterfactual ban on advertising in 2008 and measure the evolution of consumption shares throughout the year. We evaluate the policy by using the estimated transition matrix, recovered DMA-month shocks (ξ_{jt}), and advertising levels set to zero. We compare the outcomes to the baseline outcomes simulated at our estimates with observed advertising levels. Figure 9 shows the effect of halting all advertising in January 2008 on monthly shares of all drugs

[40] A single national ad costs $93,000, and we estimate that such an ad induces 40 (45) additional consumers to begin a Crestor (Lipitor) regimen. Given a 3% probability of discontinuing the statin regimen each month estimated in Table 19, the firm can expect the average new consumer to generate 33 months of revenue. Given an average price of $90 per consumer-month, this implies that an ad generates approximately $120,000 ($130,000) of marginal revenue. The OLS results would imply that the costs of advertising greatly exceed the benefits. We note that the regulatory action is a large shock to Lipitor advertising levels, and we estimate a constant elasticity. To the extent that there are diminishing returns to advertising, inframarginal effects are likely to be larger.

in the Medstat sample for the rest of the year.[41] The solid line shows the baseline estimates at observed advertising levels, and the dashed lines are the counterfactual with all advertising set to zero. The share of consumers taking any statin slowly falls, as does the share taking the non-advertised statins.

A ban decreases overall category demand, with the effects concentrated among new consumers and within non-advertised drugs. Overall, 636,500 fewer consumers initiate a statin regimen in 2008 under a ban (representing approximately a 2% decrease in total patients), leading to 3.9 million fewer patient-months of revenue for manufacturers.[42] New consumers are responsible for 87% of this revenue reduction.[43] Among the largest advertisers, the effect of the ban is mixed. Note that the ban has a differential effect during the regulatory action period: only Crestor's ads are removed, as Lipitor's advertising had ceased. The ban has a small net effect on Crestor sales, as the main effect and business stealing effects approximately cancel out. The effect of a ban on Lipitor sales is positive during the regulatory action period and negative during the rest of the sample period; banning Crestor ads during the period in which Lipitor did not advertise is unequivocally good for Lipitor. While incentives generated by the business stealing nature of ads may determine the level of advertising, most of the decrease in consumption under a ban is driven by non-advertised drugs.

The relative importance of non-advertised drugs is due to two factors. First, all advertising has a positive impact on the consumption of non-advertised drugs. Second, while the reduced form spillover elasticities are smaller in magnitude than own- and rival-effects for Crestor and Lipitor, these results represent revenue elasticities. Non-advertised drugs are substantially cheaper than advertised drugs and their market share is larger; as a result, the level effect on the number of consumers initiating a statin regimen and months of consumption (pills) is larger. We next turn to calculating the impact of increased consumption.

Our results – when combined with external estimates – indicate that a demand expansion of this size (636,500 additional new consumers initiating a statin regimen) would avert approximately 7500 heart attacks.[44] Given potentially large health effects, we next compare the cost of advertising

[41] As the Medstat sample represents only a subset of the total market, below we scale up the effects when discussing levels to reflect a parallel impact among the other patient populations in the US.

[42] We use our estimates to simulate differences in shares by market of consumers beginning a statin regimen as well as total individual prescription fills. In order to obtain a total demand increase relevant for both policymakers and firms, we aggregate these shares to the national level. We then rescale to the total population enrolled in Medicare and employer-sponsored insurance, noting that the reduced-form elasticities we obtain are similar across these consumer groups.

[43] This implies that each consumer initiating therapy accounting for 5.3 months of revenue during 2008, slightly less than expected under uniform take-up over the course of the year due to imperfect adherence. Under perfect adherence and uniform take-up, the average consumer would take a statin for 6 months.

[44] Our estimates of clinical effectiveness come from NICE, who estimate that statin use reduces non-fatal heart attacks by 22 per 1000 for patients with chronic conditions and 7 per 1000 for patients without chronic conditions; we conservatively assume that only 1/3 of the Medicare population has a chronic condition. The average cost of such

with benefits using results from Stomberg et al. (2016), which studies a similar expansion in statin consumption (specifically, making the drugs available over-the-counter). We follow their approach of examining outcomes associated with consumers initiating statin therapy and measure the health benefits of expanded statin consumption in terms of quality-adjusted life years (QALYs); 1 QALY equates to a year in perfect health. Armed with a measure of benefits, they calculate the "cost-per-QALY," known as an incremental cost-effectiveness ratio (ICER). They calculate an ICER associated with an expansion in statin demand of $5667 per QALY. Estimates within the literature value a year of life in perfect health at $75,000 to $100,000 Cutler (2004); given this, statins represent an extremely cost effective intervention.[45]

In order to obtain an ICER in our setting – for statins inclusive of advertising costs – we begin with the benefits of Stomberg et al. (2016), which include reductions in cardiovascular events such as heart attacks, and (non-advertising) costs, which include the costs of the drugs themselves as well as physician visits and side effects. We then add the cost of a year of advertising ($135M in 2008), spread over the number of additional consumers who begin a statin regimen due to DTCA. Specifically, in our setting, we compute the ICER as equal to the sum of non-ad costs and ad costs per consumer initiating statin therapy divided by the number of QALYs generated per consumer initiating statin therapy:

$$ICER = 5667 + \frac{\text{DTCA Spending}}{\text{Number of Consumers Initiating Therapy due to DTCA}} / \text{QALYs generated per consumer initiating therapy},$$

where DTCA spending is $135M, 636,500 additional consumers initiate therapy due to DTCA, and, given the Stomberg et al. (2016) estimates, 0.22 QALYs are generated per consumer initiating therapy.[46] Because "DTCA Spending" is denominated in dollars and we divide through by a measure of QALYs per new consumer, the second term above is a measure of ad dollars per QALY generated. When combined with $5667 in non-advertising spending per QALY, we obtain a measure of total incremental spending per QALY generated due to DTCA of $6631. In other words, $6631 in spending on statin DTCA generates one more QALY, which is well within the bounds of what is considered an effective health intervention.

a health event to the Medicare program alone was $43,120 in 2008 (Likosky et al. (2013)), implying a fiscal savings from non-fatal heart attacks averted of $323M, more than the cost of the ads themselves. Given that a great deal of medical spending occurs within the last six months of life, we want to avoid complications associated with attributing spending to the cardiovascular even that could have been avoided with statin use.

[45] The National Institute for Health and Clinical Excellence (NICE) in the United Kingdom has adopted a cost effectiveness threshold range for approved interventions of £20,000 (approximately €29,500 or $40,000 in 2007) to £30,000 per quality adjusted life year (QALY) gained (Appleby, Devlin and Parkin (2007)).

[46] We use our estimates to simulate differences in shares by market of consumers beginning a statin regimen as well as individual prescriptions. In order to obtain a total demand increase relevant for both policymakers and firms, we aggregate these shares to the national level. We then rescale to the total population enrolled in Medicare and employer-sponsored insurance, noting that the elasticities we obtain are similar across these consumer groups.

Our results show that DTCA of statins can generate substantial social value; however, advertising less cost-effective drugs or those with substantial side effects may not generate substantial benefits. Despite this, we note that the benefits of increasing statin use likely exceed the direct cost of *all* DTCA in 2008. To see this, we can replace the statin ad spend ($135 million) with total DTCA spend for all prescription drugs ($4.4 billion) and obtain an ICER of approximately $37,000 (Ventola (2011)).[47]

Our calculations indicate that DTCA spending would be well within the range of interventions that would be considered cost effective. Because the largest costs in this calculation are the costs of advertising itself, the results are robust to the precise assumptions about drug costs, the appropriateness of treatment for marginal patients, or the value of a QALY.[48] In addition, we only consider the health benefits to new patients beginning a statin regimen (from the outside option), leading to a purposefully conservative estimate. While our calculations imply that the value of DTCA exceeds its cost, we do not claim it is the optimal or lowest cost means of expanding the use of high value medical interventions. In addition, the population of "compliers" who begin a statin regimen due to an advertising campaign may be different than the average population. We explore the potential for heterogeneity in Appendix Table 20, and find no evidence of differences across high and low advertising markets in observable demographic characteristics of consumers ever taking a statin or those beginning a statin regimen in 2008. The marginal consumer is observationally similar to the average consumer; advertising increases sales among consumers who are equally appropriate for treatment based on observables.

Finally, while our estimates examine the impact of a ban on consumer utilization and, ultimately, health care spending, firms could adjust along other margins. Specifically, we consider the impact of a ban on other marketing channels and pricing.

[47] While this calculation assumes that there is no harm associated with increased consumption of other advertised drugs or harms associated with lower match quality within a class, the Federal Drug Administration (FDA) is tasked with assuring that drugs are safe and efficacious and such effects would have to be substantial to overturn our basic finding. Some would argue that the FDA is overly conservative in some drug categories (see Montazerhodjat and Lo (2015) for additional discussion), but this issue is outside the scope of this paper. We also assume that DTCA does not negatively affect consumers by steering them to lower quality drugs within a class, which is of little concern in our setting. Given our large potential gains from advertising, we think this effect is unlikely to negate the benefits of advertising.This exercise is similar in spirit to Cutler's argument that the increase in medical spending since 1950 has been roughly offset by just the value associated with treatments for cardiovascular disease and low birth-weight babies; while additional spending on the margin may or may not be socially valuable, DTCA is valuable on average (Cutler (2004)).

[48] Stomberg et al. (2016) assume a cost of $25.42 per thirty day supply; even if the true cost was twice this amount, the break even demand increase is still only 10% of the actual demand increase. Stomberg et al. (2016) assume that 85% of marginal patients are appropriate for treatment and that the cost of treatment for inappropriate patients is $3551 per year. Given these numbers, advertising is still cost effective if 50% of marginal patients are inappropriate for treatment. Finally, we have used extremely conservative numbers for the valuation of a QALY. Choosing to include or exclude monitoring costs, such as cholesterol testing has little impact on the overall calculation. This is where our calculation may differ the most from the Stromberg et al. (2016) calculation. In our setting, monitoring costs include physician visits and should more clearly be included in the overall calculation.

