

Physician-Industry Interactions: Persuasion and Welfare

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July 17, 2018

Abstract

In markets where consumers seek expert advice regarding purchases, firms seek to influence experts, raising concerns about biased advice. Assessing firm-expert interactions requires identifying their causal impact on demand, amidst frictions like market power. We study pharmaceutical firms' payments to physicians, leveraging instrumental variables based on regional spillovers from hospitals' conflict-of-interest policies and market shocks due to patent expiration. We find that the average payment increases prescribing of the focal drug by 73 percent. Our structural model estimates indicate that payments decrease total surplus, unless payments are sufficiently correlated with information (vs. persuasion) or clinical gains not captured in demand.

1 Introduction

In many markets, consumers seek expert advice before making a purchase decision. In health care and financial services, for example, consumers often select a product in conjunction with an intermediary, typically a physician or certified financial adviser. Experts can provide valuable information about complex products, helping to increase market efficiency. However, experts frequently receive various forms of remuneration from firms selling in the market, raising concerns that their advice may be biased. Whether and how expert-firm interactions

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The data used in this paper were generously provided, in part, by Kyruus, Inc. We gratefully acknowledge financial support from the Wharton Dean's Research Fund, Mack Institute, and Public Policy Initiative. Gi Heung Kim and Donato Onorato provided excellent research assistance. Abby Alpert, Alexandre Belloni, Colleen Carey, Pierre Dubois, Gautam Gowrisankaran, Robin Lee, and Amanda Starc, as well as numerous seminar and conference audiences, provided helpful discussion and feedback. Any errors are our own.

impact welfare are contentious and important policy questions, animating debates over recent initiatives in the United States to address conflicts of interest, including the Department of Labor’s Fiduciary Rule (2016), and in the health care context we study, the Physician Payment Sunshine Act (2010).¹

In this article, we examine the causal and welfare impact of payments from pharmaceutical firms to cardiologists in the market for statins, an important class of drugs that reduce cholesterol and the likelihood of heart attack and stroke. In general, both policy-making and empirical research regarding expert-firm interactions are complicated endeavors. (1) Interactions are often not transparent or systematically recorded. (2) Interactions are not randomly chosen, making it difficult to infer the causal links among firm activities, expert advice, and consumer decisions. (3) Assessing the welfare implications of any causal effect must take into account other frictions such as market power, negotiated prices, insurance, and agency problems. We address each of these challenges in this study.

In the US, many physicians receive payments and other in-kind compensation, such as meals, from manufacturers of products they prescribe, inject, or recommend.² Recently, data on payments from firms to physicians in the US has become publicly available: select pharmaceutical and medical device firms began self-reporting payments around 2010; all such firms have been required to report payments on [OpenPayments.CMS.gov](https://www.cms.gov/medicare/physician-and-medical-device-payment-transparency-under-the-sunshine-act) since mid-2013.

Section 2 describes the setting, data, and identification strategy. We link data on physician-firm-year-level payments to physician-drug-year-level prices and quantities observed in a large market – the Medicare Part D prescription drug insurance program for the elderly in the US. We focus on meals, which are the single most popular in-kind payment from pharmaceutical firms to physicians. Meals are also particularly relevant for our counterfactual analyses, having been subject to statutory bans in several states and health systems.³ We further focus our examination on the market for branded statins in 2011-2012, as it is one of a few important markets with complete payment data prior to the introduction of [OpenPayments.CMS.gov](https://www.cms.gov/medicare/physician-and-medical-device-payment-transparency-under-the-sunshine-act).⁴ During this period, there were two branded statins (Pfizer’s

¹For commentary, see, e.g. [Rosenbaum \(2015\)](#); [Steinbrook et al. \(2015\)](#), or the May 2017 issue of the *Journal of the American Medical Association*, which was entirely devoted to this topic.

²As noted in [Scott Morton and Kyle \(2012\)](#), promotion of pharmaceuticals embodies both potential inducements to use firms’ products and some scientific information. In our study, we focus on payments from manufacturers to physicians, which is just one component of firms’ promotional strategies. [Millenson \(2003\)](#) presents an overview of these practices for drugs and medical devices.

³Massachusetts, Minnesota, and Vermont had certain statutory gift bans during 2011-2012. As described in [Larkin et al. \(2017\)](#), nineteen academic medical centers nationwide introduced limits or bans on pharmaceutical representatives providing meals, branded items, and educational gifts between October 2006 and May 2011.

⁴The transparency introduced by [OpenPayments.CMS.gov](https://www.cms.gov/medicare/physician-and-medical-device-payment-transparency-under-the-sunshine-act) may alter the nature of physician-industry interactions; we consider this an interesting area for future research as more data become available.

Lipitor and AstraZeneca’s Crestor) – 59 percent of cardiologists received a meal from one or both in 2011 – and several generic substitute statins. The expiry of Lipitor’s patent at the end of 2011, and ensuing generic entry, also provides variation that allows us to disentangle market power effects for our welfare analysis.

We employ an identification strategy that accounts for the potential endogeneity of these meals by exploiting regional variation in Academic Medical Centers’ (AMCs) Conflict of Interest (CoI) policies, which were designed to curb interactions between physicians and industry. We document significantly lower rates of sponsored meals in regions with strict AMC CoI policies – e.g., those that ban on-site interactions – a result we argue is consistent with economies of scale in firms’ marketing efforts.⁵ Importantly, conditional on rich controls, AMCs’ policies are plausibly unrelated to the latent preferences of *unaffiliated physicians* in the same region, motivating our use of these policies as instrumental variables.

Discussions with industry participants, supported by our data, indicate that payments between a firm and physician are highly persistent over time, implying that analysis based on within-physician meal variation is unlikely to recover the full magnitude of the treatment effect relevant for examining the welfare impact of these payments. Our identification strategy is thus cross-sectional in nature, making it critical to include a rich set of physician- and market-level controls related to prescribing. The size of the potential control set we assemble, and the fact that we allow these variables to enter the model nonlinearly and interacted with other variables, creates a dimensionality and sparsity problem, which we address by drawing on the recent literature at the intersection of machine learning and econometrics. We follow a procedure outlined in [Belloni et al. \(2017\)](#), using LASSO regressions to select controls and an “orthogonalized” two-stage least squares (2SLS) regression to estimate the treatment effect of interest in a way that is robust to small errors in the variable selection process.⁶ Section 3 presents details on the estimation procedure and our instrumental variables (IV) regression results regarding the effects of meals on prescribing.

Existing empirical studies on this topic document positive correlations between firms’ payments to physicians and prescribing of those firms’ products.⁷ The study that is perhaps closest to ours is [Carey et al. \(2017\)](#), which analyzes similar payment data, but in contrast uses physician fixed effects to address physician selection and focuses on patients who switch prescribers to address patient selection. Across all drugs in their data, they find that

⁵We measure these policies using the American Medical Students Association (AMSA) Conflict of Interest Report Card scores. In related work using these data, [Larkin et al. \(2017\)](#) examine the direct effects of conflict-of-interest policies, finding that they have a modest, but significant negative effect on prescribing.

⁶LASSO stands for “least absolute shrinkage and selection operator.” It is a commonly-used form of penalized regression that shrinks the least squares regression coefficients in a high-dimensional linear model towards zero ([Varian 2014](#)).

⁷[Kremer et al. \(2008\)](#) provides a review of early research on this topic.

payments from a firm raise expenditures on promoted products by around 6 percent. For statins 2011-12, we find that the average payment increases prescribing of the focal drug by 73 percent. We attribute the difference primarily to our cross-sectional strategy, which seeks to identify the effect of an entire relationship, vs. a panel data strategy, which estimates the incremental effect of an additional payment.⁸

To put our estimate in context, it is equivalent to an increase in promoted drugs' cardiovascular prescribing market share from 2.7 percent, the sample average, to about 4.6 percent. This increase is close to half of a standard deviation in the observed prescribing heterogeneity across physicians. This IV estimate is larger than the corresponding OLS estimate, consistent with pharmaceutical sales representatives successfully targeting meals to physicians who would otherwise prescribe less of the firm's drug. We present evidence on heterogeneity in treatment effects suggesting that most of this effect is driven by increasing prescribing among low and moderate prescribers of branded statins. We also show that there are no marginal returns to higher-value meals, conditional on providing any meal. Finally, our results are robust to a placebo test performed within states banning or limiting meals.

Even as meals (and associated interactions) causally shift physician decisions toward the sponsoring firm, the implications for efficiency are ambiguous (Inderst and Ottaviani 2012). For example, if patients consume too little of a product due to manufacturer market power or other frictions, then payments may move utilization toward the optimum, though perhaps at great cost to patients/payers. To account for this, in Section 4, we estimate a structural model of demand and supply in order to shed light on the welfare effects of demand inducement in the presence of important real-world distortions: market power, strategic interactions, negotiated prices, insured demand, and behavioral hazard.⁹ In this dimension, our approach adds to several recent studies on causal welfare effects of marketing in oligopoly settings: e.g., Dubois et al. (2018) examine the effects of a ban on junk food advertising; and Shapiro (2018), Sinkinson and Starc (2018), and Alpert et al. (2015) estimate causal effects of direct-to-consumer advertising (DTCA) on drug utilization.

