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, simulations quantify the magnitude of business stealing and explore heterogeneity in policy effects.

JEL classification: I11, L10

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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 paper provides evidence on the impact of advertising and, therefore, strategic incentives facing firms. In addition, our estimates focus on a policy-relavant 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 under-advertise. We utilize a model of firm advertising decisions that allows us to quantify the direction of potential bias and highlight the need for 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 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-difference 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 .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 -.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

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

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 important impact on drug sales. Competitive interaction between rivals is an important feature of the market, and rival advertising can have a significant business-stealing effect among some drugs, while having a beneficial effect on others.

We use our estimates in a number of policy simulations. First, we show that the estimated business-stealing effect is economically meaningful: revenue for branded advertised drugs would 21-24% higher absent the effect of rival advertising. Second, banning DTCA harms sales of unadvertised drugs. For advertised drugs, business stealing mutes the impact of a ban: the net effect of eliminating both the positive and negative effects of advertising is a modest 2% reduction in quantity for Lipitor and a reduction of less than 1% for Crestor.

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). 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 (2014), which estimates economically significant spillover effects in the anti-depressant market using a cross-border strategy and structural model of demand. Our paper is consistent with these previous studies, while finding an additional, economically important role for business stealing in the statin market 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 Rao (2013) and Blake, Nosko and Tadelis (2013) 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 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

[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).

bias. Section 3 describes the data and empirical strategy, while Section 4 presents results and robustness checks. Section 5 details simulations, 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 1991, as was Pravachol.

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 was manufactured by Pfizer starting in 1997, and Crestor was manufactured by AstraZeneca starting in 2003. 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] 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 is scheduled to expire in 2016.

Manufacturer strategies for differentiating their products often rely on results from clinical trials showing efficacy. Zocor marked an early use of clinical trials in marketing drugs (largely to physicians): Merck showed in the Scandinavian Simvastatin Survival Study (4S) that Zocor prevented additional heart attacks among patients who had already suffered a heart attack. In April 2008, 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.

[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.

[7] They use the results of this trial in marketing. See, for example, http://www.crestor.com/c/about-crestor/crestor-

Two issues affected the marketing of statins during our sample period. First, 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 discouraging 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] Second, 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%.[13]

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.[14] Insurance coverage 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.

By contrast, most seniors obtain their drug coverage through the Medicare Part D program. Consumers in Medicare Part D face a very non-linear insurance contract: there is an initial deductible, followed by (an average of) 25% co-payment rates up to an initial coverage limit. Once a consumer hits the initial coverage limit, they must pay for all of their expenditure in the "donut hole" or coverage gap until they meet a catastrophic cap. The donut hole is now closing due to the Patient Protection and Affordable Care Act (ACA), but this basic structure would have been in place during our sample period. There are many plans available to most consumers and these plans are likely to vary substantially in terms of their formularies, that is, the specific drugs covered by the plan.

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] See 11.

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

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.

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). Both plan selection and physician agency are outside the scope of this paper. While they influence the market, their effects are likely to remain fixed over our short time period, allowing us to focus on measuring the impact of DTCA given consumer price sensitivity and agency.

2.2 Firm Advertising Decisions and Estimation Bias

The direction of bias in OLS estimates is ambiguous in the context of firm advertising decisions. Consider a static, simultaneous move advertising game among two single-product firms with demand for drugs $j \in 1,2$ given by

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

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.[15]

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

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

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

Because the demand shock ξ is unobserved to the econometrician, OLS estimates of β_1 and β_2 suffer from omitted variables bias.[16]

Advertising levels depend on consumer responsiveness to ads, which will in turn depend on the functional form and parameters of the demand system.[17] In the case of a single firm advertising (so that $a_{-j} = 0$ for that firm), optimal advertising choices that create a positive correlation between demand shocks and advertising lead to upward bias in OLS estimates. By contrast, a negative correlation between demand shocks and advertising leads to downward bias in OLS estimates.

In the case of multiple firms advertising, the levels of a are equilibrium objects of a game, where a firm's best response to rival advertising may be to either increase or decrease its own advertising. Consider an example: Lipitor has a positive demand shock in a market, which increases their return to advertising. Lipitor's heightened advertising increases Crestor's return from advertising, so both firms advertise at high levels. This would create positive correlation between Crestor ads and positive demand shocks for Lipitor. Such correlation would lead the econometrician to conclude that Crestor advertising has a 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. Section 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.[18]

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

[17] 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.

[18] This is nicely illustrated in the market for antidepressants by Shapiro (2014).

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, with the majority of states using government-run primary elections, and the remainder using party-run caucuses. The staggered nature of the primaries increases the national attention on and importance of early contests in Iowa and New Hampshire, as well as South Carolina, Florida and Nevada.[19] 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 likely to be far more valuable than in "safe states", leading to large variations in the numbers of ads different markets are exposed to (Gordon and Hartmann, 2013). For example, in October of 2008, New York, NY had 0 television ads for presidential candidates (547 for Governor/House/Senate candidates), while Cleveland, OH had 8,073 television ads for presidential candidates (and another 2,439 for Governor/House/Senate candidates). Political campaigns have preferential rules for buying advertising and both them and outside influence groups often purchase premium advertising slots that can pre-empt previously purchased advertising.[20] 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

[19] 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

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

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-premptible, 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"[21] due to the steady increase in election spending and the 2010 *Citizens United* Supreme Court ruling.[22]

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 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 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 market-month varies widely during the Jan 2007-Nov 2008 time period: half of the month-market observations during this period have zero ads, while some markets have over 20,000 political ads in a month (e.g. Denver, CO in October of 2008). Figure 1 shows the progression of the political ad shocks for the first six months of 2008, where each DMA is represented by a circle sized proportionally to the number of political ads. The mean number

[21] "Navigating Media Through Political Season", Mark Buchele, Gragg Advertising. URL: http://www.graggadv.com/navigating-media-political-season/

[22] 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.

of monthly ads by market from Jan 2007 to Nov 2008 is 535, with a standard deviation of 1600. By contrast, there are fewer drug ads in general: when combining national ads with local ads, the average number of statin ads aired in a market during a month is 98 with a standard deviation of 59. ("National" and "local" refer to the level of the ad buy, not the content.) Figure 2 shows the total number of monthly national ads for the advertised statins during our sample period, while Figure 3 shows the highest number of monthly local ads for each of the drugs (the minimum is always zero).[23] Local advertising can be a substantial portion of a firm's total advertising. While some markets receive no additional advertising, the maximum amount of local advertising is often higher than the national advertising, indicating that a substantial proportion of advertising comes from local ads and that there is substantial geographic variation.

We combine this advertising data with prescription drug usage and revenue data from two sources. First, we used Truven MarketScan data, which draws from a convenience sample of large, self-insured firms. These data represent individuals enrolled in traditional, employer-sponsored insurance. Our sample consists of market-level aggregated revenues, quantities, and covered lives.[24] 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.[25] The sample is younger than the population on the whole, and a relatively small proportion of this population takes statins. The largest branded drug captures just less than 5% of the total market, defined as all enrollees in the Truven sample.