First, firms could increase detailing to physicians in response to a ban. In the short- to medium-run, this is unlikely. Shapiro (2016) shows that detailing levels and DTCA are uncorrelated geographically and, furthermore, that there was not a large increase in detailing in response to the 1997 regulatory changes that drove increased DTCA. Alpert, Lakdawalla and Sood (2015) confirm this intuition in the post-2006 time period (after the Part D expansion). Our own analysis shows that detailing is not responsive to the political cycle, even during later elections with higher and more predictable ad spending.[49]

Second, drug prices may be affected by a DTCA ban. Over the short- to medium- run, insurance contracts are fixed and drug prices are negotiated nationally between payers (largely insurers) and manufacturers, making price adjustments less likely. Furthermore, unlike consumers, insurers are likely already aware of substitutes within a therapeutic class and are sophisticated buyers unlikely to be affected by advertising directly. Therefore, advertising is unlikely to affect upstream prices; to support this intuition, we show in Appendix Table 20 that retail prices and out-of-pocket costs to consumers do not depend on the level of advertising in the cross-section.[50] As before, we split the sample into low and high advertising markets. Retail prices and out-of-pocket costs (for all drugs and statins specifically) are not statistically different across these DMAs.[51] We conclude that retail prices and out-of-pocket costs are not correlated with advertising across geographic areas in our data. Modeling the bargaining game between manufacturers and insurers that determines prices in this setting is outside the scope of this paper, but is an important avenue for future research.

5.5 Discussion

We find that the presence of DTCA expands the market for statins despite the business stealing effects of ads among branded drugs. 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 Shapiro (2016)). Our results are consistent with these studies in finding spillovers to non-advertised drugs.[52] Here, in addition, we argue that despite the fact that substan-

[49] Furthermore, detailing largely has a business stealing rather than market expanding effect; it is unlikely to provide the same social benefits we estimate for DTCA. In addition, by a revealed preference argument, increased detailing in place of DTCA would be unlikely to a private benefit nearly as large as the status quo. Finally, increased detailing may exacerbate any agency issues.

[50] If anything, out-of-pocket costs are slightly lower in high advertising areas once we condition on statin consumption, though the differences are small in magnitude and economically insignificant. This also implies that we have limited variation to identify price responsiveness.

[51] For all drugs, out-of-pocket costs are slightly smaller in high advertising markets, consistent with more advertising in places with more insurance (Alpert, Lakdawalla and Sood (2015)).

[52] Our setting is also one in which there is little switching between drugs, allowing us to cleanly isolate the spillover effect of advertising on initial prescriptions. However, we estimate smaller spillover effects than Shapiro (2016).

tial advertising expenditure appears defensive, eliminating DTCA would significantly reduce the number of patients taking an effective, safe drug.

While our results present a consistent story, there are a number of caveats. First, we do not consider selection into insurance plans or explore the role of physician agency. Given that we are looking at short-run shocks, we do not believe these factors bias our results. Second, all of our results take the decision to advertise a drug at all as given. This decision is non-random, and our treatment effects need not generalize. Third, our elasticities are local average treatment effects and specific to the market we study, which has a limited number of advertisers who are close clinical substitutes. However, the key dynamic that drives our simulation results – the net effect of own and rival ads is small among branded drugs, yet these ads create spillovers to non-advertised drugs – may be important in other drug classes as well, including antidepressants (Shapiro (2016)) and erectile dysfunction drugs (as Viagra will face generic competitors in 2017). A final caution is that – as discussed above – these are partial equilibrium calculations. Future work should explore additional strategic decisions, including formulary placement and detailing, dynamic effects, and heterogeneity both within and across classes.[53]

Our identification strategy is likely to be useful in a number of product markets, including other drug classes; additional variation will be necessary to separately identify the impact of rival advertising. 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. However, we note that the health gains from statin advertising in 2008 plausibly exceed the entire direct spending on DTCA during the same period.

6 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; however, we also document strong business-stealing effects among advertised drugs, and an economically significant spillover to non-advertised drugs. We estimate the effect in two samples: among the privately insured and among Medicare beneficiaries. In the Medicare sample, we show that the effect is primarily driven by new prescriptions.

We model consumer demand for statins in order to quantify the effect of a ban on drug consumption in 2008. We find that a ban would have had a meaningful effect on the number of con-

[53] We also do not consider strategic entry by generic manufacturers, as described in Scott Morton (1999).

sumers initiating statin regimens, particularly with non-advertised drugs. Our results help quantify the trade-offs that policy makers may face when regulating pharmaceutical firms: increases in information versus wasteful advertising. Given the reduced form business stealing results, one might conclude that additional regulation of drug advertising in the United States may be welfare enhancing. However, the overall category expansion effect implies that a ban on DTCA would reduce overall statin sales, eliminating the large benefits to patients they convey. Using established cost-effectiveness standards, we find that spending on statin advertising can be justified by the benefits of the drugs. Therefore, policies aimed at reducing or eliminating DTCA have the potential to reduce social surplus.

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Figures

Figure 1: Statin Market Timeline

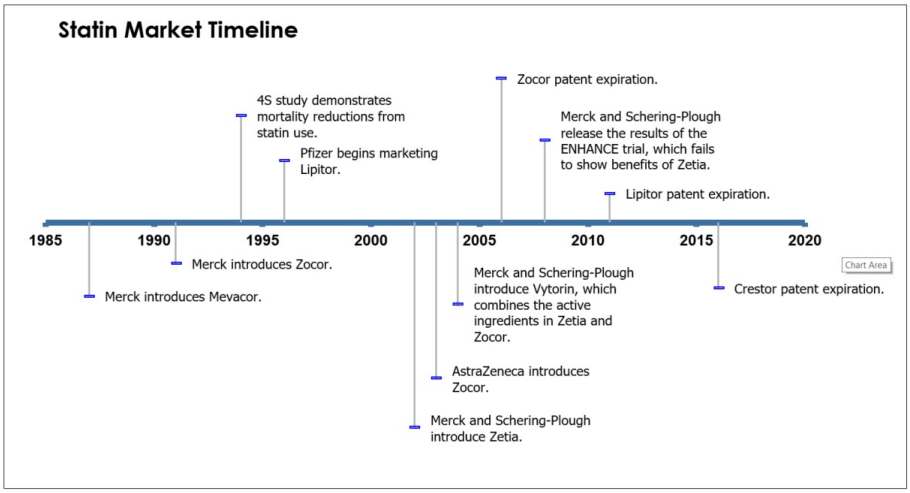
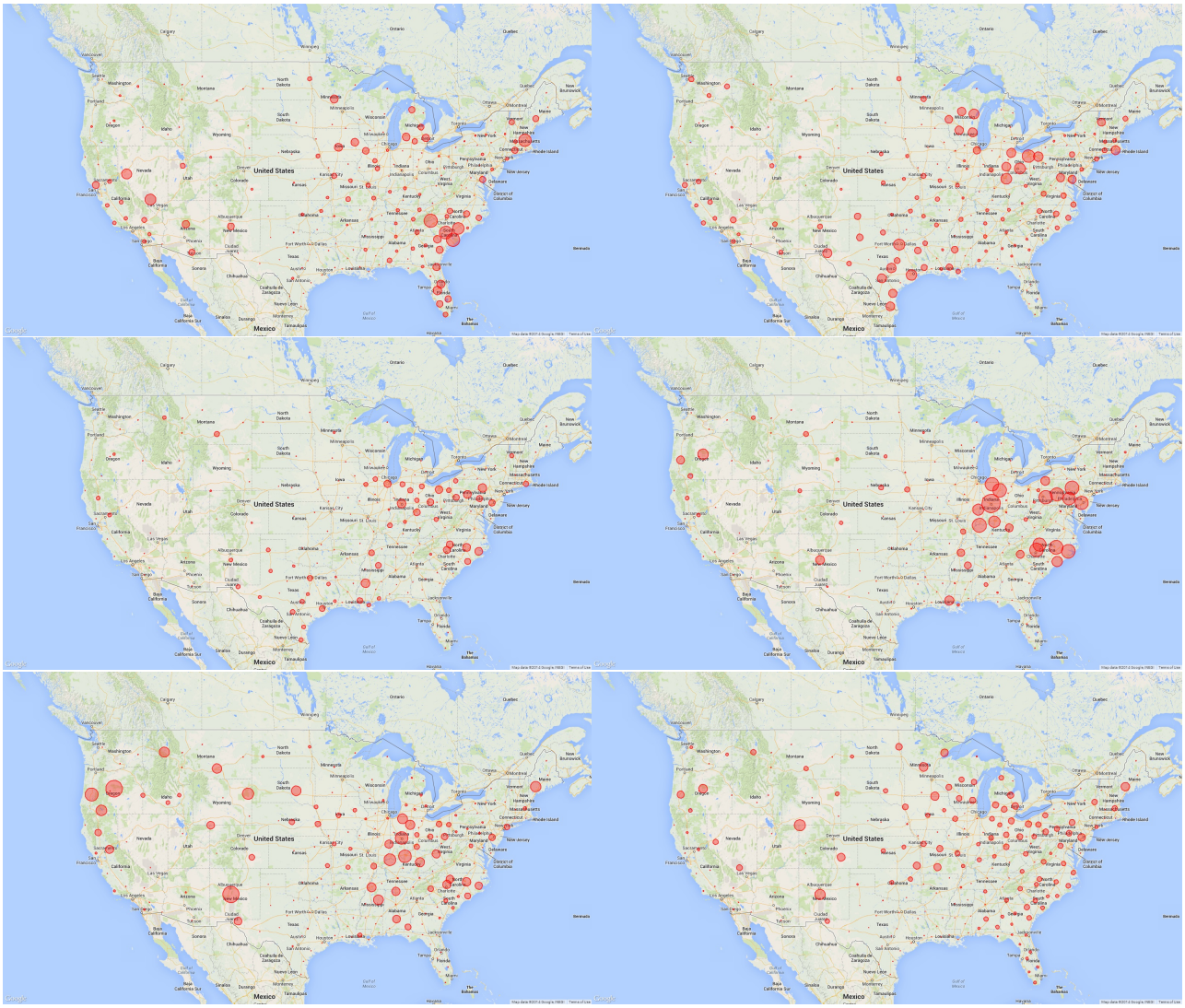
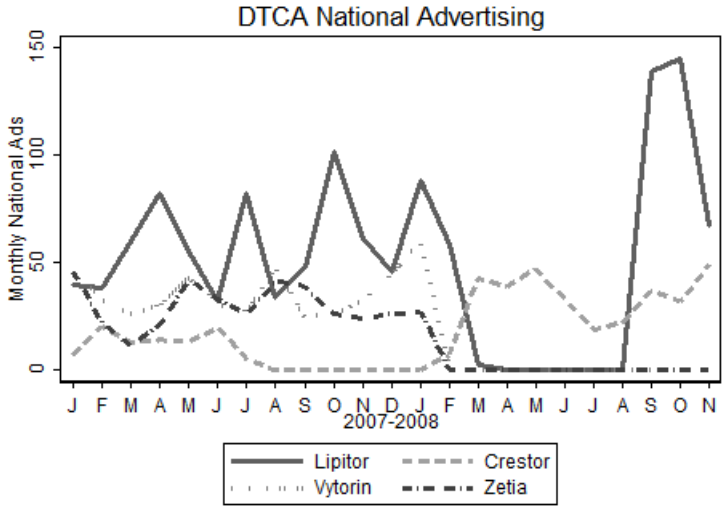