In order to examine the interactions between market power and payments to physicians, we estimate a nested logit model of statin choice, integrating our demand estimation with the machine learning procedures from Belloni et al. (2017), similar to Gillen et al. (2015).

⁸At least two other published studies we are aware of incorporate physician-level fixed effects: Mizik and Jacobson (2004) and Datta and Dave (2016). In a market such as statins, where relationships likely pre-date the beginning of the payment data, a fixed effect intuitively controls for the unobserved relationship. In a new market, where relationships and prescribing behavior are still being established, the effect captured by a fixed effect estimator would be more nuanced.

⁹We use the term "behavioral hazard" to mean the phenomenon recently characterized in Baicker et al. (2015), in which patients' decision utility over a treatment may, for a number of potential reasons, be biased upward or downward relative to its true medical value.

Lipitor’s patent expiration results in generic entry, price changes, and changes in meals for both Lipitor and Crestor. Because the timing of generic entry is driven by patent length, it provides plausibly exogenous variation that traces out substitution patterns as different products respond differently to entry. In addition to substantial meal effects, our demand estimates indicate low price sensitivity in this insured and subsidized population.

We also estimate a bargaining model between upstream manufacturers/distributors and insurers to capture the forces driving the point-of-sale prices that insurers pay for pharmaceuticals. Our results are sensible in that the estimated bargaining parameters are consistent with branded manufacturers receiving a large portion of the surplus they create, while competition among many manufacturers drives down margins on generics dramatically.

The estimated demand and supply models allow us to consider the equilibrium response of prices and quantities to a ban on meals, and map those outcomes into welfare. We also analyze a counterfactual efficient benchmark scenario, where meals are banned and out-of-pocket prices are set at marginal cost. These exercises draw on the logic in [Inderst and Ottaviani \(2012\)](#), where hidden kickbacks allow firms to expand market share without lowering prices, and welfare implications depend on the primitives and strategic interaction.¹⁰

In the market studied here, payments cause the market to overshoot the efficient level of branded statin usage. Our baseline estimates, with consumer welfare measured by our revealed preference demand estimates and meals assumed to be purely persuasive, indicates payments lower consumer welfare by \$190M relative to a counterfactual equilibrium with payments banned. These consumer losses outweigh producer gains, so that payments decrease total surplus as well. However, if the additional patients receiving statins due to meals are clinically appropriate (perhaps because agency or other biases cause physicians to under-prescribe), then the clinical literature would imply a 10 percent increase in life years gained due to payments, which would be worth about \$1.2B at standard valuations.¹¹

2 Setting, Data, and Empirical Strategy

In this Section, we describe the market for statin medications, our data sources, and our approach to identifying causal effects of payments from statin manufacturers to prescribers.

¹⁰One might speculate that the disclosure policy embodied in the Physician Payment Sunshine Act (2010) would be analogous to a ban in its effects on conflicts of interest. However, as noted in [Inderst and Ottaviani \(2012\)](#), disclosure may have limited real-world effects. E.g., [Pham-Kanter et al. \(2012\)](#) find that early state-based physician payment disclosure laws had a negligible to small effect on physicians switching from branded therapies to generics and no effect on reducing prescription costs.

¹¹The assumption that marginal patients consuming statins due to payments are appropriate for statin therapy is strong. We provide these results as a suggestive caveat specific to this setting, due to the strong evidence that statins are underutilized in practice (see summary in [Baicker et al. \(2015\)](#)).

Statin medications reduce blood levels of low-density lipoprotein cholesterol (LDL, or “bad” cholesterol), and in turn reduce the risk of coronary heart disease and heart attacks. We focus on cardiologists treating enrollees in the Medicare Part D program in 2011 and 2012. This sample and time horizon are useful for several reasons. (1) We have physician-firm interaction data for the two major on-brand statin producers during this time. Pfizer (which produces Lipitor) and AstraZeneca (which produces Crestor) accounted for 49 percent and 33 percent of statin revenue in Medicare Part D in our sample in 2011, respectively. This is before the Open Payments website created under the Physician Payment Sunshine Act was published, implying that we can analyze the effects of payments prior to the shock of broad disclosure. (2) These statins were each the chief source of revenue from cardiologists’ prescribing for these two firms, with Lipitor accounting for 84 percent of Pfizer’s cardiologist-driven revenues and Crestor similarly accounting for 80 percent of AstraZeneca’s cardiologist-driven revenues. Thus, if a Pfizer or AstraZeneca representative were taking a cardiologist out to lunch in this time period, it is very likely that statins were the focus of any drug-related discussions.¹² (3) Lipitor’s patent expiration offers a large and visible shock to statin prices and substitutes, helping to identify demand curves.¹³

Statins are generally considered to be effective drugs with few side effects. The American College of Cardiology (ACC)’s 2013 guidelines recommended statin therapy for adults with elevated risk of atherosclerotic cardiovascular disease; full adoption under these guidelines would have increased statin use by 24 percent ([American College of Cardiology 2017](#)). Statins are close substitutes for most patients, but atorvastatin (Lipitor) and rosuvastatin (Crestor) are available as high-intensity statins appropriate for some patients with elevated risk.¹⁴

2.1 Physician-Firm Interactions

Firms’ promotional strategies generally include direct-to-consumer advertising, “detailing” to physicians, advertisements in venues targeted to physicians, and various payments. “Payments” from firms to physicians include meals that are bundled with sales details, compensation for travel, speaking, consulting, and education, research-related payments, and payments related to physicians’ firm ownership interests. In the current study, we focus on payments in the form of meals, which are expected to be accompanied by sales effort.

¹²Cardiologists as a specialty account for 10 percent of Part D statin claims. Even though cardiologists write relatively few prescriptions, they are targeted because specialist prescriptions are sustained by primary care physicians ([Fugh-Berman and Ahari 2007](#)).

¹³[Carrera et al. \(2018\)](#) find that cross-sectional variation in patients’ copays has a modest impact on statin prescribing ($\varepsilon = -0.31$), while large changes in average copays due to patent expiration imply much larger responses ($\varepsilon = -0.76$).

¹⁴A moderate-intensity statin is expected to reduce LDL by 30 to 50 percent, while a high-intensity statin would reduce LDL by 50 percent or more ([ConsumerReports 2014](#)).

Field sales forces are considered “the most expensive and, by consensus, highest-impact promotional weapon” in pharmaceutical firms’ arsenals (Campbell 2008). Sales representatives target prescribers with product presentations regarding safety, efficacy, side effects, convenience, compliance, and reimbursement. This targeting approach is described in greater detail below, particularly as it relates to our identification strategy.

We examine the statin market at the end of 2011, 15 years after Lipitor was introduced and 8 years after Crestor was introduced. Statins as a class have been available since Mevacor was introduced in 1987 by Merck. By 2011, there was likely very little information regarding the atorvastatin and rosuvastatin molecules that was not available to cardiologists. The classic justification for physician-industry interactions is that they allow physicians to learn about a drug’s features. Below, when we document evidence of a causal effect of interactions on prescribing, it is unlikely to be due to firms providing new information about the promoted drugs, though interactions may act as persuasive nudges or reminders.¹⁵

2.2 Sample and Data Sources

Our analysis of prescribing focuses on physicians treating enrollees in Medicare Part D (see Appendix A.1 for detail on the program). The structure of Medicare Part D implies that enrollees should be sensitive to price variation across and within branded and generic drugs.¹⁶ This sensitivity may be muted by various frictions, including enrollees’ limited understanding of coverage and physicians’ imperfect agency.¹⁷ Part D plan issuers’ strategies and profits are heavily regulated by the Centers for Medicare and Medicaid Services (CMS), but they have both motive and opportunity to constrain costs through formulary design (drugs’ placement on tiers), negotiations with drug manufacturers, and negotiations with pharmacies.¹⁸

2.2.1 Data on Medicare Part D, prescribing, and provider characteristics

We obtain data on physician demographics, specialties, and affiliations from CMS’ Physician Compare database, which contains all physicians treating Medicare patients.¹⁹ Each physician’s practice location is matched to his or her relevant Hospital Service Area (HSA)

¹⁵In the Appendix Section B.3, we document trends in scientific publications on these statins to support our assumption that 2011-12 is not characterized by any flurry of new information.

¹⁶See Chandra et al. (2010) and Goldman et al. (2007) for helpful reviews of the literature.

¹⁷E.g., enrollees are more responsive to current prices than marginal prices, and respond disproportionately to salient coverage changes such as copay changes for entire drug classes (Abaluck et al. 2018).

¹⁸E.g., Duggan and Scott Morton (2010) show that initial introduction of Part D in 2006 lowered the price of drugs by increasing insurer market power relative to drug manufacturers.