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 markets that experience more or less than the median level of political ads during our entire sample period. Table 2 provides summary statistics; the unit of observation is the DMA. We consider age, gender, and race as well as mortality rates (a crude measure of health) and dual eligible status (a crude measure of poverty). None of the differences between the two groups are statistically

[23] 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)).

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

[25] 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.

different with the exception of % dual eligible. Consumers in markets with fewer political ads seem to be slightly poorer; if anything, income effects would only increase drug demand in above median markets 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 4 shows a scatter plot highlighting the relationship between political advertising and statin advertising, where observations are de-meaned by market and drug-year-month, and then binned to create a scatter plot of the data. This highlights the negative and non-linear relationship between political advertising and drug advertising looking across markets within a drug-month pair. For example, this shows that an increase in political ads in Iowa in January of 2008 leads to lower Crestor (Lipitor) advertising as compared to the level of Crestor (Lipitor) advertising in other DMAs in January of 2008.[26]

In order to estimate the impact of own and rival ads, we need an additional source of identifying variation. Appendix Figure 12 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 log of the number of statin advertisements for a drug in a market on the log of the number of political advertisements (in 1000s). The level of observation

[26] 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 levels, as we control for product fixed effects and allow for flexible product time trends.

is a DMA-month for January 2007 until November 2008. We include a variety of fixed effects across different specifications, including drug-year-month fixed effects which exploit solely cross-sectional variation. The OLS results show that a 10% increase in political advertising leads to a 1.2% decrease in statin advertising. To account for the fact that drug ads cannot be negative, the last columns of Table 3 estimates a Tobit model. We find a significantly larger effect: the elasticity of an individual drug's ads with respect to political ads in a market is -0.2598 in our preferred specification, implying that a 10% increase in political ads decreases each drug's ads by 2.6%. Appendix Table 15 shows the analogous results using levels instead of logs, with all results strongly negative and significant.

We address four possible concerns about this strategy. First, since the political cycle is known in advance, firms could have substituted ads to months before or after a market received a large number of political ads. In Appendix Table 13, we show that leads and lags of political advertising are not predictive of drug ads in the current month, indicating that there was not substitution to earlier or later months. Second, firms may substitute from TV advertising to other local media (radio, newspaper) when political ads displace television advertising. In Appendix Table 14, we show that total local drug ad spending is not affected by political ads once local TV ads are controlled for.[27] Third, firms may modify their detailing plans due to the displacement of their local TV ads by political ads. While we do not have data to directly test for this, discussions with industry managers led us to conclude that this is infeasible, as detailing plans are set at the annual level and cannot be quickly scaled up or down at the market level.[28] Finally, we do not believe that drug firms are responding to political advertising shocks by buying more advertising in less desirable time slots, which would create measurement error in the number of effective ads in our data. The relationship between political advertising and drug advertising is largely driven by availability and pre-emption as opposed to prices.

4.2 Graphical Evidence

First, we present a number of simple graphical analyses. We initially focus on unadvertised drugs, for which there is only one causal effect to estimate. During the time leading up to a primary, consumers are exposed to fewer ads for Crestor, Lipitor, Vytorin, and Zetia. If these ads have spillover effects on unadvertised (often generic) drugs, we would expect a drop in sales at the time of the primaries. The timing of primaries is staggered, giving a simple test of the effect. Figure 5 shows the effect of primaries on overall market share growth for unadvertised drugs. While sales

[27] 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.

[28] A greater 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.

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 13 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 6; the magnitude of the effect of primaries on Crestor sales is nearly twice as large as the effect on Lipitor sales. During this time period, Crestor and Lipitor are the primary advertisers. The overall effect is negative: the effect of a firm's advertising is not outweighed by its rival's advertising. Furthermore, these results 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 market-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 markets-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 regulatory 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:

$$Y_{tm} = \beta_1 + \beta_2 * P_{tm} + \beta_3 * I_t + \beta_4 * P_{tm} * I_t + u_m + \varepsilon_{tm} \tag{2}$$

where P_{tm} is an indicator for any political ads in market m and month t; I_t is an indicator for the

regulatory action months that prevented Lipitor from advertising; and u_m is a market 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 market 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 market-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.

Finally, we present triple difference specifications in the fourth column of Table 4. We include both Crestor and Lipitor sales and allow both political advertising and regulatory action to affect sales, and include product and market fixed effects. The coefficient of interest is still on the interaction of political advertising and the regulatory action period, as Lipitor is the omitted category. Again, we see a positive and statistically significant effect that indicates a strong business stealing effect: absent a direct effect of Lipitor ads, the impact of a decrease in rival ads is positive. By contrast, the triple interaction term is negative. By limiting Crestor's ability to advertise, local political advertising decreases Crestor sales throughout the sample period. The political ads displace business-stealing Crestor ads.

4.4 Regression Results

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

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

where X represents a vector of covariates. In all specifications, we include product and market fixed effects and cluster at the market level. Because the specification is log-log, we can interpret the coefficients as elasticities. We control for time trends in product demand in three ways; first, we simply include drug-year fixed effects. We can also include a drug-specific linear trend as Appendix Figure 11 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 affect drug sales through ads, and can allow for drug fixed effects that vary

before, during, and after the regulatory action period.

Table 5 shows the results of OLS specifications for advertised drugs; we regress this month's revenue on the averages of this month's and the previous month's advertising levels. Previous research has shown that advertising can be cumulative and/or have a lagged effect (Dubé, Hitsch and Manchanda 2005), but that the effects of DTCA can depreciate quickly (Iizuka and Jin (2007)). Furthermore, the need to obtain a prescription from a doctor is likely to delay sales after ad impressions. In each regression, the level of analysis is the DMA-month-drug. We include each of the drugs advertised during our sample period from July 2007 through November 2008 that are classified in the same in Truven Redbook class 059: Lipitor, Crestor, Vytorin, and Zetia. The dependent variable is logged drug revenue per insured individual in the market.

We control for product specific time trends in a number of different ways, including product-year fixed effects, product-regulatory action fixed effects (for before, during, and after the regulatory action), and product specific time trends; the first three specifications 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 partially determined by the political process. Therefore, we believe that 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 steal (an elasticity of -.18).

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. Our identification strategy exploits both the timing of the political process and the pulling of Lipitor ads featuring Dr. Robert Jarvik. 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.[29] In the third and fourth columns of Table 6, we still interact the regulatory action with the level of political advertising and utilize the "intensity of treatment" across areas as a second instrument. 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.[30] In this specification, we also include flexible product-time dummies

[29] 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.

[30] 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.

interacted with dummies for before, during, and after the regulatory action period. These allow consumer tastes to evolve more flexibly over time in addition to allowing the regulatory action to have a direct effect on drug sales. Our instruments are remarkably strong predictors of own and rival advertising. The test statistic of joint significance of the excluded instruments, the partial F-stat, in the first stage of our final specifications is 722.9 for own advertising and 1679.0 for rival advertising. While the partial F-statistics fall when we include the regulatory action timing in both stages of the regression, they are still large enough to alleviate weak instrument concerns (Rossi 2014).[31]

We also document the causal impact of advertising in Table 6. In the first specification with product-year fixed effects, 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.[32]

In the second specification, we show that our results are robust to including flexible product fixed effects that vary before, during, and after the regulatory action. This addresses concerns that the regulatory action could have a direct effect on revenues while also flexibly capturing time trends in product demand. The results are similar in magnitude and we cannot reject that they are statistically the same. We use several strategies to control for residual time variation while still exploiting our excluded instruments. Our preferred specification allows for linear time trends in drug quality; these are the most conservative estimates, as seen in the final two columns, of Table 6. While the estimates that allow for product-specific time trends are smaller in magnitude, we again cannot reject that they are statistically the same.