Figure 2: Political Ad Levels, January-June 2008



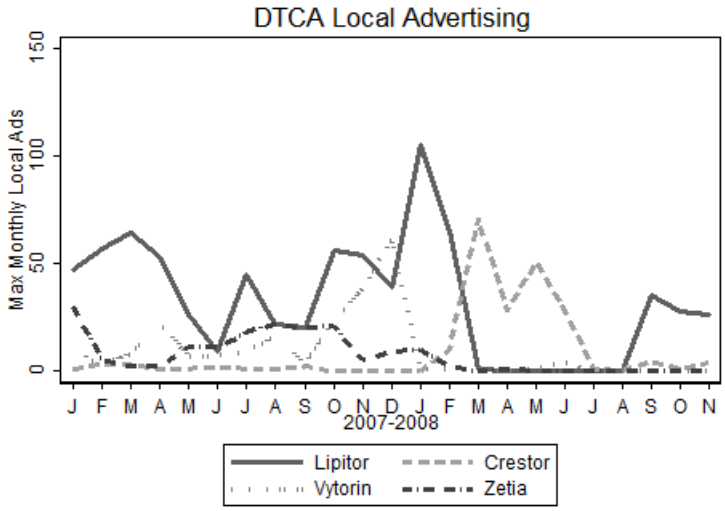
Notes: The above maps show a circle for each DMA in the USA. The diameter of each circle is proportional to the number of political ads aired in that market, in that month, for all races (Presidential, Senatorial, House, Gubernatorial). The first row are January and February; second row are March and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

Figure 3: National Pharmaceutical Ad Levels for Statins



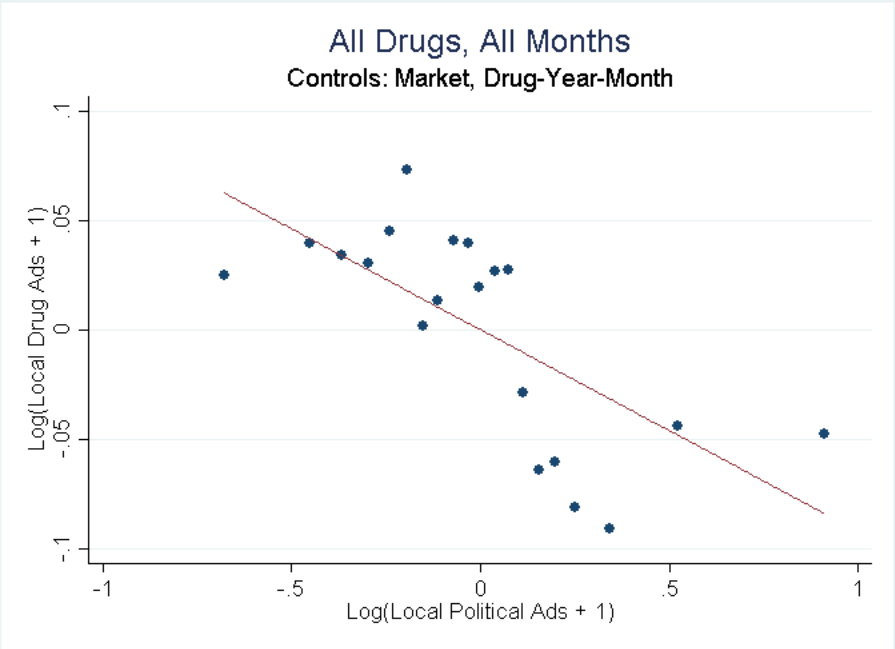
Notes: The above graphic plots national advertising spots from the Kantar data. Data spans January 2007-November 2008.

Figure 4: Variation in Local Pharmaceutical Ad Levels for Statins



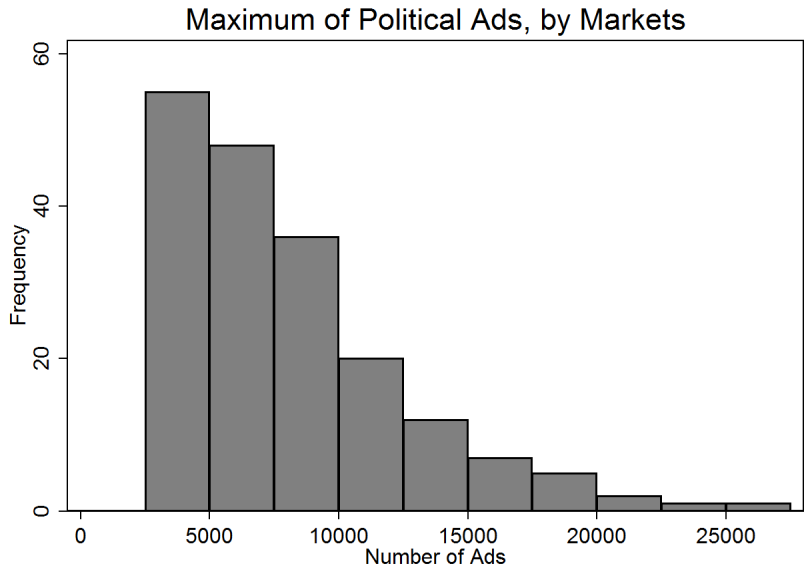
Notes: The above graphic plots the maximum of local advertising spots across DMAs from the Kantar data. Data spans January 2007-November 2008.

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



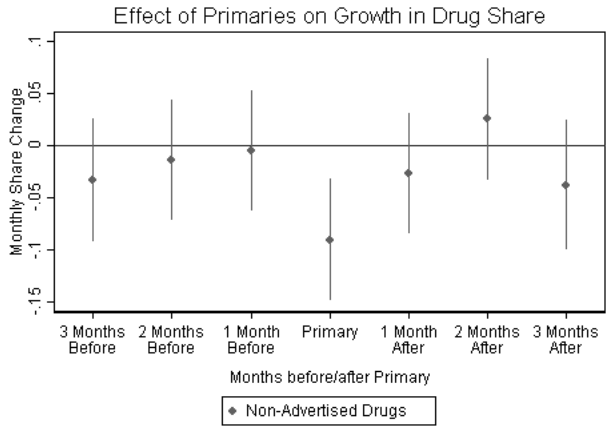
Notes: The above plots bins of observations from July 2007 to November 2008 at the market-month level after residualizing by market and drug-year-month fixed effects. A "market" is defined as a DMA; we utilize data from 189 DMAs and bin them into twenty groups. The plot uses local drug ads, although the plot that also includes national is identical due to the drug-year-month fixed effects. Twenty bins are used. The fitted line is based on a regression of all underlying data, not only the binned values.

Figure 6: Histogram of Political Ads by Market



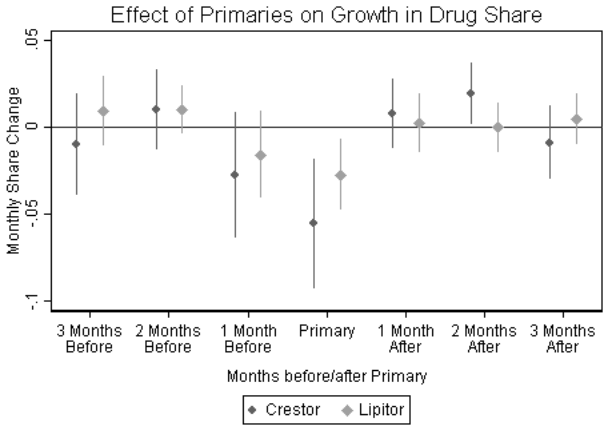
Notes: The above plots bins a histogram of the maximum number of political ads in a market over any individual month in the course of our sample period.