¹⁹See: <https://data.medicare.gov/data/physician-compare>.

and Hospital Referral Region (HRR) according to the Dartmouth Atlas.²⁰

Prescribing behavior is based on the publicly-available CMS Part D claims data for 2011 and 2012.²¹ These claims data describe total prescription claims and spending for each prescriber-drug-year. The prescriber information includes physicians' National Provider Identifier (NPI), which allows us to link claims data to the Physician Compare database as well as industry interaction data. Drugs are defined by brand and molecule name (if the drug is "generic," these two are equivalent). Claims may vary in terms of unobserved drug dosages, days supplied, and formulation. However, we are unaware of any evidence that industry payments target particular dosages or presentations, so we follow prior studies in analyzing claims directly (Einav et al. 2015).

Our price variables are the plan enrollment-weighted average point-of-sale and unsubsidized out-of-pocket prices per one-month supply for each Part D pricing region-drug-year from the Medicare Part D Public Use Files. One month is the modal supply per claim.

Using the name of the drug, we also match branded drugs in the prescribing data to their respective manufacturers using the FDA's Orange Book and match all drugs to their WHO Anatomical Therapeutic Classification (ATC) codes. The ATC codes provide a hierarchy of drug categories that reflect similarities in drug mechanism and disease intended to treat. In that way, it usefully mimics the choice sets faced by physicians. We focus mostly on two measures of prescribing outcomes: (1) log quantity of the focal drug's claims; and (2) (for the structural analysis) the focal drug's share of all cardiovascular (ATC code = "C") and statin (ATC code = "10AAC") prescribing within physician-year.

2.2.2 Data on manufacturer payments to providers

Although federally mandated reporting of manufacturer-provider payments did not begin until 2013, nationwide interest had been growing for some time. By 2010, states had begun to institute their own payment limitations and/or public reporting rules;²² a number of high-profile lawsuits found conflicts of interest between physicians and manufacturers to be

²⁰See: <https://www.dartmouthatlas.org> for more. HRRs represent regional health care markets for tertiary medical care. Each HRR has at least one city where both major cardiovascular surgical procedures and neurosurgery are performed. HSAs are local health care markets for hospital care. An HSA is a collection of ZIP codes whose residents receive most of their hospitalizations from the hospitals in that area. There are 3,436 HSAs and 306 HRRs in the US.

²¹See: <https://www.cms.gov/Research-Statistics-Data-and-Systems/Research-Statistics-Data-and-Systems.html>.

²²The District of Columbia, Maine, and West Virginia required disclosure of payments and gifts to physicians prior to our time horizon; Massachusetts, Minnesota, and Vermont required disclosure and had certain statutory gift bans.

a punishable offense;²³ and calls from politicians and patient advocacy groups were gaining momentum (Grassley 2009). Amidst this growing concern, a number of firms, including Pfizer and AstraZeneca, began to publicly release data on payments to physicians, often due to legal settlements.²⁴ These documents are the basis of our payments data, which were generously shared by Kyruus, Inc.²⁵

Our analyses primarily focus on two “payment” variables: (1) a dummy that equals one if a physician received a meal from the focal drug’s firm in a given year; and (2) the dollar value of all meals for the physician-firm-year. As discussed below, the vast majority of “general (non-research)” payments during the period of interest were in the form of a meal. Moreover, meals are the most likely type of physician-firm interaction to be related to pure persuasion. This is in contrast to, for example, payments associated with consulting or speaking activities, which are likely to be payments for services rendered. This restriction is not intended to imply that other payment types do not influence physicians. However, the limited numbers of these payments inherently limits the welfare impact of any biases they might induce in prescribing patterns of the physicians receiving them. Our identification strategy is also not designed to examine quasi-random variation in these types of interactions.

2.3 Data Set Construction and Summary Statistics

Starting with the full sample of cardiologists in the Medicare Physician Compare database, as identified by their self-reported primary specialty, we restrict our sample to “active” Medicare prescribers with at least 500 Part D claims on average in 2011 and 2012; this is approximately the 10th percentile of claims per physician-year. The final sample used in our analyses contains about 15,000 cardiologists.²⁶

The first panel of Table 1 summarizes the cardiologist-year-level quantity data for the six statins (two branded, four generic) available during 2011-2012.²⁷ The effect of entry by generic atorvastatin in December 2011 is clear – in its first full year of availability, this new

²³For example, in 2009 Eli Lilly paid a \$1.4 billion fine following allegations of the off-label promotion of its drug Zyprexa (United States Department of Justice 2009).

²⁴The existence of some voluntary disclosures is not entirely surprising. In 2009, the industry trade association PhRMA introduced a voluntary Code on Interactions with Healthcare Professionals limiting informational presentations to the workplace and entertainment to “modest meals,” and prohibiting trips to resorts, sponsored recreation, and gifts to the physicians. For more, see: <https://projects.propublica.org/d4d-archive/>.

²⁵The raw disclosures were published in a wide variety of formats both across firms and within firms over time. In order to account for irregularities in formatting – primarily of names – a machine-learning algorithm was developed by Kyruus to create a disambiguated physician-level dataset of payments from Pfizer and AstraZeneca in 2011 and 2012. Appendix B.2 compares this data to that made publicly available post-Sunshine Act and finds no evidence of any major biases or censoring in our data.

²⁶Table A3 presents summary statistics for the full set of control variables and instruments.

²⁷These account for more than 99 percent of statin claims and expenditures in this period.

alternative accounted for roughly 24 percent of cardiologists' statin claims, while Lipitor's share dropped from 22 percent in 2011 to about 5 percent in 2012.²⁸

The second panel of Table 1 summarizes prices. As expected, branded Lipitor and Crestor both had high out-of-pocket (OOP) and point-of-sale (POS) prices in 2011, relative to generics. In 2012, generic atorvastatin entered with intermediate OOP and POS prices due to limited generic competition in its first year. While other generic drugs' prices were somewhat lower in 2012 than in 2011, both Pfizer and AstraZeneca increased their POS prices in 2012. Finally, while Crestor's OOP price was the same in 2011 and 2012, Lipitor's OOP price increased dramatically, as insurers removed Lipitor from their formularies (Appendix A.2 provides further detail on 2012 pricing).

The bottom panel of Table 1 describes the average payment amounts (all and the meal-related subset) from Pfizer and AstraZeneca. Meal-related payments account for more than 90 percent of these interactions, with the vast majority of these meals being valued at less than \$150.²⁹ The Table also includes the percentiles of the non-zero distributions for each variable, which highlights the extremely skewed nature of payments. It is clear that Pfizer and AstraZeneca implemented different strategies in this timeframe: cardiologists were about two and a half times as likely to receive a meal from AstraZeneca compared to Pfizer in 2011 (48 percent vs. 18 percent), and conditional on receiving a meal, AstraZeneca's median meal value per cardiologist was twice as large in 2011 (\$38 vs. \$24).

2.4 Identification Strategy – Responsiveness to Meals

Our primary identification strategy exploits variables that shift the costs of interacting with physicians, but which are plausibly exogenous to those physicians' latent preferences over drugs or responsiveness to interactions. The intuition of this approach is that drug firms, directly or via their marketing contractors, typically first determine marketing budgets and strategies based on aggregate market characteristics. Then the firms' "boots-on-the-ground" representatives use their knowledge of specific physicians to target high-value individuals.

Firms' marketing models can be very detailed and data-driven, and pharmaceutical sales forces maintain rich databases on prescribers' practice characteristics, prescribing behavior, and history of interactions with the firm (Campbell 2008). They then target physicians

²⁸A number of papers have examined market dynamics around these loss-of-exclusivity events. In particular, Aitken et al. (2018) detail the shifts in prices and quantities surrounding these events for a number of high-profile molecules. They cover the Lipitor case we study here, outlining the legal events and entry of generics during the time period in our sample. They also note Pfizer's response to the event of instituting an aggressive coupon program around this time, but importantly, this program was not relevant to Medicare enrollees, nor even well taken up by those eligible. Thus, we do not know of any evidence that Pfizer or AstraZeneca made any strategic responses that we do not capture via prices or payments.

²⁹The data does not specify the total number of interactions within a year for each physician.