We focus on the two-month trailing average specification with drug specific time trends moving forward. Our preferred own-revenue elasticity estimates for advertised drugs is 0.0761, from column 5. This implies that a 10% increase in advertising would yield a 0.76% increase in revenue. Our preferred cross-revenue elasticity estimate is -0.0547. We believe this 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.

[31]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.

[32]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.

Table 7 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 adverting values. Appendix Table 16 shows similar results when including a stock of advertising as a control. In addition, we show that our estimates are stable when adding product-market fixed effects or if the outcome of interest is quantity (market share) instead of revenue. In all specifications, we cluster at the market 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, we run two specifications in which we omit the effect of rival ads. The results are in columns 1 and 3. These specifications explicitly violate our exclusion restriction: shocks to political advertising affect drug sales not only through changes in my own advertising, but changes in my rival's advertising as well. Therefore, we do not interpret these estimates as causal. When we do not control for rival advertising, the estimated own-advertising elasticity is much smaller (0.0163), less than 15% of the effect measured in columns 2. Both my advertising and my rivals' advertising are endogenous and the outcome of dynamic game; our identification strategy allows us to capture both effects.

Table 18 presents results that focus only on Crestor and Lipitor, where the results are slightly smaller in magnitude. Column 1 replicates the last specification in Table 6 for Crestor and Lipitor alone. The second column uses only the linear and quadratic terms as instruments, while the third column uses only the linear terms. The final specifications includes a linear time trend and product-regulatory action period fixed effects in both stages of the regression. The results are consistent across specifications. While the regulatory action is a key part of our identification strategy and has significant predictive power, our results are robust to specification that flexibly control for the impact of the regulatory action in both stages of the regression.

Table 8 estimates the spillover effects for unadvertised drugs. Column 2 replicates the last specification in Table 7 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 small, but significant spillover effect. A 10% increase in advertising for the class leads to a 0.19% increase in sales of unadvertised drugs. Our results support a model in which advertising has largely persuasive or business-stealing effects, but also spillovers to unadvertised drugs, consistent with informational effects.

4.5 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 elasticities is 0.061 for the two-month trailing average, while the estimate from the employer-sponsored sample was 0.076. 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 market-month-product level and replicate the same analysis. We have slightly fewer observations as we do not observe "new" prescriptions in every DMA-month-product cell. Otherwise, the specifications are the same as previous specifications but utilize a different dependent variable.

The results are presented in the last two columns of Table 9. We report specifications with product specific time trends. There are two key observations. First, the own advertising elasticity is nearly six times as large in magnitude for new consumers (0.349 versus 0.061 for the entire sample). Second, the rival elasticities are larger in magnitude among new consumers as well (0.200 vs. 0.037 for the entire sample).[33] We conclude that the effect is largely being driven by new consumers, rather than switchers. This has important implications for firm strategy, which we hope to explore in future research.

5 Simulations

A back-of-envelope calculation shows that our estimates are quite sensible.[34] Outside of the regulatory action period, Lipitor spent just under $10M per month on DTCA in 2008. A 1% increase in two-month advertising would represent $197,960 in extra spending.[35] US revenue for 2008 was

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

[34] In this calculation, we assume that ad prices are constant, so that an increase in spending is equivalent to an increase in the number of ads. Additional data are from Pfizer and AstraZeneca accounting filings for the year 2008.

[35] This is roughly equivalent to a single national ad.

$6.3B, and their financial statements indicate that costs of sales were between 16.8% and 46.9% of revenue.[36] Given our elasticity estimate of 0.076, this implies that a 1% increase in advertising ($197,960) increases total revenues net of costs by $211,869-$331,968. While this does not exactly equate marginal costs and marginal revenues, it is a partial elasticity: it holds rival advertising fixed. [37]

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

First, we calculate sales of advertised drugs in the absence of a business-stealing effect of competitor advertising. To do this, we set the coefficient on rival ads equal to zero in the main specification (column 4 of Table 7) and calculate the percentage change in revenue. We do not alter the level of the ads themselves. This is important as firms still benefit from the content of their own advertising. However, we are not measuring an equilibrium outcome; firms may choose higher or lower levels of advertising absent a business-stealing effect.

Table 10 presents the results. Panel A shows that business-stealing has a sizable impact on revenues. Absent the negative impact of rival ads, sales would be 23.5% higher for Lipitor and 21.1% higher for Crestor over the sample period.[38] To the extent that business-stealing is less likely to be seen as welfare-enhancing, this has important implications for policy. This also suggests that DTCA can create a prisoners' dilemma, where an individual firm has a strong incentive to advertise, but in equilibrium, all are spending more on advertising and seeing minimal effects. Panel B performs the same simulation for non-advertised drugs, which effectively eliminates spillovers from other drug advertising. In the absence of such spillovers, revenues for unadvertised drugs would fall by 8.1%. This indicates a potentially large role for welfare-enhancing spillovers in drug advertising.

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

[36] Direct costs of sales were 16.8% of revenue, while selling expenses were 30.1% of revenue. To the extent that selling expenses are commissions paid to their sales force, they may be variable costs for Pfizer.

[37] The OLS estimates would imply that a 1% increase in Lipitor advertising would increase revenues net of costs by at most $18,677.

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

Finally, we analyze the impact of changes in the regulatory environment: a ban on DTCA. This eliminates both the effect of a firm's own ads and their rival's ads. The FDA is unlikely to be concerned about firm revenues, and so the outcome of interest is the quantity (share) of consumers taking a particular drug. We proceed with our simulations based on the quantity specification in column 3 of Appendix Table 16. Table 11 shows that all firms see fewer customers under this scenario, although the effect is not identical across drugs. Figure 7 shows the distribution of the percent change from each simulation for Lipitor, Crestor, and non-advertised drugs. The results show that in the absence of DTCA, Lipitor is significantly harmed, while Crestor is harmed to a lesser degree. In general, Lipitor advertises more than Crestor during this time period. For non-advertised drugs, we see a more dispersed but still negative effect, as these drugs benefited from rival advertising.

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

5.1 Discussion

While our results present a consistent story, there are a number of caveats. First, these are short-run elasticities. Though they are much larger for new consumers, the long-run impact is unclear. Second, we do not consider selection into insurance plans or explore the role of physician agency. Given that we are looking at short-run shocks, we do not believe these factors bias our results. Third, all of our results take the decision to advertise at all as given. This decision in non-random, and our treatment effects need not generalize. Fourth, 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. Future work should explore additional strategic decisions, including formulary placement and detailing, dynamic effects, and heterogeneity both within and across classes.[39]

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 (2014)). Our results are consistent with these studies; for example, Shapiro (2014) finds that a cooperative advertising campaign that internalized spillovers would generate five times as many ads and increase category size by 13.7% for anti-depressants. Here, in addition, we argue that

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

substantial advertising expenditure is also defensive and may not provide a great deal of value from a social perspective, but that eliminating DTCA would significantly reduce the number of patients taking an effective, safe drug.