Figure 7: Effect of Primary Timing on Non-Advertised Statins



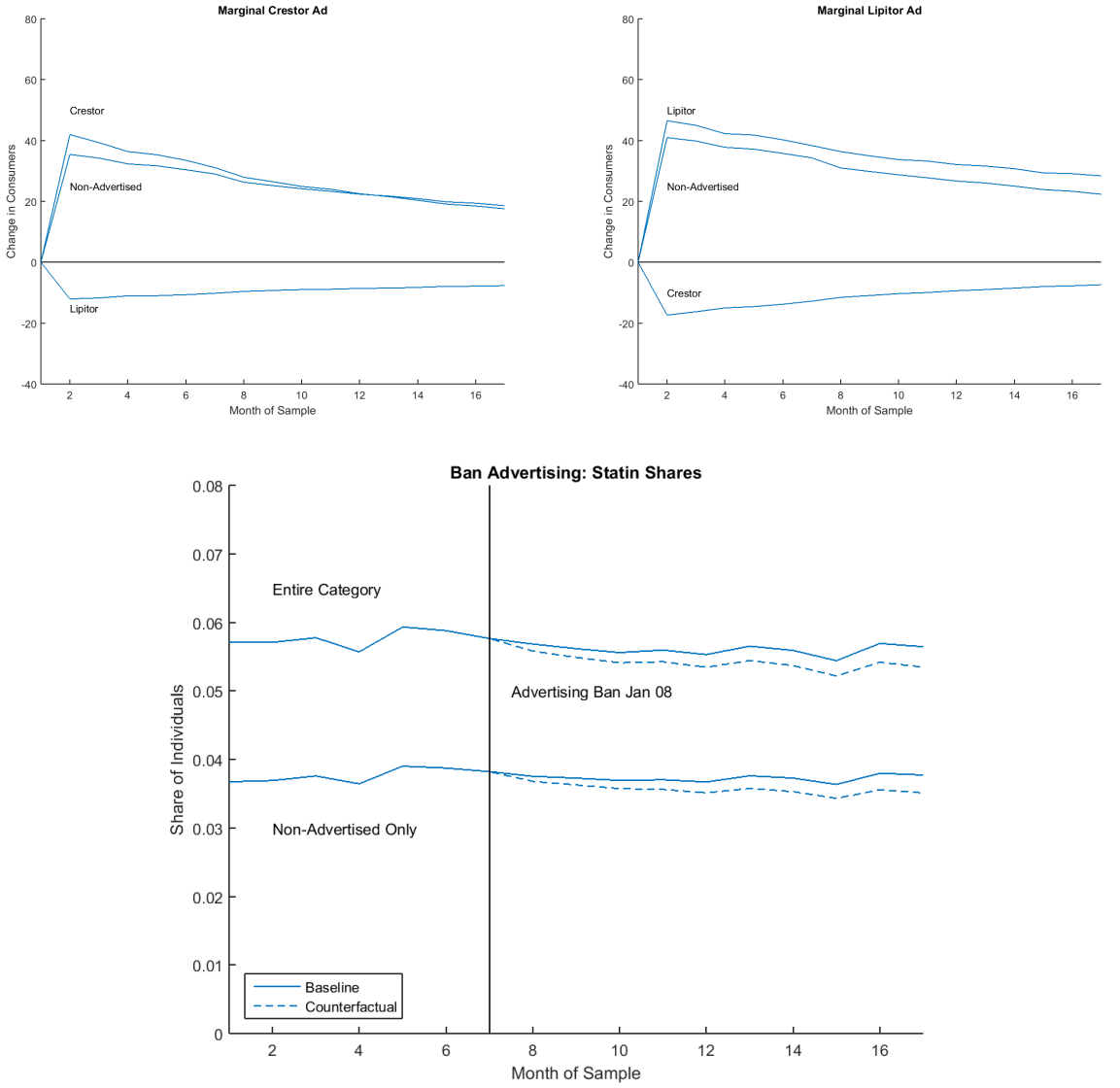
Note: The above plots estimated coefficients for timing dummies relative to a market's primary month. The dependent variable is the (one-month) change in market share, defined as the percentage of the population taking a non-advertised statin.

Figure 8: Effect of Primary Timing on 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 9: Counterfactual Simulations



Note: The above are counterfactual simulations based on our estimated model of consumer demand. The first two show the effect of one additional national ad for each drug in the first two months of our sample; The final shows the simulated effect of banning advertising at the beginning of 2008. See section 5 for an extended discussion of the methodology.

Tables

Table 1: Summary Statistics

Drug		Drug Usage (Truven Analysis Data set)	
Number of Markets	186	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	Conditional Mean of Local Ads by Drug	9.67
Standard Deviation	1,897	Range, Local Ads	(0, 105)
Minimum	0	Conditional Mean of National Ads by Drug	45.31
Maximum	22,636	Range, National Ads	(0, 145)

Notes: Unit of observation is the market-month-product combination. Data (source: left panel, Wisconsin and Kantar; right panel, Truven) span 17 months from July 2007 to November 2008. Averages in top right panel are over the entire population. Means in bottom right panel condition on advertising.

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
% Low Income Subsidy	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. Statistical significance at the 10%, 5% and 1% levels are denoted by *, **, and ***.

Table 3: Political Ads Displace Drug Ads

	Dependent Variable: Local Statin Ads			
Model:	OLS	Tobit	Tobit	Tobit
	All Drugs	All Drugs	Crestor	Lipitor
Log(Political Ads in 1000s + 1)	-0.1583^{***}	-0.6941^{***}	-0.3269^{***}	-1.2423^{***}
	(0.0217)	(0.0219)	(0.0726)	(0.1414)
Controls:				
Market FEs	X	X	X	X
Drug FEs	X	X	X	X
Drug-Year-Month FEs			X	X
N	15,869	15,869	3166	3179
(Pseudo) R^2	0.267	0.331	0.280	0.329

Notes: Unit of observation is the market-month-product combination. Data (source: Wisconsin and Kantar) span 17 months from July 2007 to November 2008 across 210 markets and include Crestor, Lipitor, Vytorin, and Zetia. OLS and Tobit standard errors clustered at the market-month level. The results with Drug-Year-Month fixed effects are identical if local plus national statin ads are used as the dependent variable. 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: Difference-in-Difference Estimates

	Dependent Variable: Log(Days Supply)	
	(1)	(2)
P_{tm}	-0.0291^{***}	-0.0955^{***}
	(0.0106)	(0.0137)
I_t		-0.0873^{***}
		(0.0160)
$P_{tm} * I_t$		0.0979^{***}
		(0.0188)
Products	Crestor, Lipitor	Lipitor
Fixed Effects	Product, DMA	DMA
Clustering	DMA	DMA
N	5,914	3,126
R^2	0.834	0.907

Notes: Unit of observation is the market-month-product combination. Data (source: Truven) span 17 months from July 2007 to November 2008. P_{tm} is an indicator for any political ads in market m in month t. I_t is an indicator for the regulatory action months that prevented Lipitor from advertising. Statistical significant at the 10%, 5%, and 1% levels are denoted by *, **, and *** respectively.

Table 5: OLS Revenue Regressions for Advertised Drugs

Dependent Variable: Log(Revenue per Insured), Two Month Trailing Average				
Own Ads	0.0239^{***}	0.0592^{***}	0.0067^{***}	0.3168^{***}
	(0.0008)	(0.0015)	(0.0007)	(0.0835)
Rival Ads	0.0017^{*}	-0.0268^{***}	0.0037^{***}	-0.1792^{***}
	(0.0009)	(0.0046)	(0.0008)	(0.0589)
Controls:				
Market FEs	X	X	X	X
Drug FEs	X	X	X	X
Year FEs	X		X	
Drug-Year FEs	X			
Drug*Reg. Action FE		X		
Drug FE*Time Trend			X	
Drug FE*Year-Month FE				X
Clustering	DMA	DMA	DMA	DMA
N	11,465	11,465	11,465	11,465
R^2	0.843	0.844	0.847	0.852

Notes: Unit of observation is the market-month-product combination. Data (source: Truven) span 17 months from July 2007 to November 2008 and include Crestor, Lipitor, Vytorin, and Zetia. "Own Ads" and "Rival Ads" are constructed as log(1+X), where X is own ads and the sum of all other category ads, respectively. "Two Month Trailing Average" indicates that the independent variables are constructed as the average of advertising during the revenue month and the month before. Number of observations is smaller than Table 3 because these specifications drop DMAs with no local advertising. This primarily affects small DMAs. "Reg. Action" refers to an indicators for before, during, and after the months in which Lipitor was prevented from advertising. Statistical significant at the 10%, 5%, and 1% levels are denoted by *, **, and *** respectively. Standard errors are clustered at the market level.

Table 6: IV Revenue Regressions for Advertised Drugs

Dependent Variable: Log(Revenue per Insured), Two Month Trailing Average						
Panel A: Second Stage Estimates						
Own Ads	0.1470^{***}		0.0761^{***}		0.1395^{**}	
	(0.0107)		(0.0216)		(0.0557)	
Rival Ads	-0.1140^{***}		-0.0547^{***}		-0.1155^{**}	
	(0.0086)		(0.0180)		(0.0594)	
Panel B: First Stage Estimates (Excluded Instruments)						
	Own	Rival	Own	Rival	Own	Rival
Pol	-0.2099^{***}	0.0463	0.1254^{***}	0.1427^{***}	-0.2602^{***}	-0.1573^{***}
	(0.0465)	(0.0198)	(0.0447)	(0.0256)	(0.0530)	(0.0215)
Pol^2	0.0218^{***}	0.0002	-0.0079	-0.0083^{*}	0.0248^{***}	0.0108^{***}
	(0.0070)	(0.0037)	(0.0062)	(0.0044)	(0.0060)	(0.0023)
Pol^3	-0.0006^{**}	-0.0001	0.0001	0.0001	-0.0007^{***}	-0.0002^{***}
	(0.0003)	(0.0001)	(0.0002)	(0.0002)	(0.0002)	(0.0001)
Reg.Action	-1.6043^{***}	-1.3562^{***}	-1.3412^{***}	-1.2869^{***}		
	(0.0966)	(0.0705)	(0.0977)	(0.0735)		
Reg.Action·	-0.0979	-0.7648^{***}	-0.0127	-0.7374^{***}	0.3948^{***}	0.0103
.....Pol	(0.1049)	(0.0813)	(0.1167)	(0.0895)	(0.0625)	(0.0390)
Reg.Action·	0.0749^{*}	0.2276^{***}	-0.0248	0.1977^{***}	-0.0449^{***}	0.0353^{***}
.....Pol^2	(0.0388)	(0.0328)	(0.0465)	(0.0365)	(0.0122)	(0.0109)
Reg.Action·	-0.0065^{*}	-0.0170^{***}	0.0023	-0.0143^{***}	0.0019^{***}	-0.0017^{**}
.....Pol^3	(0.0035)	(0.0030)	(0.0043)	(0.0033)	(0.0008)	(0.0007)
Fixed Effects	Product-Year		Product-Time Trend		Product-Reg.Action	
Partial F-Stat	1455.9	2979.3	722.9	1679.0	51.6	28.9

Notes: Unit of observation is the market-month-product combination. Number of observations is 11,465 in all specifications. Data (source: Truven) span 17 months from July 2007 to November 2008 and include Crestor, Lipitor, Vytorin, and Zetia. "Own Ads" and "Rival Ads" are constructed as log(1+X), where X is own ads and the sum of all other category ads, respectively. All specifications are a "Two Month Trailing Average," which indicates that the independent variables are constructed as the average of advertising during the revenue month and the month before. "Pol" is the number of political ads in a market-month, in thousands. "Regulatory Action" is a dummy variable for April-August 2008, when congressional action forced Lipitor to stop advertising. All specifications include market, year, and drug fixed effects. The second specification allows for drug-specific linear time trends. The final specification includes all possible interactions between product dummies and three regulatory-period dummies for before, during, and after the regulatory action, which control for both the effect of the regulatory action in both stages of the regression and the time trends. F-statistics are for excluded instruments in Panel B. Specifically, the regulatory action is included in both stages in the final specification, excluded from Panel B, and implies the lower Partial F-stat. Standard errors are clustered at the market level. Statistical significant at the 10%, 5%, and 1% levels are denoted by *, **, and *** respectively.