Table 1: Summary Statistics

	2011 (Pre)			2012 (Post)				
Claims	\bar{q}	$\sigma(q)$		\bar{q}	$\sigma(q)$			
Cardio	2,404	1,993		2,590	2,029			
Statins	418	349		468	375			
Lipitor	92	96		22	29			
(Atorvastatin)	-	-		112	107			
Crestor	64	83		66	82			
Other Generics (3)	246	224		226	206			
Prices	\$ <i>OOP</i>	\$ $\sigma(OOP)$	\$ <i>POS</i>	\$ <i>OOP</i>	\$ $\sigma(OOP)$	\$ <i>POS</i>		
Lipitor	39	5	140	88	8	163		
(Atorvastatin)	-	-	-	11	1	31		
Crestor	42	8	137	39	7	160		
Other Generics (3)	5	0.5	12	4	0.4	9		
Payments	Frac > 0	\$ $p50 _{>0}$	\$ $p90 _{>0}$	\$ $p99 _{>0}$	Frac > 0	\$ $p50 _{>0}$	\$ $p90 _{>0}$	\$ $p99 _{>0}$
Lipitor (All)	0.19	28	149	5,137	0.03	23	157	3,576
(Meals)	0.18	24	119	358	0.02	20	128	430
Crestor (All)	0.48	40	123	274	0.53	77	260	19,950
(Meals)	0.48	38	114	230	0.52	71	237	920

Note: $N=28,290$ cardiologist-year observations during 2011-2012. Payments include all non-research interactions, for example, speaking fees, consulting payments, reimbursements for travel, and meals.

based on the expected incremental costs and benefits of sales effort. The expected benefit of interacting with a given physician depends on the size and appropriateness of the physician's patient panel, the physician's latent preferences over substitute products, and the physician's expected responsiveness to inducement. Costs include the labor costs of additional sales representatives, the opportunity costs of diverting sales effort from other physicians, and any direct costs of the interaction (e.g., meal expenditure); they also implicitly include factors that limit or prohibit access for sales representatives. For example, the consulting firm ZS Associates publishes the *Access MonitorTM* survey, which focuses on characterizing pharmaceutical representative access to physicians. The 2015 *Access MonitorTM* report notes several key factors restricting access: academic medical centers' restrictive access policies, specialty-specific physician employment by hospitals and health systems that have central purchasing or otherwise limit physicians' autonomy, pressures on physicians that limit available time for firm interaction, etc. (Khedkar and Sturgis 2015).

Pharmaceutical sales territories are defined by geography and other organizing principles, such as therapeutic area (Campbell 2008). Given the fixed costs of deploying a sales force to a market, individual physicians' interactions with pharmaceutical firms will experience spillover effects from market-level characteristics. Thus, conditional on variables that proxy for individual physicians' attractiveness to pharmaceutical representatives – which may be correlated with physicians' underlying preferences – variables that proxy for attractiveness of *other* physicians in the same geographic market are useful instruments for interactions.

The variables we focus on for identification are academic medical centers' (AMCs') con-

flict of interest policies. These are described in detail in [Larkin et al. \(2017\)](#). We rely on data on AMC's conflict of interest policies from the American Medical Student Association's (AMSA) conflict of interest scorecard. The AMSA scores evaluate the strictness of AMC policies regarding physician interactions with pharmaceutical/device companies, including salesperson access to AMC facilities, gifts to physicians, and enforcement of the policies.³⁰

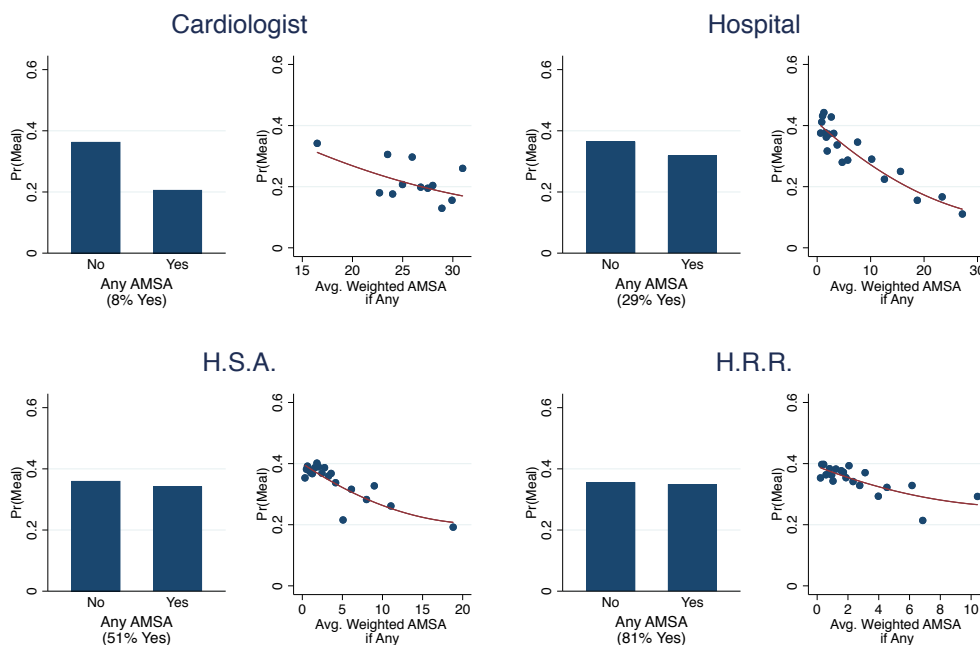
To get a sense of our approach, consider Sioux Falls, SD and Lubbock, TX, two cities whose major HRRs surround moderately-sized state universities with associated AMCs: the University of South Dakota and Texas Tech University, respectively. These markets each have 24-28 cardiologists in our sample. However, the USD AMSA CoI score is 30, vs. only 24 at Texas Tech, and many more of the cardiologists in the Sioux Falls region are faculty than in the Lubbock region. These differences are associated with large differences in meal rates: 16 percent in the Sioux Falls region vs. 41 percent in the Lubbock region. Simultaneously, we see large differences in prescribing of branded statins: 2.1 percent of cardiovascular drug prescriptions by Sioux Falls-area cardiologists are for branded statins, vs. 2.9 percent for Lubbock-area cardiologists. Of course, there may be other important differences in Sioux Falls vs. Lubbock that we want to account for, including the illness of the patient population, insurance rates, managed care penetration, and so on, which motivates our control inclusion and selection procedure described below.

Moving to the aggregate numbers, the top two panels in [Figure 1](#) show the raw extensive and intensive margin relationships between meal receipt and AMSA CoI scores at the individual cardiologist (left panel) and hospital (right panel) levels. At the individual cardiologist level, AMSA scores are shown for AMC faculty; the faculty linkage is from the Association of American Medical Colleges (AAMC) faculty roster. Only 8 percent of sample cardiologists are AMC faculty and there are strong extensive and intensive margin associations between AMC linkage and meals – faculty are a little more than half as likely to receive meals as non-faculty, and within faculty, those with the strictest (highest) AMSA CoI policies are a little more than half as likely to receive meals as those with the weakest (lowest) CoI policies. At the hospital level, scores are aggregated across all cardiologists affiliated with the hospital, other than the focal cardiologist (non-faculty physicians receive

³⁰In every school year since 2007, medical schools have been asked to submit their policies to the AMSA for rating. Each institution's policy is graded in 13 different categories, including Gifts, Consulting, Speaking, Disclosure, Samples, Purchasing, Sales Reps, On-Campus, Off-Campus, Industry Support, Curriculum, Oversight, and sanctions for Non-Compliance. For each category except Oversight and Non-Compliance, the institution is assigned a numerical value ranging from zero to three. A zero is awarded if the institution did not respond to requests for policies or declined to participate; a one if no policy exists or the policy is unlikely to have an effect; two if the policy represents "good progress" towards a model policy; and a three if the policy is a "model policy." We generate aggregate AMSA scores for each institution; this aggregate ranges from 11 to 31-32 in 2011-2012.

zeros, which mechanically shifts the scale of the x-axis toward zero).³¹ The extensive margin association between AMSA scoring and meals is muted at the hospital level – the 29 percent of sample cardiologists whose affiliated hospitals have any AMSA scores have slightly lower meal incidence. However, the intensive margin association is strongly negative.

Figure 1: Raw AMSA Score – Meal Correlations



Note: Each panel plots raw meal probabilities (averaged over all firm-years). Bar graphs: sample is split by whether cardiologists are exposed to any AMSA score at a given level of aggregation (where the Cardiologist panel is for faculty only), with the percentage of exposed cardiologists in parentheses. Binned scatter plots: conditional on being exposed to any AMSA score, these plots show average meal probabilities vs. average AMSA score, with a marker for each quintile of the relevant AMSA score distribution.

While we might expect conflict of interest policies to have large effects on pharmaceutical company interactions with the cardiologists and hospitals under their jurisdiction, that does not make them valid instruments. The exclusion restriction may fail due to direct effects of conflict of interest policies on norms regarding prescribing, or due to unobservable factors correlated with selection into more restrictive policies. We address this concern by leveraging identification from jackknifed versions of AMSA scores at the HSA and HRR levels. These raw relationships are shown in the bottom two panels in Figure 1. There is now essentially

³¹Each analysis is performed at the cardiologist level, and each AMSA variable excludes the lower units of aggregation associated with the same focal cardiologist. I.e., “Hospital” AMSA scores exclude the scores of the focal cardiologist; “HSA” (hospital service area) scores exclude the scores associated with cardiologists at the focal cardiologist’s hospital; and “HRR” (hospital referral region) scores exclude the scores associated with cardiologists in the focal cardiologist’s HSA.

no extensive margin effect – cardiologists exposed to any AMSA policy in their HSA/HRR are about as likely to receive meals as cardiologists with no local AMC faculty. However, exposure to stricter local AMC conflict of interest policies still has a significant negative effect on meals, even though those policies do not directly govern the focal cardiologist’s own or affiliated hospital’s behavior.