Our identification strategy is likely to be useful in a number of product markets, including other drug classes. However, additional variation will be necessary to separately identify the impact of rival advertising. Future work should also further explore the potential health consequences of DTCA; we find spillovers to unadvertised drugs, which indicated an informational, potentially welfare-enhancing role for these ads. A final caution is that these are only partial equilibrium calculations. Firms may alter their pricing or detailing strategies in response to changes in the competitive environment. Future work should further explore firm decisions to advertise in an equilibrium model. Building on the intuition in Section 2.2, we would like to explore a model of advertising competition that can be estimated and used for additional counterfactual calculations. This model should be both tractable and dynamic to capture firm incentives.

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.

Our simulations highlight the role of advertising competition, shedding light on strategic interaction between firms. Furthermore, the impact of DTCA is a question of critical policy importance; the simulations highlight the potential for an advertising ban to reduce wasteful advertising spending. While sales of unadvertised drugs fall by nearly 4%, the savings from eliminating television advertising are substantial. Our results help quantify the tradeoffs that policy makers may face when regulating pharmaceutical firms: increases in information versus wasteful advertising. Given the magnitude of our results, additional regulation of drug advertising in the United States may be welfare enhancing.

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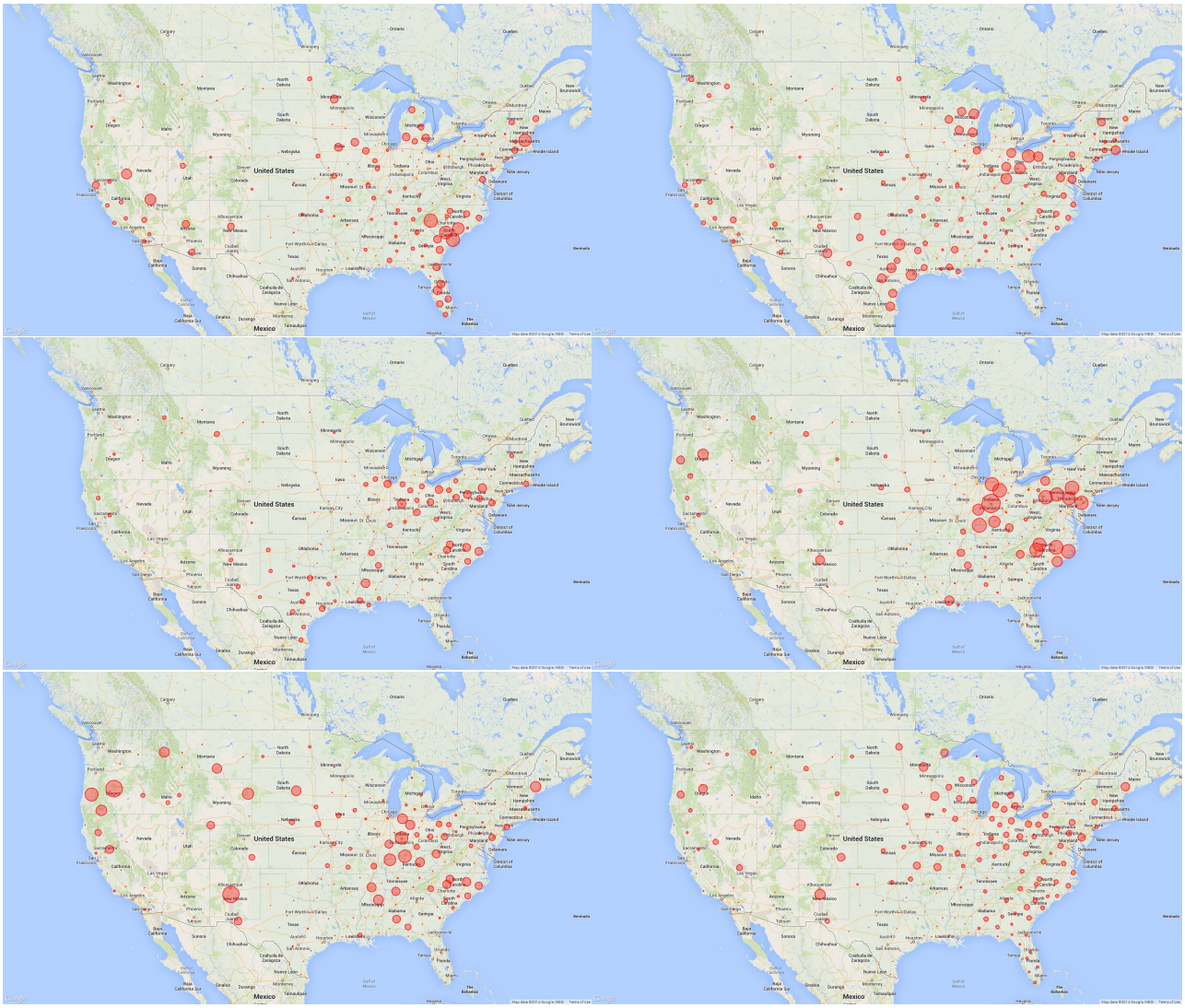
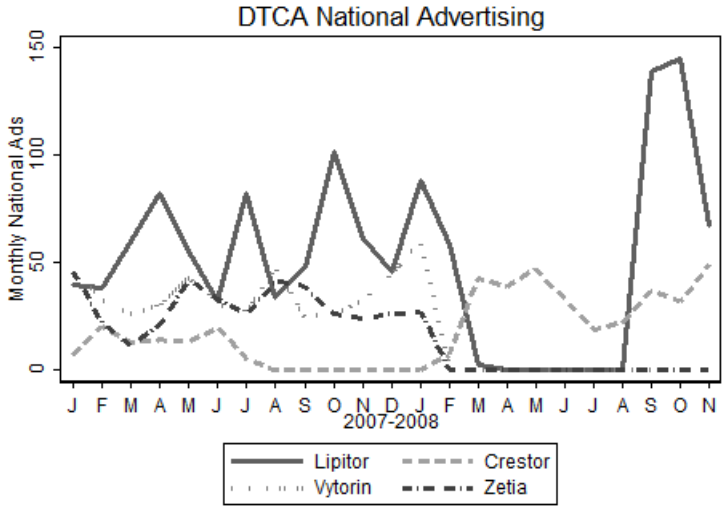
Figures