Table 7: Heterogeneity across Drugs

Dependent Variable: Log(Revenue per Insured)			
Drug:	All	Crestor	Lipitor
Own Ads	0.1395**	0.3186**	0.1014**
	(0.0557)	(0.1499)	(0.0401)
Rival Ads	−0.1155**	−0.1319**	−0.2069***
	(0.0594)	(0.0608)	(0.0667)
Controls:			
Market FEs	X	X	X
Year FEs	X	X	X
Drug FEs	X		
Drug FE*1(Regulatory Action)	X		
Drug FE*1(Post-Regulatory Action)	X		
1(Regulatory Action)		X	X
1(Post-Regulatory Action)		X	X
Clustering	DMA	DMA	DMA
N	11,466	2,788	3,126
R^2	0.824	0.545	0.837

Notes: Unit of observation is the market-month-product combination. Data (source: Truven) span 17 months from July 2007 to November 2008 and include Crestor, Lipitor, Vytorin, and Zetia. "Own Ads" and "Rival Ads" are constructed as log(1+X), where X is own ads and the sum of all other category ads, respectively. "This Month" indicates contemporaneous advertising, while longer time spans indicates that the independent variables are constructed as the average of advertising during the revenue month and the months before. First stage excluded instruments are political advertising, its square and cube, and the interactions of the political variables and the regulatory action dummy. Statistical significant at the 10%, 5%, and 1% levels are denoted by *, **, and *** respectively. Standard errors are clustered at the market level.

Table 8: Spillovers for Non-Advertised Drugs

Dependent Variable:	Log(Revenue per Insured), Non-Advertised Drugs		Log(Revenue per Insured), Full Category	
Model:	OLS	IV	OLS	IV
Category Ads	0.0018	0.0188**	0.0069**	0.0128**
	(0.0021)	(0.0093)	(0.0032)	(0.0063)
Controls:				
Market FEs	X	X	X	X
Time Trends	X	X	X	X
Clustering	DMA	DMA	DMA	DMA
N	3,112	3,112	3,126	3,126
R^2	0.880	0.883	0.898	0.898

Notes: Unit of observation is the market-month combination. Data (source: Truven) span 17 months from July 2007 to November 2008 and include all non-advertised drugs. "Category Ads" is constructed as log(1+X), where X is the average of category advertising during the revenue month and the month before. First stage excluded instruments are political advertising, its square and cube. Differences in the number of observations across specifications is due to censoring in our Truven dataset. Non-advertised drugs are defined in this regression as all drugs in the class except Crestor, Lipitor, Vytorin, and Zetia. Statistical significant at the 10%, 5%, and 1% levels are denoted by *, **, and *** respectively.

Table 9: IV Revenue Regressions for Advertised Drugs, Part D Data

	All Prescriptions		New Prescriptions	
Model:	OLS	IV	OLS	IV
Own Ads	0.0165^{***}	0.1310^{***}	0.0574^{***}	0.371^{***}
	(0.00171)	(0.0311)	(0.00744)	(0.133)
Rival Ads	0.00940^{**}	-0.0234^{***}	0.0752^{***}	-0.211^{***}
	(0.00454)	(0.0138)	(0.0200)	(0.0819)
Controls:				
Market FEs	X	X	X	X
Year FEs	X	X	X	X
Drug FEs	X	X	X	X
Drug FE*	X	X	X	X
... 1(Regulatory Action)				
Drug FE*	X	X	X	X
... 1(Post-Regulatory Action)				
1(Regulatory Action)	X	X	X	X
1(Post-Regulatory Action)	X	X	X	X
Clustering	DMA	DMA	DMA	DMA
N	11,466	11,466	10,743	10,743
R^2	0.879	0.867	0.599	0.536

Notes: Data created by restricting Medicare Part D event data to either all Crestor, Lipitor, Vytorin, and Zetia fills or the first prescriptions by beneficiary of those drugs and collapsing to the market-month-product level. Unit of observation is the market-month-product combination. Data span 17 months from July 2007 to November 2008. "Own Ads" and "Rival Ads" are constructed as log(1+X), where X is own ads and the sum of all other category ads, respectively. All dependent variables are the "Two Month Trailing Average." First stage excluded instruments are political advertising, its square and cube, a dummy that takes on a one during April 2008-August 2008 (regulatory action dummy), and the interactions of the political variables and the regulatory action dummy. Statistical significant at the 10%, 5%, and 1% levels are denoted by *, **, and *** respectively. In all specifications, we dropped markets with less than 30 days supply of new prescriptions, which reduces the sample size for new prescriptions.

Table 10: Demand Model

From \ To	Crestor	Lipitor	Non-Advertised	Outside Good
		Panel A:		
		Monthly Transition Matrix by DMA, Model		
Crestor	P_{cc}	P_{cl}	P_{cn}	P_{co}
Lipitor	P_{lc}	P_{ll}	P_{ln}	P_{lo}
Non-Advertised	P_{nc}	P_{nl}	P_{nn}	P_{no}
Outside Good	P_{oc}	P_{ol}	P_{on}	P_{oo}
		Panel B:		
		Parameterization of $\delta_{jkt} - \xi_{kt}$ by Drug		
Crestor	$\beta_0^{cc} + ...$ $\beta_1^{cc} log(1+a_{kt}) + ...$ $\beta log(1+a_{-kt})$	β_0^{cl}	β_0^{cn}	-
Lipitor	β_0^{lc}	$\beta_0^{ll} + ...$ $\beta_1^{ll} log(1+a_{kt}) + ...$ $\beta_2^{ll} log(1+a_{-kt})$	β_0^{ln}	-
Non-Advertised	β_0^{nc}	β_0^{nl}	$\beta_0^{nn} + ...$ $\beta_2^{nn} log(1+a_{-kt})$	-
Outside Good	$\beta_0^{oc} + ...$ $\beta_1^{oc} log(1+a_{kt}) + ...$ $\beta_2^{oc} log(1+a_{-kt})$	$\beta_0^{ol} + ...$ $\beta_1^{ol} log(1+a_{kt}) + ...$ $\beta_2^{ol} log(1+a_{-kt})$	$\beta_0^{on} + ...$ $\beta_2^{on} log(1+a_{-kt})$	-
		Panel C:		
		Empirical Yearly Transition Matrix		
Crestor	0.6716	0.01925	0.1599	0.1493
Lipitor	0.01312	0.7129	0.1512	0.1228
Non-Advertised	0.01547	0.0206	0.8133	0.1506
Outside Good	0.00910	0.0212	0.1004	0.8693

Notes: Panel A shows the basic structure of the transition matrix where each row represents a consumption state in the prior period while columns represent subsequent periods. Given this structure, $\sum_{k\in\{c,l,n,o\}} P_{jk} = 1 \, \forall j$. Panel B shows the exact parameterization for each of the probabilities, where a_{kt} represents own advertising of good k, represented by the column, and a_{-kt} represents rival advertising for good k. Panel C shows the average annual (not monthly) empirical transition probabilities from the Medicare Part D data.

Table 11: Model Estimates Drug

	Fixed Effect β_0^{jk}	Own Advertising Effect β_1^{jk}	Rival Advertising Effect β_1^{jk}
from Crestor $(j=c)$			
... to Crestor $(k=c)$	3.5486	-0.0040	0.0030
	(0.0036)	(0.0107)	(0.0211)
... to Lipitor $(k=l)$	-1.3689		
	(0.0003)		
... to Non-Advertised $(k=n)$	-1.0511		
	(0.0001)		
from Lipitor $(j=l)$			
... to Crestor $(k=c)$	0.1885		
	(0.0004)		
... to Lipitor $(k=l)$	4.1009	0.0069	-0.0052
	(0.0029)	(0.0121)	(0.0109)
... to Non-Advertised $(k=n)$	1.2212		
	(0.0006)		
from Non-Advertised $(j=n)$			
... to Crestor $(k=c)$	-2.5898		
	(0.0001)		
... to Lipitor $(k=l)$	-3.8303		
	(0.0001)		
... to Non-Advertised $(k=n)$	4.8008		0.0077
	(0.0022)		(0.0106)
from Outside Good $(j=o)$			
... to Crestor $(k=c)$	-7.0652	0.0154	-0.0211
	(0.0046)	(0.0083)	(0.0288)
... to Lipitor $(k=l)$	-8.5700	0.0996	-0.0031
	(0.0025)	(0.0107)	(0.0097)
... to Non-Advertised $(k=n)$	-5.4054		0.0431
	(0.0034)		(0.0186)
Nesting Parameters			
λ_d		0.5906 (0.0053)	
λ_a		0.3918 (0.0041)	

Notes: Table shows GMM estimates of model parameters with asymptotic standard errors in parentheses. See Section 5 for details. We collapse Vytorin and Zetia into the non-advertised group in order to simplify the model. As shown in Figure 3, Vytorin and Zetia dramatically scale back advertising in February of 2008 and cease advertising entirely by June 2008, so they are not central to the exercise. Whenever we observe Vytorin and Zetia advertising, it is included in our measure of rival ads.

Appendix

Supplemental Appendix For Online Publication

A Model Simulation

A.1 Results

Consider a static, simultaneous move advertising game among two single-product firms with demand for drugs $j \in 1,2$ given by

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

where a_j is firm j's advertising level and a_{-j} is rival advertising. The vector ξ is a set of shocks to demand for each good, $\xi = \{\xi_1, \xi_2\}$.

In equilibrium, firms choose a_j such that the marginal benefit of advertising equals its marginal cost. Firms observe their demand shock, but not their rivals', 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 ξ.[54]

The econometrician estimates the demand elasticity of own and rival advertising using a specification such as

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

Because the demand shock ξ is unobserved to the econometrician, OLS estimates of β_1 and β_2 suffer from omitted variables bias.[55] 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

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

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

[55] In our example, this heterogeneity is positively correlated with the input of interest, e.g. $\frac{\partial a_j}{\partial \xi_j} > 0$, as Lipitor's positive demand shock in a market increases their return to advertising. The bias will depend on the correlation between demand shocks and advertising.