This first stage relationship is assumed to be driven by marketing economies of scale that result in local spillovers at the HSA and HRR levels. This exclusion restriction assumption is consistent with conversations with current and former pharmaceutical sales executives and pharmaceutical marketing consultants. Under this assumption, instruments based on jackknifed HSA and HRR AMSA variables are exogenous with respect to the focal cardiologist’s own preferences over drugs and susceptibility to inducement, conditional on a rich set of controls for cardiologist and market characteristics. We cannot test this directly, but we examine placebo checks on this assumption in Section 3.

2.4.1 A note on “meals” and cross-sectional identification

Our identification strategy has two nuances that deserve further discussion. First, our estimates of the effects of “meals” on prescribing behavior may be proxying for the effects of a long-term sales relationship between a physician-firm pair. Second, our cross-sectional instrumental variables approach is intended to address the endogenous selection of physicians into receiving meals based on their patients’ diagnoses and preferences, as well as the physicians’ own preferences.

We consider our approach to be appropriate for several reasons. First, as many researchers have noted, extensive margin effects of payments are large and the evidence on heterogeneity of effects by payment size is mixed (see, e.g., [Carey et al. \(2017\)](#), [Yeh et al. \(2016\)](#), and [DeJong et al. \(2016\)](#)). We confirm this in our analyses in Section 3: cardiologists’ tendency to prescribe firms’ drugs is not increasing significantly in the dollar value of interactions. Second, our conversations with pharmaceutical marketing specialists and consultants indicate that physician-firm relationships involve repeat interaction by design. This is confirmed in our data, in which payments are highly persistent across years: about 70 percent of cardiologists that received a meal from AstraZeneca in 2012 also did so in 2011.

This places our study in contrast to [Carey et al. \(2017\)](#), [Datta and Dave \(2016\)](#), [Mizik and Jacobson \(2004\)](#), in which the researchers include physician fixed effects to take out persistent unobserved differences across physicians.³² The average treatment effect of a pharmaceutical firm providing one fewer meal to a physician in the context of a long physician-firm

³²[Carey et al. \(2017\)](#) contains an additional innovation: they address patient panel endogeneity using patients’ moving behavior.

relationship, or of providing the first meal to a physician at the initiation of a physician-firm relationship, may be very different than the average treatment effect of turning an entire relationship on or off. Thus we argue that a cross-sectional identification strategy is most appropriate for considering a counterfactual ban on meals, our interest here. This emphasizes the importance of controlling for a rich and flexible function of physician, hospital, and regional variables, to account for heterogeneity in prescribing patterns.³³

3 The Effects of Meals on Prescribing

In this Section, we describe our main instrumental variables (IV) specifications and results regarding the causal effects of physician-firm interactions (meals) on prescribing for the branded drugs Lipitor (Pfizer) and Crestor (AstraZeneca) for 2011 and 2012. We estimate a linear IV model:

$$\ln(q_{jdt}) = \beta^m 1_{\{m_{jdt}>0\}} + x'_{jdt} \beta_{jt}^x + \beta_{jt} + \varepsilon_{jdt} \quad (1a)$$

$$1_{\{m_{jdt}>0\}} = x'_{jdt} \gamma_{jt}^x + z'_{jdt} \gamma_{jt}^z + \gamma_{jt} + \mu_{jdt} \quad (1b)$$

where the utilization outcome q_{jdt} for a cardiologist d and branded molecule j in year t depends on whether or not the drug's manufacturer provided a meal to the cardiologist in that year ($1_{\{m_{jdt}>0\}} = 1$). In each equation, we control for a potentially high-dimensional set of exogenous covariates x_{jdt} that proxies for heterogeneity in physicians' patient populations and other preference-relevant factors. We allow for the effects of these variables to vary by year and drug (and thus firm). For example, high-volume prescribers may have different preferences over Crestor and Lipitor; similarly, AstraZeneca and Pfizer may employ different physician-targeting models, and those models likely respond differently to Lipitor's patent expiration. The focal parameter β^m describes the effect of industry interaction on the physician's treatment decisions. Since these interactions do not randomly occur, we are concerned that simple ordinary least squares (OLS) estimation of Eq. 1a will over- or underestimate β^m , which motivates the instrumental variable approach using z_{jdt} . The term $z_{jdt} \gamma^z$ in Eq. 1b represents the exogenous component of the physician targeting function that is based only on market-level variation in nearby AMC conflict of interest policies described in Section 2.4.

The cross-sectional nature of our identification strategy makes a rich set of controls and

³³A particular strategic decision of firms that may be correlated with meals are other advertising efforts (i.e. DTCA). But since these sorts of initiatives typically target broad geographic territories (i.e. via television markets), we believe that we can adequately account for any impact they may have with our regional level controls under the assumption that firm's advertising decisions are some function of these controls.

flexible functional form especially important. Relatedly, we have no a priori theory for the functional form relating our potential instruments to meals. As we allow the control and instrument sets to grow larger and more flexible, we run into the issues of sparsity and collinearity which have been the topic of a growing literature at the intersection of econometrics and machine learning.

3.1 LASSO Regression and Orthogonal Controls/Instruments

Here we discuss how we construct large, flexible sets of potential controls \tilde{x} and instruments \tilde{z} , and our strategy for estimating β^m . The first challenge is to identify subsets of relevant instruments and controls, noting that z may only be a valid set of instruments conditional on a relatively small set of variables x in \tilde{x} , whose identities are a priori unknown. This presents a variable selection problem, which may be prone to error – under a given variable selection method, irrelevant variables may be erroneously included or relevant variables may be erroneously excluded. To address this issue, we use the procedure from [Chernozhukov et al. \(2015\)](#) (which also presents a particularly clear description of the problem – see [Belloni et al. \(2017\)](#) for a general treatment). We use a series of LASSO regressions to select controls for the prescribing and meals equations, and construct “orthogonal” moment conditions that immunize estimation against small errors in model selection. In the remainder of the paper, we call this the “orthogonal 2SLS approach” (O2SLS) for the sake of brevity.

3.1.1 Potential Control Variables

Table 2 below outlines the sets of variables and transformations thereof included in our estimation procedure. To summarize, we include sets of variables that capture the number of patients a physician treats with certain types of drugs, variables that describe a wide range of characteristics related to the sizes and types of own and adjacent organizations, and variables regarding the insurance and health status of local populations. Together, these form our potential controls set \tilde{x} . We generate these variables for four levels of observation: individual cardiologists, hospitals, HSAs, and HRRs, with each unit subsuming the last.

We identify each physician’s drug-class-specific historical claims volumes using the 2010 Medicare Part D claims data. We calculate volume metrics at two levels of ATC drug classes: Cardiovascular and Statins. Statins are a subset of Cardiovascular drugs. We control for historical cardiovascular drug claims to proxy for latent characteristics of the local patient population. The HRR-, HSA-, and Hospital-level volume metrics are calculated using jack-knife procedures in which each physician is excluded from the Hospital-level measures, each physician’s hospital is excluded from the HSA-level measures, etc. This is an

Table 2: Overview of Potential Control Set

Cardiologist	Hospital	HSA & HRR
2010 Claims, Statins ¹	2010 Claims, Statins ¹	2010 Claims, Statins ¹
2010 Claims, Cardiovascular ¹	2010 Claims, Cardiovascular ¹	2010 Claims, Cardiovascular ¹
2010 Claims, Total ¹	2010 Claims, Total ¹	2010 Claims, Total ¹
Num. Practice Zip Codes ¹	Num. Cardio. & Doc. Affiliated ¹	Num. Cardio. & Doc. Affiliated ¹
Num. Hospital Affiliations ¹	Num. AAMC Affils. ²	Num. AAMC Affils. ²
Num. Practice Affiliations ¹	Num. AAMC Faculty ²	Num. AAMC Faculty ²
Num. Specialties ¹	Share Doc. AAMC Faculty ²	Share Doc. AAMC Faculty ²
Is AAMC Faculty ²	Hospital Beds & Admissions ³	Teaching Hosp. Bed & Adms. Share ³
		Medicare Advantage, N Eligible & %Covered ⁴
		Pop., %Uninsured & %Medicaid ⁵

Note: Hospital, HSA and HRR aggregations of each variable are averaged at the cardiologist. In each level of aggregation, the next level down associated with the focal cardiologist is excluded in a jackknife procedure. Superscripts indicate data source. ¹CMS Part D Public Use Files & CMS Physician Compare Data; applicable to all claims data. ²American Academic Medical Center Faculty Roster. ³American Hospital Association Annual Survey. ⁴CMS Medicare Advantage enrollment and landscape files. ⁵Behavioral Risk Factor Surveillance Survey.

effort to minimize collinearity and endogeneity while mimicking a firm’s marketing efforts, wherein resources are allocated to regions, hospitals, and physicians with larger patient pools indicated for the firm’s drug.