Figure 1: Political Ad Levels, January-June 2008



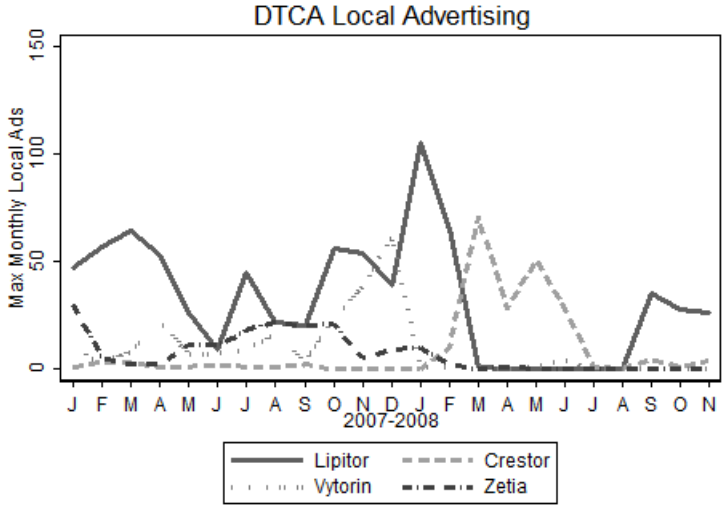
Notes: The above maps show a circle for each DMA in the USA. The diameter of each circle is proportional to the number of political ads aired in that market, in that month, for all races (Presidential, Senatorial, House, Gubernatorial). The first row are January and February; second row are March and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

Figure 2: National Pharmaceutical Ad Levels for Statins



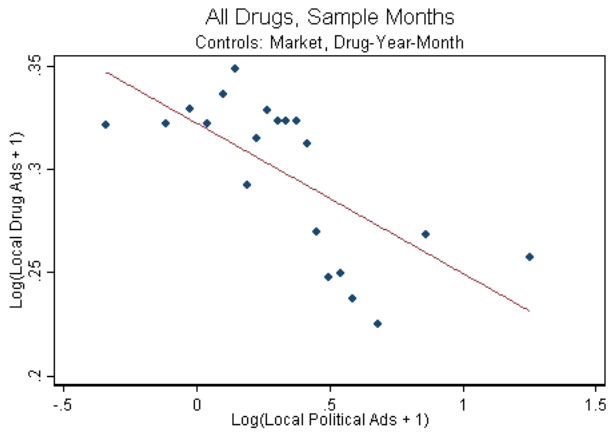
Notes: The above graphic plots national advertising spots from the Kantar data. Data spans January 2007-November 2008.

Figure 3: National Pharmaceutical Ad Levels for Statins



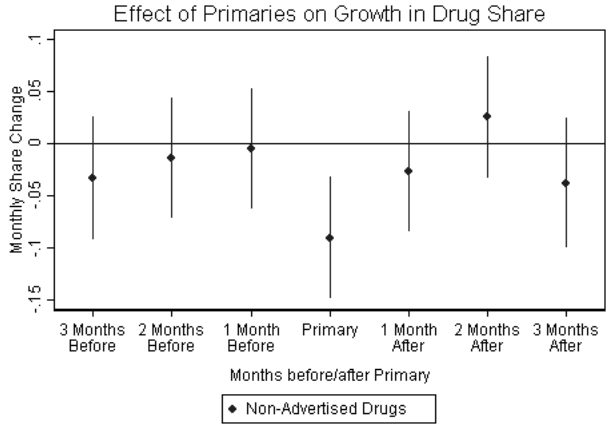
Notes: The above graphic plots the maximum of local advertising spots across DMAs from the Kantar data. Data spans January 2007-November 2008. The axes are the same as the previous figure.

Figure 4: Political Ads Displace Local Drug Ads, Binned Scatter plot



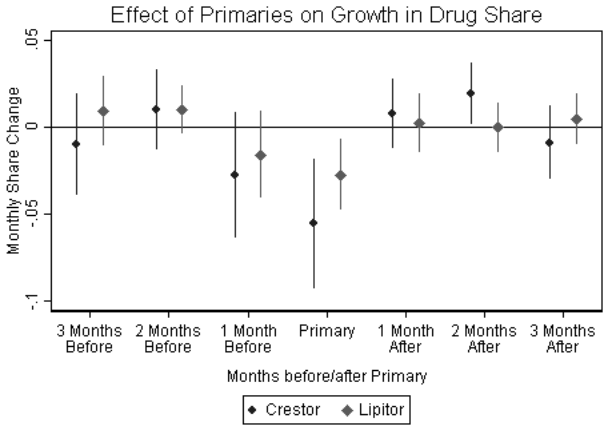
Notes: The above plots bins of observations from July 2007 to November 2008 at the market-month level after residualizing by market and drug-year-month fixed effects, and adding back the sample mean. The plot uses local drug ads, although the plot that also includes national is identical due to the drug-year-month fixed effects. Twenty bins are used. The fitted line is based on a regression of all underlying data, not only the binned values.

Figure 5: Effect of Primary Timing on Non-Advertised Statins



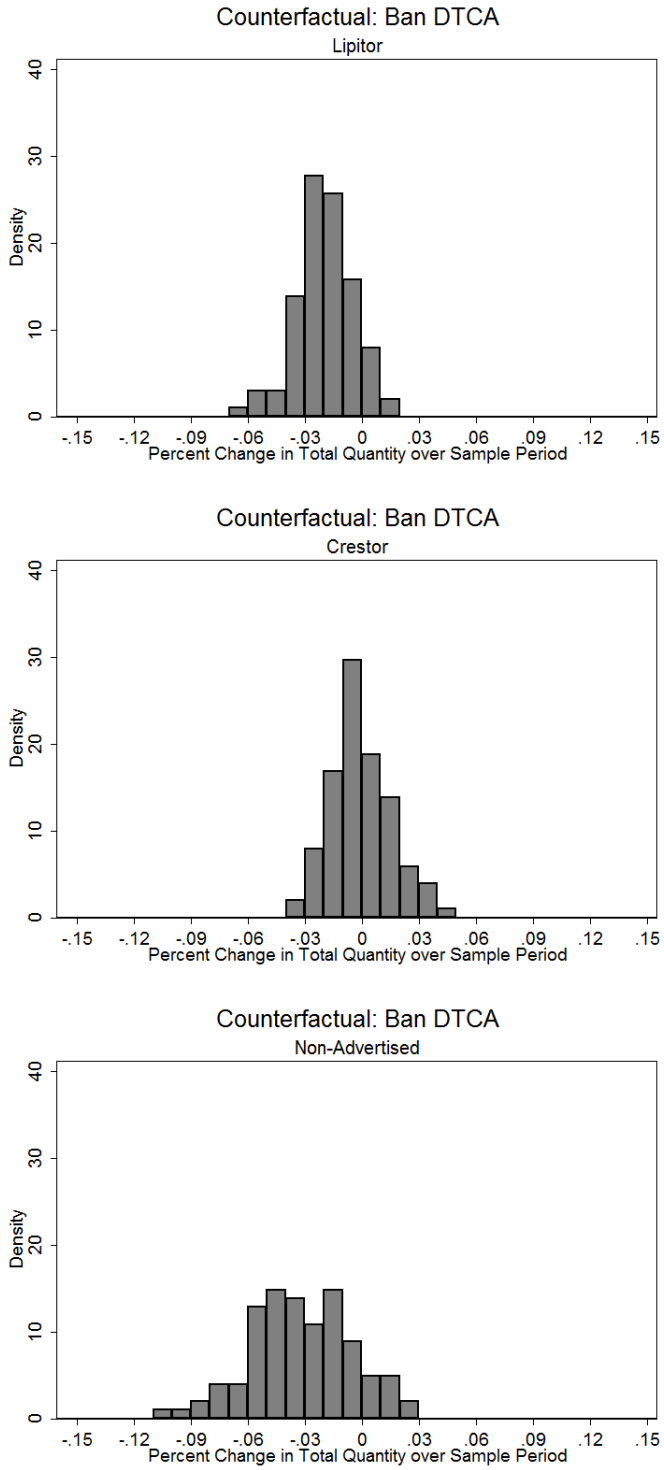
Note: The above plots estimated coefficients for timing dummies relative to a market's primary month. The dependent variable is the (one-month) change in market share, defined as the percentage of the population taking a non-advertised statin.