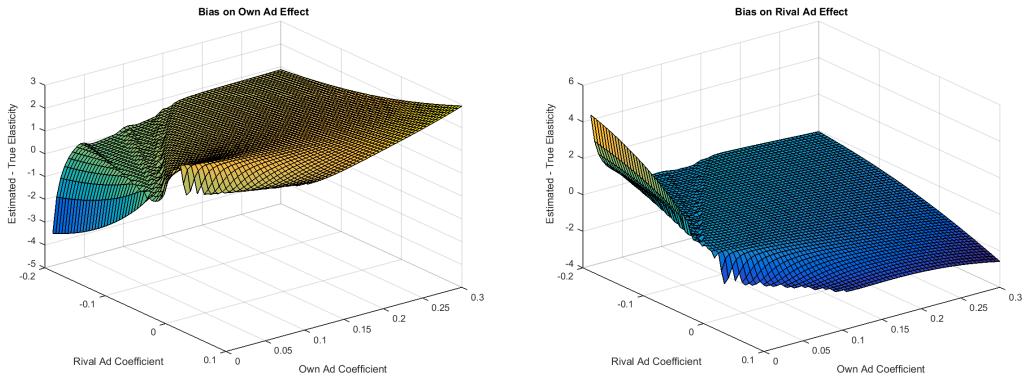
where u_{i0} denotes the utility of the outside good. Assuming ε_{ijm} is i.i.d. type I extreme value, market shares D_{jm} can be computed given parameters and advertising levels using the standard Logit formula:

$$D_{jm} = \frac{exp\left(\alpha_j + \beta_1 ln(1+a_{jm}) + \beta_2 ln(1+a_{\_jm})\right)}{1+\sum_j exp\left(\alpha_j + \beta_1 ln(1+a_{jm}) + \beta_2 ln(1+a_{\_jm})\right)}.$$

The per-unit cost of advertising is c, and profit per unit sold is ρ. Firm profits in this model are given by $\pi_{jm} = \rho D_{jm} - c a_{jm}$, where ρ is the margin on an individual unit. We draw values of ξ_{jm} and 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.

We simulate 200 markets and optimal advertising decisions for both firms at a range of parameter values for β_1 and β_2. The plots in Figure 10 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.[56]

Figure 10: Simulations of OLS Estimate Bias



A.2 Simulation Details

Parameters were set to the following values: $\alpha_1 = \alpha_2 = -0.3$, $c = 1$, $\rho = 1000$. 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 ξ 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

[56] Table 12 shows estimates and standard errors for a particular set of parameter values.

observations of

$$\begin{aligned}\eta_{own} &= \frac{a_j}{1+a_j}\left(\beta_1(1-s_j)-\beta_2 s_{-j}\right)\\ \eta_{rival} &= \frac{a_{-j}}{1+a_{-j}}\left(\beta_2(1-s_j)-\beta_1 s_{-j}\right)\end{aligned}$$

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 $\beta_1 \in [0.01, 0.2]$ and $\beta_2 \in [-0.2, 0.1]$, both in increments of 0.005. We drop cases where $\beta_2 > \beta_1$ as firms would choose negative advertising. The share of simulations where the bias in estimating own advertising elasticity was less than 5%, was only 0.3% of simulations, and 1.5% for rival advertising elasticity. Table 12 shows for one particular set of parameter values the OLS bias in estimating elasticities of own and rival ads.

For completeness, we also performed the same analysis where only a single firm chooses advertising. Figure 11 shows the bias from OLS estimation of the elasticity of revenue with respect to own advertising. As is clear, the bias can be positive or negative.

Figure 11: OLS Bias with One Firm

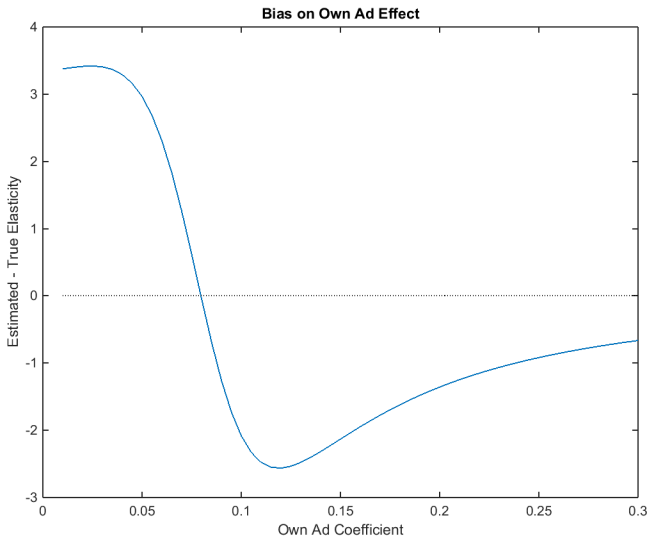


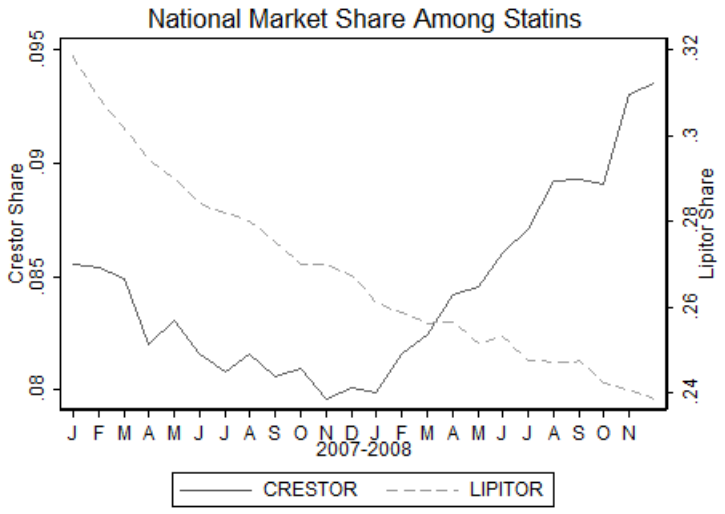
Table 12: Sample Model Simulation Results

Dependent Variable:	Log(Revenue)		
Specification:	Naive	With ξ	Analytic Values
	(1)	(2)	(3)
Log(1+Own Ads)	0.0290^{***}	0.0763^{***}	0.0796
	(0.0065)	(0.0006)	
Log(1+Rival Ads)	-0.0770^{***}	-0.1305^{***}	-0.1266
	(0.0059)	(0.0006)	
Control: ξ		X	
N	200	200	
R^2	0.687	0.998	

Notes: Parameter values for these results were $\beta_1 = 0.06$ and $\beta_2 = -0.15$. Firm optimal advertising levels were solved for using Matlab's FSOLVE routine and first-order conditions for profit maximization. Estimates are OLS results for equation 1, with ξ_j and ξ_{-j} as additional controls in the second column. Analytic values are computed as the means of the expressions for η_{own} and η_{rival} shown above. The controlled version does not perfectly match the analytic values as the Logit model creates a non-linear error term when estimating equation1.

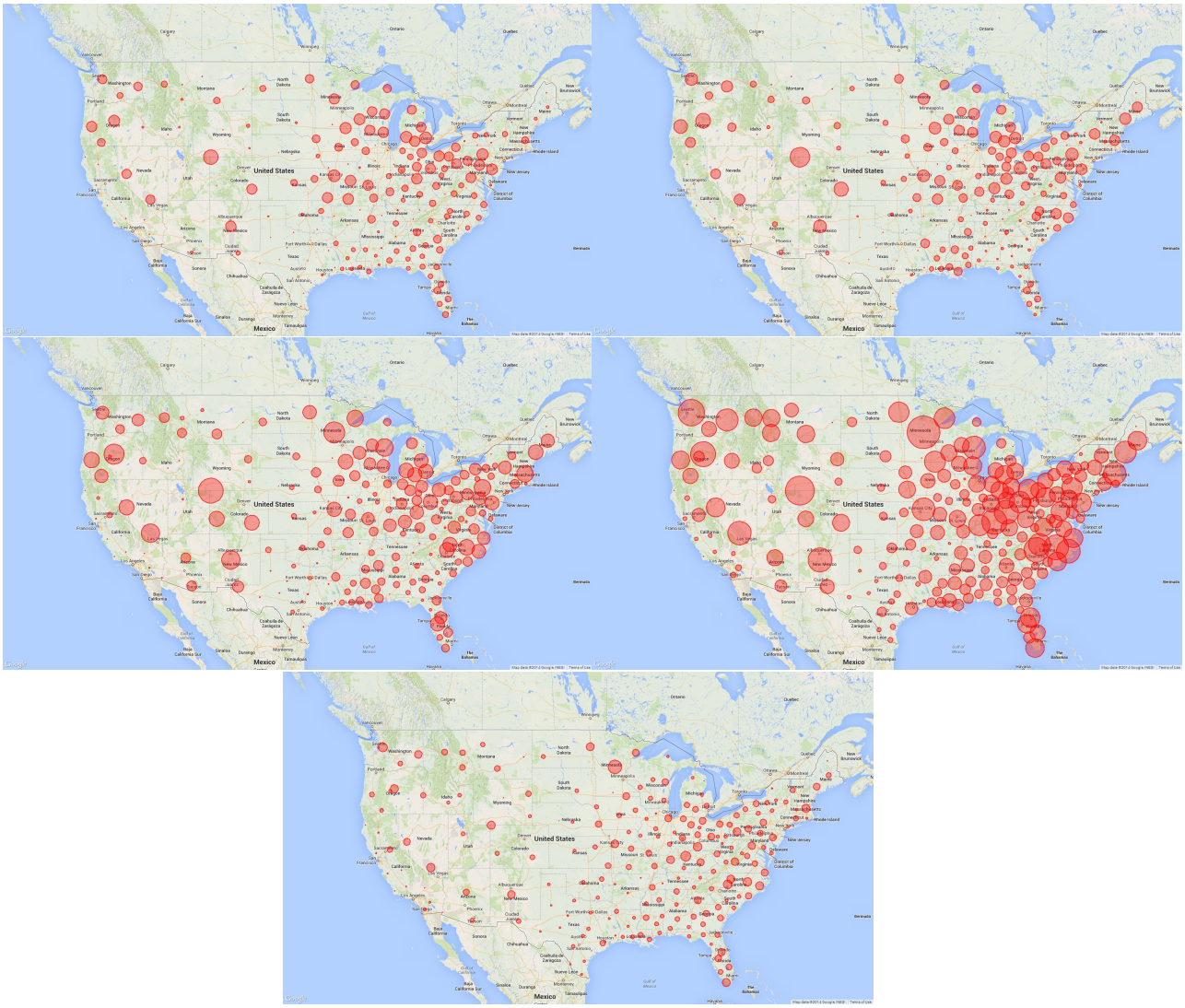
B Additional Summary Statistics and Robustness Checks

Figure 12: Time Trends



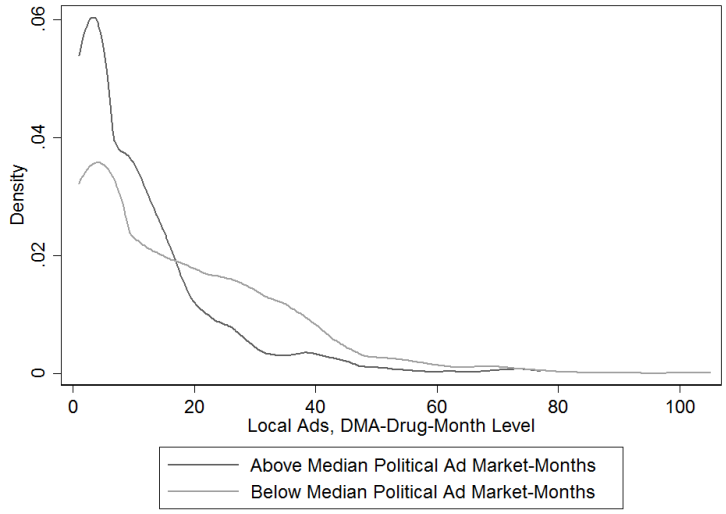
Notes: The above graphic plots the share of Lipitor and Crestor from the Truven data as a percentage of total category sales over the period of January 2007-November 2008. Note different axes.