In addition to cardiologist-specific claims history, specialty, practice characteristics, and faculty status (as additional proxies for patient population size and complexity), we also control for a number of hospital- and market-level affiliation and density metrics. These include: number of cardiologists and doctors, number of academic medical centers and associated faculty, share of physicians who are faculty, total hospital beds and admissions, and total teaching hospitals beds and admissions share.

Finally, we include HRR-level data from CMS and the Behavioral Risk Factor Surveillance System (BRFSS). From CMS, we obtain local Medicare Advantage penetration variables, noting that managed care penetration may impact price sensitivity. Relatedly, from the 2011 BRFSS we identify three additional variables: (1) population; (2) the uninsurance rate; and (3) the Medicaid enrollment rate. Together these variables proxy for variation market size, health insurance coverage, and incomes.

Beginning with these raw volume and attribute variables (46 in total), we include: squared terms; log-transformations; and interactions of all linear terms. This yields a set \tilde{x} of 1,173 candidate control variables (for each drug-year, yielding 4,692 in total).

3.1.2 Instruments

As described in Section 2.4, we rely on spillovers from local hospitals’ conflict of interest policies to generate pseudo-random identifying variation in meal receipt. We have four candidate instruments: HSA-level average and faculty-weighted average AMSA score across

all AMC faculty not affiliated with the focal cardiologist’s hospital; HRR-level average and faculty-weighted average AMSA score across all AMC faculty not affiliated with hospitals in the focal cardiologist’s HSA.³⁴ In theory, each variable could contribute independent identifying variation: the first and third describe strictness of local AMCs’ conflict of interest policies; the second and fourth add information on how many local physicians are faculty; and the different levels of geography may have distinct, additive effects on sales force allocations.

We have no a priori theory on the functional form of the relationship between these instruments and meals, so starting with our 4 baseline instruments, we again include: squared terms; log-transformations; and interactions of all linear terms. This yields a set of 18 candidate instruments for meals for each drug-year (72 in total).

3.1.3 LASSO Selection Results

Following Chernozhukov et al. (2015), the O2SLS algorithm is as follows, omitting subscripts for simplicity:³⁵

1. LASSO of q on \tilde{x} , selecting a covariate vector x^q , and use it to form a post-LASSO residual $r^q = q - \hat{q}$
2. LASSO of $1_{\{m>0\}}$ on \tilde{x} and \tilde{z} , selecting a covariate vector (x^{m1}, z^{m1}) , and use it to form a post-LASSO prediction $\hat{1}_{\{m>0\}}^1$
3. LASSO of the prediction $\hat{1}_{\{m>0\}}^1$ on \tilde{x} , selecting a covariate vector x^{m2} , and use it to form a second post-LASSO prediction $\hat{1}_{\{m>0\}}^2$
4. Create the residualized endogenous regressor $r^m = 1_{\{m>0\}} - \hat{1}_{\{m>0\}}^2$ and the orthogonal instrument $z^m = \hat{1}_{\{m>0\}}^1 - \hat{1}_{\{m>0\}}^2$
5. 2SLS regression of r^q on r^m , instrumenting with z^m , to obtain $\hat{\beta}^m$

See Belloni et al. (2017) for a helpful review of how this form of orthogonal moment construction “immunizes” the estimation of the parameter of interest β^m to small errors in the selection of the model controls.

While the large sets of controls aid with flexible prediction, they do make standard approaches to assessing results by looking at tables of parameters unwieldy. In an effort to shed some light in this direction, Table 3 displays the ten most “important” control variables selected by the LASSO in the claims $\ln(q_{jdt})$ and meals $1_{\{m_{jdt}>0\}}$ equations for Pfizer in

³⁴In generating each of the “faculty-weighted” instruments, we assign AMSA scores of zero to non-faculty, intuitively measuring the regional importance of the AMC.

³⁵See Appendix C.1 for details on penalty selection in the LASSO.

2011, along with the effect of removing the given control variable on the R^2 of the post-LASSO-selected “flexible” model with polynomials, log transformations, and interactions. The analogous results for AstraZeneca and Pfizer in 2012 are available in Appendix Tables A5-A7.

Table 3: Important Variables, Pfizer 2011

Utilization Equation ($y: \log(Claims_{jdt})$)		Meal Equation ($y: 1_{\{m_{jdt}>0\}}$)	
X Var.	Flexible ΔR^2	X Var.	Flexible ΔR^2
NPI '10 Claims, Statins	-.156	NPI '10 Claims, Statins	-.083
HRR Medicare Advnt. Penet.	-.007	HSA N Faculty	-.056
HRR Faculty Shr.	-.007	HRR Faculty Shr.	-.054
HRR N Cardiol.	-.007	Hosp-Card. '10 Claims, Statin	-.053
HRR Pop. %Medicaid	-.006	HRR Medicare Advnt. N Elgbl.	-.052
HSA N AMCs	-.005	Hosp-Card. '10 Claims, Year	-.042
HSA Medicare Advnt. Penet.	-.005	HSA Medicare Advnt. N Elgbl.	-.035
Hosp. N AMC Affls.	-.005	HSA-Card. '10 Claims, Statin	-.034
HRR N Docs.	-.005	Hosp. N Cardiol.	-.031
HRR N AMCs	-.005	HRR N AMCs	-.031
		Z Var.	Flexible ΔR^2
		HRR AMSA CoI, Wgt.	-.021
		HRR AMSA CoI	-.015
		HSA AMSA CoI, Wgt.	-.002
		HSA AMSA CoI	-.001

In Panel A, as expected, we see that an individual cardiologist’s claims history has a large effect on current utilization. We also generally see large effects from the share of local physicians that are AMC faculty, and from the share of Medicare Advantage enrollment. In the right Panel B, we see that past use of statins is also strongly correlated with meals, as are several market density variables. The bottom of Panel B shows the effects of market-level AMSA scores on meals. HRR-level AMSA scores are stronger predictors of meals than HSA-level scores when all are included. All of these patterns are also borne out graphically and in the more detailed first stage results in Appendix C.2.

3.2 Main Results

Table 4 shows the estimated effects of meal receipt on the logarithm of claims. Here we focus on the pooled regressions where variable selection and parameters vary by drug-year, but the meal parameter β^m is assumed constant across drug-years in our main specification.³⁶

Columns (1) and (2) present the results of estimating Equation 1a via OLS. Without controls (1), β^m indicates that meal receipt is correlated with a 39 percent increase in promoted drug prescribing. However, when we include our base set of (46×4) controls for cardiologist,

³⁶Specifications that allow drug-year specific meal effects are summarized below in Section 3.2.1, and full results can be found in Appendix C.4.

hospital, and market characteristics (2), the estimate drops to 16 percent, emphasizing the likely non-random selection on part of the drug firms.

Column (3) continues to use our base set of controls, and estimates the 2SLS regression, using our base set of (4×4) instruments for meals. The IV estimate of β^m is significantly larger than the OLS, indicating the average physician receiving a meal will increase their utilization by 83 percent. There is still, however, the chance that even this relatively large set of controls may not sufficiently capture regional and physician variation in prescribing, leaving omitted variable bias that drives up the effect through a correlation between the instruments and unobserved determinants of prescribing.

Table 4: Main Results, $\log(\text{claims})$

	(1)	(2)	(3)	(4)	(5)	(6)
β^m	0.385*** (0.00888)	0.157*** (0.00811)	0.833*** (0.144) [9.49]	0.733*** (0.195) [60.36]	0.925*** (0.184) [74.71]	0.660*** (0.158) [93.96]
N_{obs}	52419	52419	52419	52419	52419	52419
Spec.	OLS	OLS	2SLS	O-2SLS	O-2SLS	O-2SLS
Incl. X		X	X	$L(X)$	$L^+(X)$	$L^{min}(X)$
X Set		B	B	E	E	E
N_X		184	184	1375	685	1917
N_Z			16	31	24	40

Variable sets: “B” = baseline, totaling 46 X and 4 Z variables; “E” = exploded baseline set via interactions, logs, and squares, totaling 1173 X and 18 Z variables; all models with X controls also include firm-year fixed effects. The preferred LASSO penalty λ (Col. 4) is chosen via cross-validation (“CV”) as the largest λ within 1 s.e. of the out-of-sample MSE-minimizing λ ; alternative control sets $L^+(X)$ and $L^{min}(X)$ formed using penalties that are one log point higher than the 1 s.e. λ and the MSE-minimizing λ , respectively. $N_{X/Z}$ indicates the total number of control/instrumental variables selected across firm-years, averaging the number of X variables selected across the 3 selection routines within each firm-year. Standard errors clustered at the physician-level are shown in parentheses. F-statistics are shown in brackets.