Figure 6: 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 7: Simulation Results: Eliminating DTCA



Note: The above plots are histograms of the change in quantity for each drug (or drug group) from bootstrapped simulations that eliminate DTCA from the market over the sample period. See section 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: 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 4: Difference-in-Difference Estimates

	Dependent Variable: Log(Days Supply)		
	(1)	(2)	(3)
P_{tm}	-0.0291^{***}	-0.0955^{***}	-0.0705^{***}
	(0.0106)	(0.0137)	(0.0174)
I_t		-0.0873^{***}	-0.1051^{***}
		(0.0160)	(0.0187)
$P_{tm} * I_t$		0.0979^{***}	0.1220^{***}
		(0.0188)	(0.0275)
$Crestor * P_{tm}$			0.0621^{**}
			(0.0259)
$Crestor * I_t$			0.1291^{***}
			(0.0349)
$Crestor * P_{tm} * I_t$			-0.1459^{**}
			(0.0565)
Products	Crestor, Lipitor	Lipitor	Crestor, Lipitor
Fixed Effects	Product, DMA	DMA	Product, DMA
Clustering	DMA	DMA	DMA
N	5,914	3,126	5,914
R^2	0.834	0.907	0.835

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

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). 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. F-statistics are for excluded instruments only. Statistical significant at the 10%, 5%, and 1% levels are denoted by *, **, and *** respectively. Standard errors are clustered at the market level. All specifications include market, year, and drug fixed effects.

Table 7: Timing Assumption Sensitivity

	Dependent Variable: Log(Revenue per Insured)			
Advertising Timing:	This Month	Two Month	Three Month	One Month Lag
Own Ads	0.0961^{***}	0.0761^{***}	0.0568^{***}	0.0613^{**}
	(0.0331)	(0.0216)	(0.0165)	(0.0276)
Rival Ads	-0.0608^{**}	-0.0547^{***}	-0.0539^{***}	-0.0392^{*}
	(0.0243)	(0.0180)	(0.0196)	(0.0233)
Controls:				
Market FEs	X	X	X	X
Year FEs	X	X	X	X
Drug FEs	X	X	X	X
Drug FE*Time Trend	X	X	X	X
Clustering	DMA	DMA	DMA	DMA
N	11,466	11,465	11,465	11,465
R^2	0.807	0.824	0.843	0.832

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). "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, 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 8: Spillovers for Non-Advertised Drugs

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

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. "Rival Ads" is constructed as log(1+X). All specifications have a "Two Month Trailing Average" as the independent variable, indicating that the "Rival Ads" is 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. 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	0.00835***	0.0610***	0.0476***	0.349***
	(0.000709)	(0.0180)	(0.00373)	(0.0728)
Rival Ads	0.00210**	−0.0369***	0.0516***	−0.200***
	(0.000830)	(0.0132)	(0.00549)	(0.0630)
Controls:				
Market FEs	X	X	X	X
Year FEs	X	X	X	X
Drug FEs	X	X	X	X
Drug FE*Time Trend	X	X	X	X
Clustering	DMA	DMA	DMA	DMA
N	11,466	11,466	10,743	10,743
R^2	0.878	0.863	0.595	0.325

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). 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: Revenue Simulations

Panel A: Simulations for Advertised Drugs			
	% Change in Revenue:	Crestor	Lipitor
(1)	Eliminating Business-Stealing	0.2108	0.2350
	Confidence Interval	(0.0046, 0.4171)	(0.0081, 0.4619)
(2)	Eliminate Political Ads	0.0166	0.0131
	Confidence Interval	(−0.0035, 0.0368)	(−0.0019, 0.0282)
Panel B: Simulations for Non-Advertised Drugs			
	% Change in Revenue:	Unadvertised Drugs	
(3)	Eliminate Spillovers	−0.0808	
	Confidence Interval	(−0.1362, −0.0254)	

Notes: Estimates from "Two Month Trailing Average" and drug-specific time trend specifications are used in all simulations. Simulation (1) sets the coefficient on rival advertising in column 3 of Panel A of Table 6 equal to zero. Simulation (2) estimates the number of drug ads in the absence of political ads, and then estimates sales at those levels of advertising. In Panel B, (3) sets the coefficient on rival advertising in column 2 of Table 8 equal to zero. Estimates are bootstrapped by re-sampling the data set, re-estimating the primary specifications, and re-computing the counterfactual exercise on the observed data. We use 100 bootstrap replications and report the 2.5%-97.5% confidence interval as well as then mean.

Table 11: Quantity Simulations

% Change in Quantity	Crestor	Lipitor	Unadvertised
Ban All Advertising	−0.0007	−0.0197	−0.0356
Confidence Interval	(−0.0301, 0.0360)	(−0.0540, 0.0104)	(−0.0917, 0.0228)

Notes: Estimates from "Two Month Trailing Average" and drug-specific time trend specifications are used in all simulations. The dependent variable is the log of the market share of a product. The simulation sets the coefficient on own and rival advertising equal to zero. Estimates are bootstrapped by re-sampling the data set, re-estimating the primary specifications, and re-computing the counterfactual exercise on the observed data. We use 100 bootstrap replications and report the 2.5%-97.5% confidence interval as well as then mean. Figure 7 shows the distributions of the simulated outcomes.

Appendix

Supplemental Appendix For Online Publication

A Model Simulation

A.1 Results

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

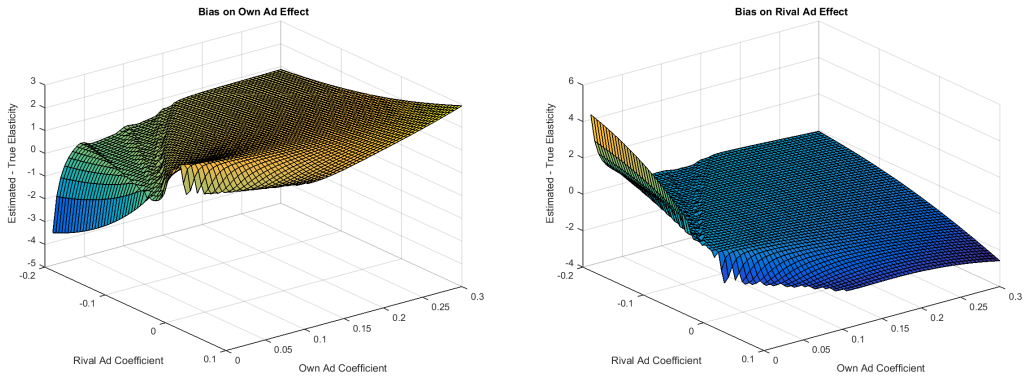
$$\begin{aligned} u_{ijm} &= \alpha_j + \beta_1 ln(1+a_{jm}) + \beta_2 ln(1+a_{-jm}) + \xi_{jm} + \varepsilon_{ijm} \\ u_{i0m} &= \varepsilon_{i0m}, \end{aligned}$$

where u_{i0} denotes the utility of the outside good. Assuming 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. The per-unit cost of advertising is c, and profit per unit sold is ρ. Firm profits in this model are given by $\pi_{jm} = \rho D_{jm} - ca_{jm}$, 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 (full details are in Appendix A).