Figure 13: Political Ad Levels, July-November 2008



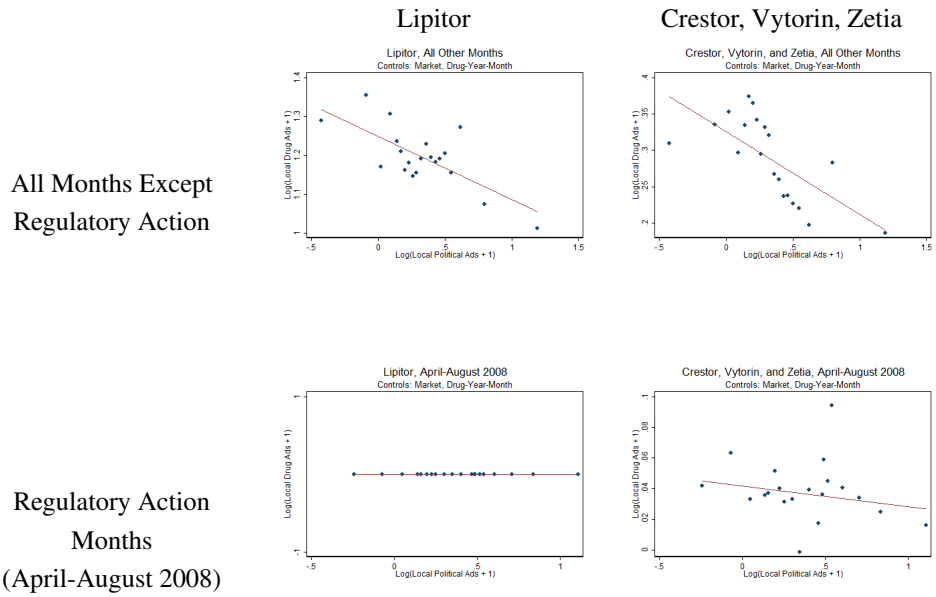
Notes: The above maps show a circle for each DMA in the USA. The diameter of each circle is proportional to the number of political ads aired in that market, in that month, for all races (Presidential, Senatorial, House, Gubernatorial). The first row are July and August; second row are September and October, and third row is November.

Figure 14: Histogram of Local Drug Advertising



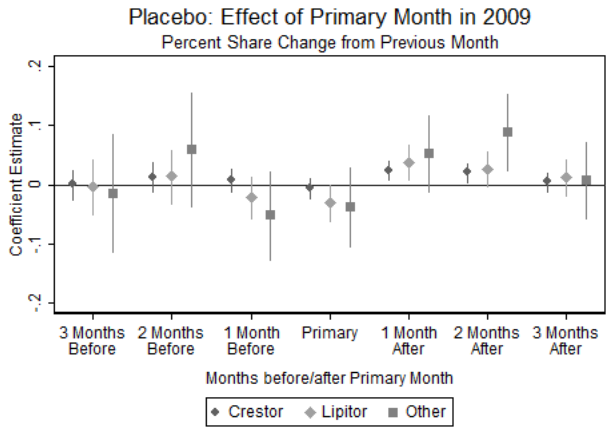
Notes: The above graphic plots the number of local ad impressions of Lipitor and Crestor from the Kantar data over the period of January 2007-November 2008. The sample is split into market-months with below median political advertising and above-median political advertising, and omit drug-market-months with no local advertising.

Figure 15: Instrument Effect Heterogeneity



Notes: The above graphic plots binned scatterplots to show that political ads displace drug ads, as Figure 5, for different sub-samples of the data. "Regulatory Action" months refer to the months when Lipitor was banned from advertising by congress.

Figure 16: 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, Crestor, or Lipitor, respectively.

Table 14: 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 in 2008. 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: Political Ads Displace Drug Ads

	Dependent Variable: Log(Local Statin Ads + 1)					
Model:	OLS	OLS	OLS	Tobit	Tobit	Tobit
Log(Political Ads in 1000s + 1)	-0.1825^{***}	-0.1063^{***}	-0.1063^{***}	-0.7729^{***}	-0.2329^{***}	-0.1940^{***}
	(0.0129)	(0.0143)	(0.0143)	(0.0494)	(0.0581)	(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	14,280	14,280	14,280	14,280	14,280	14,280
R^2	0.267	0.357	0.477	0.213	0.348	0.519

Notes: Unit of observation is the market-month-product combination. Data (source: Wisconsin and Kantar) span 17 months from July 2007 to November 2008 across 210 markets and include Crestor, Lipitor, Vytorin, and Zetia. OLS and Tobit standard errors clustered at the market-month level. The results with Drug-Year-Month fixed effects are identical if local plus national statin ads are used as the dependent variable. Statistical significance at the 10%, 5% and 1% levels are denoted by *, **, and ***. Reported R^2 is adjusted for OLS, pseudo for Tobit.

Table 15: Robustness: No Substitution to Other Media

	Dependent Variable: Local Non-TV Advertising Spending		
Model:	OLS	OLS	OLS
Political Ads (1000s)	-0.3000^{*}	-0.1731	-0.1962
	(0.1770)	(0.1802)	(0.1804)
Local TV Drug Ads		0.8664^{***}	0.9515^{***}
		(0.1366)	(0.1433)
National TV Drug Ads			-0.0724^{***}
			(0.0138)
Controls:			
Market FEs	X	X	X
Year-Month FEs	X	X	X
Drug FEs	X	X	X
N	14,867	14,867	14,867
R^2	0.074	0.086	0.087

Notes: Regressions combine the Wisconsin and Kantar data sets for the months of July 2007-November 2008. OLS standard errors clustered at the market-year-month level. Statistical significance at the 10%, 5% and 1% levels are denoted by *, **, and ***.

Table 16: Timing Assumption Sensitivity

	Dependent Variable: Log(Revenue per Insured)				
Advertising Timing:	This Month	Two Month	Two Month	Three Month	One Month Lag
Own Ads	0.1208^{***}	0.1395^{**}	0.1396^{***}	-0.0618	0.2021^{***}
	(0.0198)	(0.0557)	(0.0297)	(0.0575)	(0.0368)
Rival Ads	-0.1088^{***}	-0.1155^{**}	-0.1157^{***}	0.0018	-0.0780^{***}
	(0.0231)	(0.0594)	(0.0364)	(0.0346)	(0.0233)
Jan.-Jun. '07 Ads			0.0502		
			(0.0317)		
Clustering	DMA	DMA	DMA	DMA	DMA
N	11,465	11,465	11,465	10,795	10,797
R^2	0.804	0.824	0.820	0.843	0.800

Notes: Unit of observation is the market-month-product combination. Data (source: Truven) span 17 months from July 2007 to November 2008 and include Crestor, Lipitor, Vytorin, and Zetia. "Jan-Jun '07 Ads" supplements with additional Kantar advertising data and Truven utilization data for those months. "Own Ads" and "Rival Ads" are constructed as log(1+X). "This Month" indicates contemporaneous advertising, while longer time spans indicates that the independent variables are constructed as the average of advertising during the revenue month and the months before. First stage excluded instruments are political advertising, its square and cube, and the interactions of the political variables and the regulatory action dummy. Fixed effects include all possible interactions between product dummies and three regulatory-period dummies for before, during, and after the regulatory action, which control for both the effect of the regulatory action in both stages of the regression and the time trends. Statistical significant at the 10%, 5%, and 1% levels are denoted by *, **, and *** respectively. Standard errors are clustered at the market level.

Table 17: Effect of Business Stealing (IV Results)

Dependent Variable: Log(Revenue per Insured)				
Log Own Ads	0.0117^{***}	0.0761^{***}	0.0695^{***}	0.1395^{**}
	(0.0015)	(0.0216)	(0.0187)	(0.0557)
Log Rival Ads		-0.0547^{***}		-0.1155^{**}
		(0.0180)		(0.0594)
Fixed Effects:	Product-Time Trend		Product-Reg.Action	
N	11,466	11,466	11,465	11,465
R^2	0.849	0.810	0.844	0.824

Notes: Unit of observation is the market-month-product combination. Data (source: Truven) span 17 months from July 2007 to November 2008 and include Crestor, Lipitor, Vytorin, and Zetia. "Own Ads" and "Rival Ads" are constructed as log(1+X), where X is own ads and the sum of all other category ads, respectively. "This Month" indicates contemporaneous advertising, while "Two Month Trailing Average" indicates that the independent variables are constructed as the average of advertising during the revenue month and the month before. First stage excluded instruments are political advertising, its square and cube, a dummy that takes on a one during April 2008-August 2008 (regulatory action dummy), and the interactions of the political variables and the regulatory action dummy. Statistical significant at the 10%, 5%, and 1% levels are denoted by *, **, and *** respectively. Standard errors are clustered at the market level.