Column (4) – our preferred estimate – addresses this potential issue by using our fully flexible exploded sets of controls and instruments, and estimating the model via O2SLS. The added richness of this model appears to matter a great deal, as β^m now indicates meals are increasing prescribing among those receiving them by 73 percent, which is roughly equivalent to half a standard deviation. This is still a large number relative to the OLS, suggesting “negative selection” – sales representatives allocate meals to many physicians who otherwise would prescribe relatively low amounts of their drugs, and the interactions embodied in these meals materially affect prescribing patterns.³⁷

Columns (5) and (6) present results from O2SLS, but varying the LASSO penalty to select fewer (5) or more (6) variables. The similarity of (5) to the 2SLS results (3) with our base set of variables (despite including many more variables at 685 controls and 24

³⁷Intuitively, if there are decreasing returns to persuasion across the claims distribution – marginal claims are harder to “buy” as volume increases, a result we obtain below that is consistent with physicians being constrained in the number of suitable patients they see – then it is efficient for firms to have a strategy that targets many, smaller payments to (relatively) more responsive, but lower-volume, physicians.

instruments) suggests there is nothing about including more flexible functional forms or the O2SLS procedure per se that drives our preferred result. Instead, the key factor seems to be allowing for *enough* flexibility. The similarity of (6) to our preferred results suggests that our estimate is not overly sensitive to allowing an even more flexible model.

Our primary finding here – that the interactions surrounding meal payments from industry to physicians have a meaningful effect on physician prescribing (nearly four times that of the OLS correlation) – is large in relative magnitude, but not necessarily surprising in the context of popular press and industry insider writings on pharmaceutical sales.³⁸ It is different in nature and larger than estimates using physician fixed effects, which intuitively estimate the effect of an additional meal for an individual physician who is likely involved in an ongoing relationship; in contrast, our instrumental variables strategy seeks to estimate the causal effect of the entire meal relationship vs. the counterfactual with no relationship. However, there are a number of modeling choices underlying our estimates, and cross-sectional causal identification is inherently difficult. And the large estimated average treatment effect on the treated naturally begs the question of why meals are not even more lavish and widespread. We address each of these issues in turn in the next three subsections.

3.2.1 Robustness Checks

The pooled specification reported above is flexible at the drug-year on all variable selection and parameter estimation, except for the meals parameter. Appendix Table A8 shows results that allow meal effects to differ across drug-years. In each drug-year, we cannot reject (at the 95 percent level) the hypothesis that the drug-year meal effects are identical to the pooled effect. The deviations of the individual point estimates from the pooled also make sense: The effect for Pfizer in 2011 is larger, as might be the case if Pfizer maintains meals where they have the largest impact, even as it allocates less resources to them with the impending patent expiry. The effect for AstraZeneca 2012 is smaller, as might be the case if additional meals provided in 2012 are allocated to more marginal physicians, or if all meal relationships have less of an impact in the presence of generic atorvastatin. Pfizer 2012 has a weak instruments problem where our identification strategy has difficulty predicting the remaining few cardiologists receiving Pfizer payments in 2012. However, re-running the pooled analysis with Pfizer 2012 excluded provides similar results to our preferred estimates, so Pfizer 2012 does not affect our pooled inference on the meal effect substantially.³⁹

³⁸E.g., this is consistent with the observation in industry publications that physicians may be high-value either because they are already high prescribers, or because they are initially low prescribers but can be influenced by targeted marketing (Fugh-Berman and Ahari 2007).

³⁹We also explore the effect of meals on the extensive margin of utilization – whether they increase the likelihood of using a drug at all – and our preferred O2SLS specification cannot statistically reject a null

The specifications so far have also involved several decisions regarding: which cardiologists to include in the estimation sample, how best to address direct effects of conflict-of-interest policies, how best to control for geography, and how best to control for past prescribing. Appendix C.4 tests sensitivity of our results to these decisions. Our results remain similar if we: add AMSA scores as controls for faculty or hospitals; drop faculty, AMC hospitals, or AMC hospital affiliates altogether; add Census region fixed effects; or use more or fewer past prescribing controls.

3.2.2 Placebo Check

During 2011-2, six US states had bans on meals or gifts to physicians, and/or mandatory interaction disclosure requirements before the Physician Payment Sunshine Act went into effect: Maine, Massachusetts, Minnesota, Vermont, West Virginia, and the District of Columbia.⁴⁰ Table 5 shows the reduced form and orthogonal 2SLS results for three sets of states: no disclosures or bans (columns (1)-(2)); disclosure and ban (columns (3)-(4)); and disclosure only (columns (5)-(6)). In restrictive states, we see that meal-based interactions are substantially muted relative to the full sample (though not entirely shut down); for example, in Table 5 below, 39 percent of sample cardiologist-firm-years involved a meal from in states without such policies, vs. only 21 percent in states with disclosure rules and 3 percent in states with bans. These policies thus shut down much of the first stage relationship between market AMSA scores and meals: in Table 5, we also see that the F -statistic on the first stage relationship between AMSA scores and meals is at most 3 in restrictive states. Given the substantially limited ability of firms to detail physicians in the restrictive states, we use the restrictive state sample to explore the reduced form relationship between AMSA scores and prescribing behavior. That is, to the extent that we see a correlation between AMSA scores and prescribing that is not mediated by meals, this would invalidate our exclusion restriction.

While the restrictive state subsamples are much smaller than the full sample and thus the subsample results are noisy, we see no reduced form or IV effect of meals on prescribing in restrictive states. These results are consistent with our exclusion restriction assumption: that, conditional on controls, local AMSA scores are not correlated with preferences, absent the mediating effect of firm payments.

hypothesis of no effect, nor the OLS estimates which range from about a 2 to 10 p.p. effect (see Table A10).

⁴⁰See King and Bearman (2017) and Grolach and Pham-Kanter (2013) for details.

Table 5: Main Results across State Policies, $\log(Claims)$

	No Rules		Restrict		Restrict / Report	
	RF	IV	RF	IV	RF	IV
	(1)	(2)	(3)	(4)	(5)	(6)
Orthog.-IV, or β^m	0.777*** (0.177)	0.827*** (0.206) [56.71]	-1.213 (1.167)	-2.265 (3.352) [0.71]	-0.935 (0.722)	-1.039 (1.097) [2.75]
N	50024	50024	574	574	2395	2395
Mean(Claims)		80.70		44.90		63.60
Mean(Meal)		0.391		0.026		0.206

All IV models estimated via orthogonalized 2SLS. Reduced Form (RF) models estimated using the orthogonal instrument as the independent variable, controlling for firm-year-fixed effects and firm-year-level LASSO-selected variables from the exploded set. Instruments and residuals are calculated on the full sample, and then the regressions are separately estimated on the state subsets. “Restrict” states had some sort of restriction imposed on industry interactions (i.e., could not take place at physician’s office); “Report” states had mandates that all industry interactions must be reported to authorities for public dissemination. Standard errors clustered at the physician-level are shown in parentheses. F-statistics are shown in brackets.

3.2.3 Treatment Effect Heterogeneity by Claims and Meal \$

The results above suggested latent heterogeneity in treatment effects among cardiologists. To provide further evidence on this phenomenon, the top panel of Table 6 explores the shape of the reduced form effect of the combined orthogonalized AMSA instrument using reduced form quantile regressions. Assuming that the first-stage relationship is constant across the distribution of realized claims, any pattern in reduced form effects can inform how treatment effects vary across this distribution, noting that the absolute magnitudes of the coefficients will be slightly inflated since our instrument only increases the *probability* of meal receipt. We observe that the reduced form effect of stricter AMSA scores is smallest for the 80th percentile of the prescribing distribution. This result reinforces that the large average treatment effect of meals on the treated is driven to a greater extent by particularly large effects among otherwise low prescribers – e.g., those that would have otherwise prescribed the promoted drug to only a handful of patients.

Finally, because we can observe the aggregate dollar value associated with these meals, we can examine the extent to which effects are driven by meal receipt per se vs. the dollar value of meals. To this end, the bottom panel of Table 6 presents coefficients when restricting the sample to different maximum payment amounts at the 25th, 50th, 75th, and 90th percentiles of the *non-zero* meal value distribution. That is, column (1) shows the estimated effect of meals under \$21, and column (3) shows the estimated effect of meals under \$104. The results in the Table indicate that the effect of the interactions are not increasing in their dollar value.

Together, these results suggest that the extensive margin effect of receiving any meal leads to a large absolute and relative increase in claims for the relevant firm’s drugs. However, as larger meal values are included in the sample, the apparent returns to the marginal dollar

Table 6: Main Results Heterogeneity, $\log(Claims)$

	(1)	(2)	(3)	(4)
Panel A: Across Realized Distribution				
Orthog.-IV	0.901*** (0.211)	0.943*** (0.185)	0.840*** (0.161)	0.563*** (0.188)
Quantile Est.	.20	.40	.60	.80
N	52419	52419	52419	52419
Panel B: Across Meal Dollar Value				
β^m	1.081*** (0.325) [31.56]	0.822*** (0.230) [46.92]	0.772*** (0.213) [49.88]	0.739*** (0.196) [60.07]
Max \$ Percentile.	25	50	75	95
Max \$	21	45	104	247
N	37532	42399	47480	51420
Mean(Claims)	70.50	73.30	76	78.90
Mean(Meal)	0.138	0.237	0.319	0.371

All models are IV models using only LASSO-selected variables from the exploded set, selected separately at the firm-year level, estimated either as a quantile IV model including the union of controls selected for the dependent and endogenous independent variables (A), or via orthogonalized 2SLS (B), all including firm-year-fixed effects. Dollar value groupings in (C) are based on including all meals less than the corresponding percentile of the dollar-value distribution (Max \$ Percentile.). Standard errors clustered at the physician-level are shown in parentheses. F-statistics are shown in brackets.

are not significant. It appears that the effect is driven by the receipt of any meal, regardless of its value. It is not surprising then that the vast majority of meal values we observe are less than \$100. For this reason, in the structural analyses below we will simply focus on the dummy variable indicating any meal receipt.