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 below show the difference between estimated and analytic elasticities. The level of the surface indicates the bias in different areas of the parameter space: it is apparent that there can be upward (greater than zero) or downward (less than zero) bias in both own and rival advertising elasticities. In no simulation were own and rival elasticities both estimated with less than 5% bias.[40]

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

Figure 8: 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 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 9 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 9: OLS Bias with One Firm

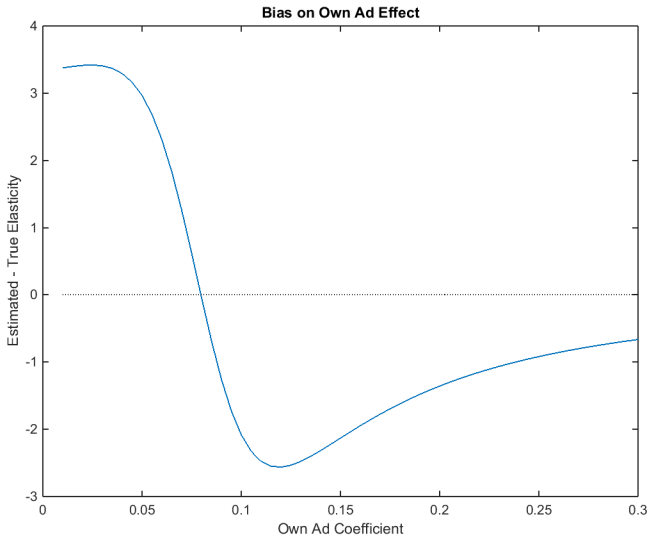


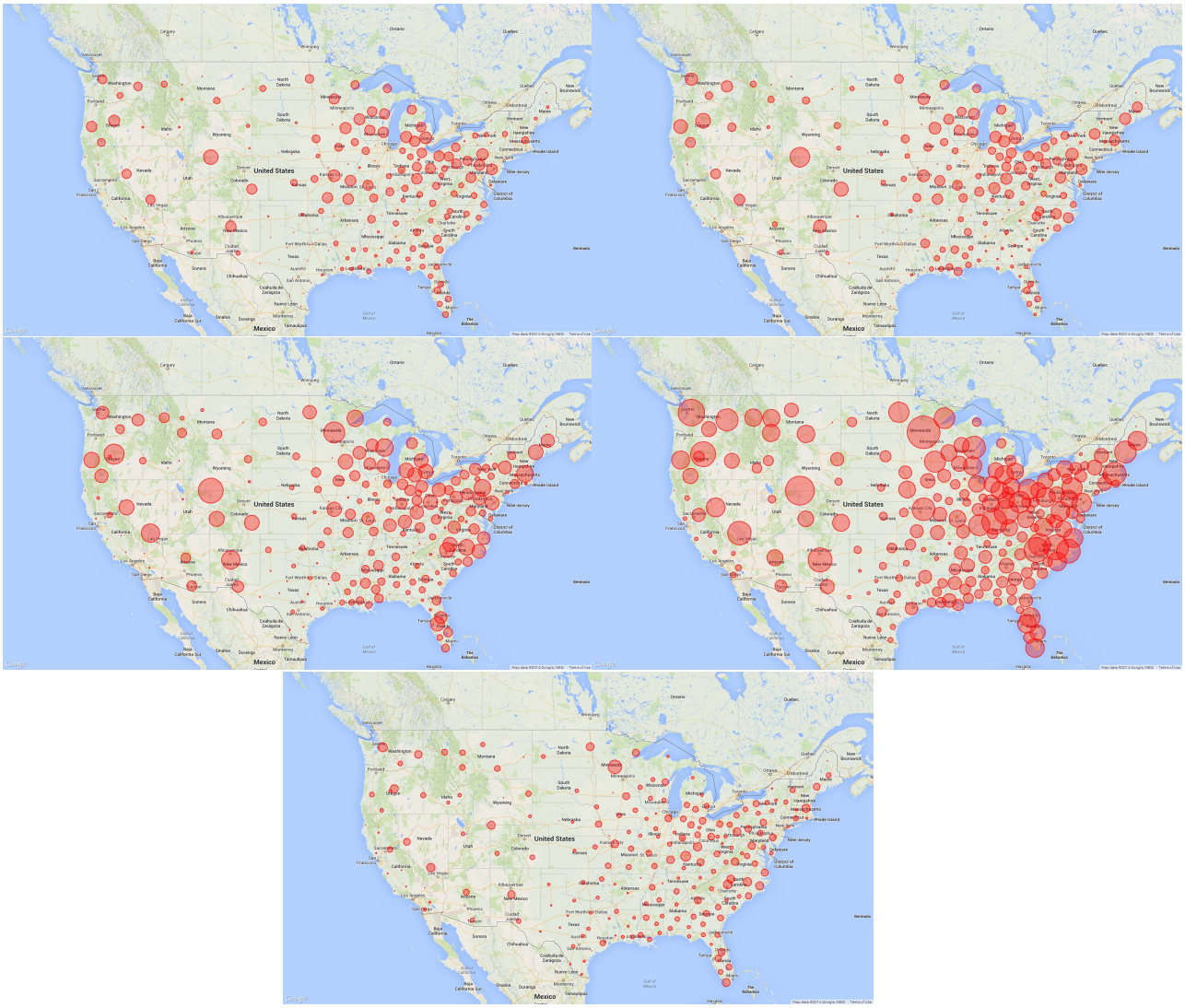
Table 12: Sample Model Simulation Results

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.

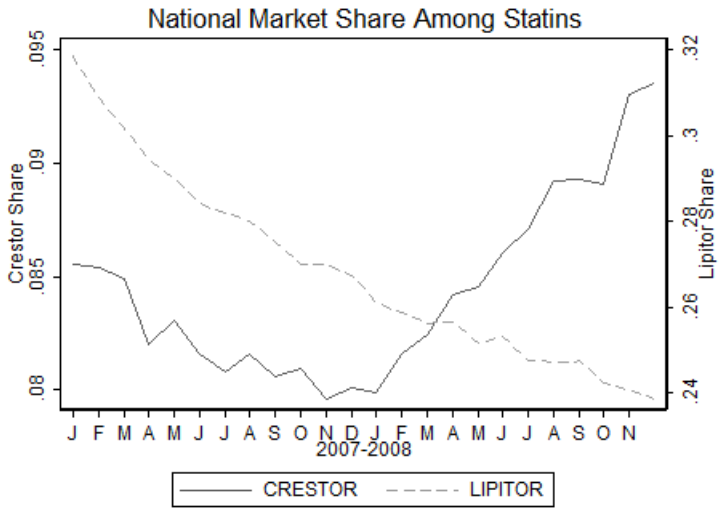
B Additional Summary Statistics and Robustness Checks