Table 18: Sensitivity to Sample Selection

	(1)	(2)	(3)
Own Ads	0.1395^{***}	0.0841^{***}	0.1378^{***}
	(0.0297)	(0.0841)	(0.0338)
Rival Ads	-0.1155^{***}	-0.1217^{***}	-0.1209^{***}
	(0.0365)	(0.0361)	(0.0423)
Sample	All	Omitting Dec. 2007-Feb. 2008	Omitting Ohio & Florida
Observations	11,465	9,425	10,362
Adjusted R-Squared	0.820	0.836	0.816

Notes: Unit of observation is the market-month-product combination. Data (source: Truven) span 17 months from July 2007 to November 2008 and include Crestor and Lipitor. "Own Ads" and "Rival Ads" are constructed as log(1+X), where X is own ads and the sum of all other category ads, respectively. All specifications are utilize a "Two Month Trailing Average," which indicates that the independent variables are constructed as the average of advertising during the revenue month and the month before. Fixed effects include all possible interactions between product dummies and three regulatory-period dummies for before, during, and after the regulatory action, which control for both the effect of the regulatory action in both stages of the regression and the time trends. Statistical significant at the 10%, 5%, and 1% levels are denoted by *, **, and *** respectively.

Table 19: Monthly Transition Matrix

Monthly Estimated Transition Matrix					
From \ To		Crestor	Lipitor	Non-Advertised	Outside Good
		$j = c$	$j = c$	$j = n$	$j = o$
Crestor	$j = c$	0.9716	0.0001	0.0004	0.0279
Lipitor	$j = l$	0.0001	0.9763	0.0075	0.0162
Non-Advertised	$j = n$	0.0001	0.0001	0.9918	0.0008
Outside Good	$j = o$	0.0002	0.0001	0.0043	0.9954

Notes: This table describes the model predictions of average monthly transition probabilities across markets at our estimates. To compare to the empirical annual transition matrix based on the Part D data, one can take the 12th power of the matrix.

Table 20: Covariate Balance, Part D Data

Total Statin Ads:	Below Median Markets	Above Median Markets	Difference
Full Sample			
Average Age	71.13	71.30	-.1642
% Female	.5449	.5563	-.0114***
% White	.8848	.8416	.0432***
Mortality Rate	.0426	.0423	.0003
% Low Income Subsidy	.6853	.6674	.0178*
Average Price Per Day Supplied, All Drugs	1.675	1.662	.0132
Average OOPC Per Day Supplied, All Drugs	.6422	.6118	.0304***
Average Price Per Day Supplied, Branded Statins	3.188	3.216	-.0275
Average OOPC Per Day Supplied, Branded Statins	.8071	.8120	-.0048
Enrollees with Statin Fills			
Average Age	71.42	71.72	-.2948
% Female	.5925	.5951	-.0026
% White	.8722	.8273	.0449**
Mortality Rate	.0336	.0339	-.0003
% Low Income Subsidy	.4352	.4173	.0180
Average Price Per Day Supplied, All Drugs	1.570	1.542	.0282
Average OOPC Per Day Supplied, All Drugs	.6009	.5667	.0342***
Enrollees with New Statin Fills in 2008			
Average Age	71.65	71.97	-.3228*
% Female	.5946	.5984	-.0038
% White	.8751	.8304	.0446**
Mortality Rate	.0291	.0292	-.0001
% Low Income Subsidy	.4077	.3833	.0244
Average Price Per Day Supplied, All Drugs	1.532	1.503	.0289
Average OOPC Per Day Supplied, All Drugs	.5863	.5543	.0319***
N	97	93	

Notes: We split the Part D beneficiary summary sample into two groups. We take the sum of statin advertising over the 2008 calendar year and compare demographics for markets above and below the median. Statistical significance at the 10%, 5% and 1% levels are denoted by *, **, and ***. Number of markets are not equal across high and low markets due to ties.

C Physician Payments Analysis

In this paper, we argue that detailing activities by pharmaceutical manufacturers are unaffected by the political cycle. In order to document this fact, we utilize ProPublica's Dollars for Docs dataset. Lawsuits over "off-label" use between 2009 and 2012 required 15 firms (including Pfizer and AstraZeneca) to disclose payments made to prescribers. The data on payments made by drug firms to prescribers come from the non-profit organization ProPublica and contain more than 2.1 million "payments" for meals, travel, speaking fees, honoraria, gifts, and research and records the dollar value and, importantly, date of each. The data covers August 2013-December 2014. As documented in Carey, Leiber and Miller (2015), this type of promotional activity has a positive impact on sales; furthermore, while the median payment is small, the distribution is highly skewed. We use payee zipcode to aggregate to firm-DMA-month level and focus on the manufacturers in our main analysis.

We explore whether payments are correlated with the political cycle. In order to test this hypothesis, we examine the 2014 midterm elections, for which data is available. While midterm elections in the United States do not coincide with the presidential race, approximately one-third of senate seats and many governorships are on the ballot; we exploit this local variation in the influence of political advertising to explore the effect on physician marketing activities. Many races were actually uncontested or not seriously contested, and political ads are rare in such markets. There were 34 senate seats and 36 governorships up for election in 2014. Of these, 5 states had "competitive" senate races and 15 states had "competitive" gubernatorial races; 13 states had both. We compare prescriber payments over 2014 (the election year) in competitive and non-competitive markets. We believe that if we do not find an effect in 2014, it is unlikely that a correlation would be present in 2008 for several reasons. First, political advertising only increased during the intervening year, due in part to the Citizens United ruling, which enabled political action committees (PACs) to increase advertising levels dramatically. Second, firms are increasingly aware of this issue and likely to react to it. Finally, the races in this analysis are all predictable events.

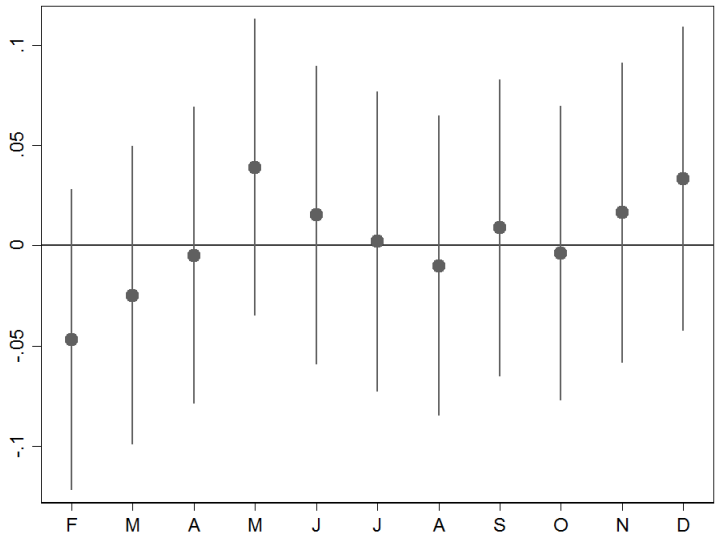
First, we explore spending across all firms in 2014. We create a "contested" variable that takes on a one in DMAs in states with a senate or gubernatorial race with a margin of victory of 5% or less. We interact this dummy with month dummies and plot the coefficients on the interactions over the course of the year. If firms substitute to detailing activity, we would expect an uptick in physician promotional spending in September and especially October leading up to the election. Figure 17 documents the lack of a relationship between the political process and promotional payments to prescribers in the full sample.

Next, we focus on the key firms of interest in this study – Pfizer (Lipitor) and AstraZeneca (Crestor) – in a series of regression results. Total AstraZeneca payments to prescribers in 2014

were $72.5M while total payments from Pfizer to prescribers in 2014 were $54.6M. We note that during the sample period, Lipitor no longer had patent protection, but that Pfizer still promotes the other drugs in its portfolio. These firms are among those with the highest payments to prescribers (Carey, Leiber and Miller (2015)); we explore variation in these payments over time and across markets. These regressions capture the impact of interaction of two dummy variables that account for timing and the competitiveness of political races. The first dummy takes on a one in October and the second takes on a one in DMAs particularly exposed the political process during October. In all specifications, we include DMA and month fixed effects. Therefore, the dependent variables of interest are indicators that are equal to one in October 2014 in markets with senate and gubernatorial elections, as well as indicators for close such elections (margin of victory of 5% or less) in October of 2014.

We estimate OLS regressions in which the dependent variable is the log of prescriber payments in a market-month for AstraZeneca and Pfizer separately. We include month and market fixed effects. A small number of DMAs see no payments in a several months; those observations are dropped. In all of the specifications, the point estimates are zero or negative and statistically indistinguishable from zero. Point estimates for AstraZeneca suggest physician payments were 7.6% lower in October 2014 markets with close elections. Point estimates for Pfizer indicate that payments were 0.87% higher in those markets. Our standard errors are conservative, yet we find no evidence that the political process drives payments to prescribers. These results are consistent with the finding in Alpert, Lakdawalla and Sood (2015) and Shapiro (2016), which find no evidence that DTC advertising and physician payments are positively correlated. Therefore, we conclude that even in cases where consumer advertising is predictably disrupted, firms do not appear to substitute towards additional physician marketing.

Figure 17: Effect of Close Elections on Physician Payments



Note: The above plots estimated coefficients for the contested election dummy (which takes on a one for markets in which there is a senate or gubernatorial election with a margin of victory less than 5%) interacted with monthly dummies relative to January of 2014. The dependent variable is the log of physician payments and the unit of analysis the firm-month.

Table 21: Sensitivity of Physician Payments to the Political Cycle

	(1)	(2)	(3)
AstraZeneca			
1(Political Contest)*1(October)	-0.0032		0.0082
	(0.1026)		(0.1035)
1(Close Senate or Gubernatorial Contest)		-0.0757	-0.0770
*1(October)		(0.1067)	(0.1078)
Observations	2483	2483	2483
Adjusted R-Squared	0.879	0.879	0.879
Pfizer			
1(Political Contest)*1(October)	-0.0888		-0.0923
	(0.1203)		(0.1207)
1(Close Senate or Gubernatorial Contest)		0.0087	0.0232
*1(October)		(0.1140)	(0.1143)
Observations	2465	2465	2465
Adjusted R-Squared	0.844	0.844	0.844

Notes: Data created by aggregating the Open Payments Data to the DMA-firm-month level. Data span 12 months of 2014, as the data is not available before 2013 and is not yet available for 2016. The dependent variable is the log of the sum of all physician payments by the firm in that month and DMA. Month and DMA fixed effects are included in all specifications. Statistical significant at the 10%, 5%, and 1% levels would be denoted by *, **, and *** respectively.