Figure 10: Political Ad Levels, July-November 2008



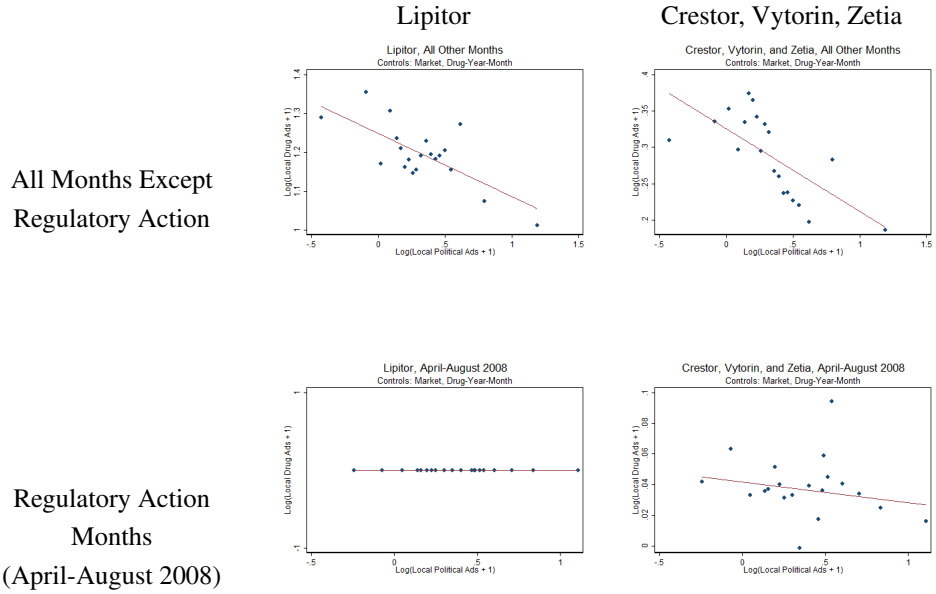
Notes: The above maps show a circle for each DMA in the USA. The diameter of each circle is proportional to the number of political ads aired in that market, in that month, for all races (Presidential, Senatorial, House, Gubernatorial). The first row are July and August; second row are September and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

Figure 11: Time Trends



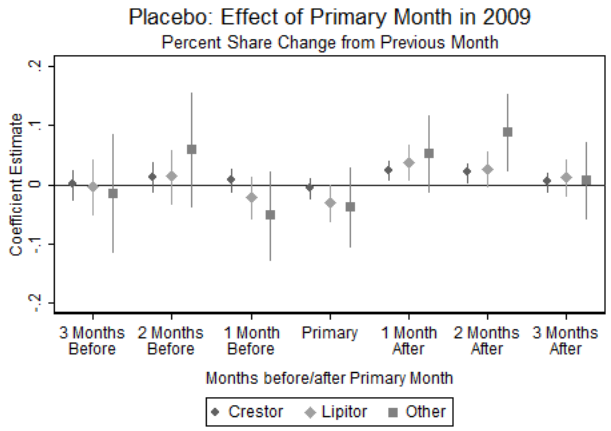
Notes: The above graphic plots the share of Lipitor and Crestor from the Truven data as a percentage of total category sales over the period of January 2007-November 2008. Note different axes.

Figure 12: Instrument Effect Heterogeneity



Notes: The above graphic plots binned scatterplots to show that political ads displace drug ads, as Figure 4, for different sub-samples of the data. "Regulatory Action" months refer to the months when Lipitor was banned from advertising by congress.

Figure 13: 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 13: Robustness: No Substitution to Earlier/Later Months

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

Notes: Regressions combine the Wisconsin and Kantar data sets 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 14: 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 15: Political Ads Displace Drug Ads

	Dependent Variable: Statin Ads					
Model:	OLS	OLS	OLS	Tobit	Tobit	Tobit
Political Ads (1000s)	-0.2668^{***}	-0.2102^{***}	-0.2102^{***}	-1.2548^{***}	-0.6824^{***}	-0.7536^{***}
	(0.0245)	(0.0263)	(0.0264)	(0.1198)	(0.0992)	(0.0228)
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	0.193	0.244	0.366	0.140	0.219	0.312

Notes: Unit of observation is the market-month-product combination. Data (source: Wisconsin and Kantar) span 17 months from July 2007 to November 2008 and 210 markets and include Crestor, Lipitor, Vytorin, and Zetia. OLS and Tobit standard errors clustered at the market-year-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 16: Robustness Checks (IV Results)

	Dependent Variable: Log(Revenue per Insured)		Dependent Variable: Log(Days Supply per Insured)	
Own Ads (2-Month Trailing)	0.0753^{***}	0.0669^{***}	0.0696^{***}	0.0601^{***}
	(0.0220)	(0.0213)	(0.0213)	(0.0209)
Rival Ads (2-Month Trailing)	-0.0536^{***}	-0.0466^{***}	-0.0522^{***}	-0.0438^{***}
	(0.0182)	(0.0177)	(0.0178)	(0.0175)
Jan.-Jun. '07 Ads	0.0013^{***}			
	(0.0004)			
...Controls:				
Market FEs	X	X	X	X
Year FEs	X	X	X	X
Drug FEs	X	X	X	X
Drug FE*Time Trend	X	X	X	X
Drug-Market FEs		X		X
Clustering	DMA	DMA	DMA	DMA
N	11,465	11,465	11,465	11,465
R^2	0.831	0.945	0.822	0.946

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). 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. 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.

Table 17: Effect of Business Stealing (IV Results)

	Dependent Variable: Log(Revenue per Insured)			
	This Month		Two-Month Trailing Average	
Log Own Ads	0.0136^{***}	0.0961^{***}	0.0117^{***}	0.0761^{***}
	(0.0018)	(0.0331)	(0.0015)	(0.0216)
Log Rival Ads		-0.0608^{***}		-0.0547^{***}
		(0.0243)		(0.0180)
...Controls:				
Market, Year, Drug FEs	X	X	X	X
Drug FEs*Time Trend	X	X	X	X
N	11,466	11,466	11,465	11,465
R^2	0.849	0.810	0.850	0.827

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). "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: Crestor and Lipitor Specifications, First Stage Robustness

	(1)	(2)	(3)	(4)
Own Ads	0.0320**	-0.00481***	0.0575**	0.0626**
	(0.0147)	(0.0178)	(0.0280)	(0.0287)
Rival Ads	-0.0280**	-0.0409***	-0.0483**	-0.1154**
	(0.00121)	(0.0146)	(0.0229)	(0.0578)
Market Fixed Effects	X	X	X	X
Year Fixed Effects	X	X	X	X
Product Fixed Effects	X	X	X	X
Prod-Time Trend	X	X	X	X
Prod-Regulatory Action Fixed Effects				X
Order of Polynomials, First Stage	Third	Second	First	First
Observations	5,914	5,914	5,914	5,914
Adjusted R-Squared	0.831	0.819	0.810	0.834

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). 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. Statistical significant at the 10%, 5%, and 1% levels are denoted by *, **, and *** respectively.