When combined with the prior comparison of OLS and O2SLS results, this apparently large role of the extensive margin effect has important implications for the policy discussions surrounding physician-firm interactions. Our results would indicate limited practical effects of policies focused on limiting high-value meals (e.g., over \$50) or high-upper-tail prescribing behavior. Firms seem to have great influence over cardiologists who otherwise would have been low- or moderate-volume prescribers, and this influence is largely driven by interactions involving a low-valued meal.

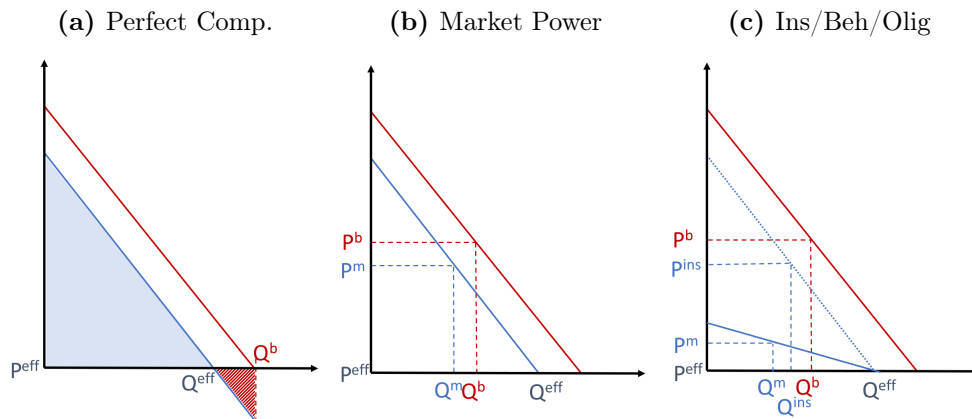
4 Welfare Analysis: Supply and Demand Model

In this Section, we present a model of supply and demand that allows us to analyze the net welfare effects of industry interactions with physicians. The results in the previous section showed that such interactions increase prescribing of related drugs. However, they have little to say about welfare on their own: in this context, welfare analysis of physician-firm interactions must account for market power, strategic interactions, insured demand, and the possibility that physician/patient decision making could be imperfect due to physician agency

or physician/patient behavioral biases. For this, we develop an explicit demand model of how consumers trade off the influences of meals and prices and substitute across competing drugs. We also develop a supply model of how prices are determined in equilibrium for given demand and market structure.

To build intuition regarding this point, consider the welfare effects of interactions that shift the demand curve outward. Panel (a) of Figure 2 presents a hypothetical demand curve in blue and a “biased” demand curve shifted outward in red. Assuming without loss of generality that the drug’s marginal cost is zero, the welfare loss under perfect competition is shown in the shaded triangle below the line segment $\overline{Q^{eff}Q^b}$ – marginal patients prescribed the drug in the presence of physician-firm interactions receive negative health benefits.

Figure 2: Welfare Analysis with Other Frictions



In a setting with perfect competition, this conceptual framework suggests that our analysis in Section 3.2 is all that is needed. However, in many empirically-relevant settings with firm payments to experts, firms also have market power, and utilization is distorted away from the social optimum due to high prices (Inderst and Ottaviani 2012). In prescription drug markets, branded products have patent protection, and they often compete with differentiated branded and generic substitutes whose manufacturers make their own strategic pricing and promotion decisions. A simple version of this model is presented in panel (b) of Figure 2: a branded pharmaceutical manufacturer faces the residual demand curve in blue, which is again shifted outward in the presence of physician-firm payments. Market power causes “unbiased” quantities Q^m to be too low; thus, payments may increase prescribing toward the optimum $Q^m < Q^b < Q^{eff}$ (pictured) or cause prescribers to overshoot the optimum $Q^m < Q^{eff} < Q^b$. In the former case, the overall welfare impact of payments is positive, though consumer surplus declines; in the latter case, both total and consumer surplus

decline. In our structural analyses, we model this supplier market power and incorporate it into our counterfactual analyses.

Finally, we must also account for reasons that the “effective” demand curve for a given drug may not represent the appropriate one for welfare analysis. A leading example is insurance, pictured in panel (c) of Figure 2. The “true” demand curve is the solid blue line; the insured residual demand curve is the dotted blue line (which is significantly less elastic with respect to the producer’s price, as insurance enrollees bear only a fraction of that price out of pocket); and the “biased” demand curve is again in red. In this hypothetical, payments from firms reinforce the effects of insurance, each increasing consumption above the uninsured equilibrium: $Q^m < Q^{ins} < Q^b$. The welfare implications are again ambiguous, and the consumer surplus effects of firm payments will depend on pass-through of producer prices to enrollee premiums. In our structural analyses, we account for the details of patient insurance and model prices as determined via bilateral bargaining between insurance plans and pharmaceutical suppliers.

The general point of panel (c) extends beyond insurance. In oligopoly, the residual demand curve can be distorted due to competitor pricing or payment behavior. This is the phenomenon highlighted in [Inderst and Ottaviani \(2012\)](#), where payments may even increase consumer surplus by improving allocative efficiency. Further, a large literature in health care markets finds that utilization of health care products and services can be biased due to information frictions and imperfect agency. As documented in [Baicker et al. \(2015\)](#), such “behavioral” biases could be positive or negative.⁴¹ While welfare analysis in the presence of behavioral frictions is notoriously problematic ([Bernheim and Rangel 2009](#)), we take advantage of the additional information available regarding pharmaceutical product effectiveness, and complement our revealed preference estimates with welfare estimates based on the clinical literature regarding the health benefits of statins.

4.1 Demand and Pricing Models

Let the utility of molecule $j \in \mathcal{J} = \{1, \dots, J\}$, branded status b , for use case i (a doctor/patient/visit combination) in each market – defined by doctor d in year t – be given by: $u_{ijbdt} = \delta_{jbd} + \varepsilon_{ijbdt}$. The use-specific i.i.d. unobservable $\varepsilon_{ijbdt} = \epsilon_{igdt} + (1 - \lambda^g)\epsilon_{ijbdt}$ is the random coefficients representation of the nested logit model ([Cardell 1997](#)), where ϵ_{igdt} is a random component common to group g , and ϵ_{ijbdt} is the standard type I extreme value error term (with scale normalized to one). As the nesting parameter $\lambda^g \in [0, 1]$ approaches 1, there is less substitution outside the nest. Our preferred specification has separate nests for ator-

⁴¹See Figure A7 for one hypothetical extension of panel (c) with a downward behavioral bias.

vastatin (Lipitor and generic), for all other statins, and for the outside good. We measure the market size of potential statin patients as the number of all cardiovascular prescriptions, including other lipid-modifying drugs, for each physician-year.

The mean utility across use cases is specified as

$$\delta_{jbd t} = \theta^m 1_{\{m_{jbd t} > 0\}} - \theta^p p_{jbd t}^{oop} + \theta_j + \theta_b + \theta_t + x'_{jbd t} \theta_j^x + \xi_{jbd t}, \quad (2)$$

where $\theta^m 1_{\{m_{jbd t} > 0\}}$ is an indicator for whether provider d received a meal from the manufacturer and its utility weight; $\theta^p p_{jbd t}^{oop}$ is the average out-of-pocket price paid by patients and its weight; θ_j is a molecule-specific coefficient; θ_b is a branded dummy and its weight; θ_t is a year-specific coefficient; $x'_{jbd t} \theta_j^x$ is the molecule-brand-specific linear function of LASSO-selected x variables to capture regional and provider variation in prescribing patterns; and $\xi_{jbd t}$ is a product-physician-year unobservable preference heterogeneity term.⁴²

Given a set of products available to a provider \mathcal{J}_{dt} and flow of choice opportunities Q_{dt} , we assume the provider/patient chooses the product that maximizes utility for each use opportunity, so that quantities demanded are given by:

$$q_{jbd t} = Q_{dt} Pr[u_{ijbd t} > u_{ikb' dt}, \forall kb' \in \mathcal{J}_{dt}] = Q_{dt} \frac{e^{\frac{\delta_{jbd t}}{1-\lambda^g}}}{\sum_{kb' \in \mathcal{J}_{dt}} e^{\frac{\delta_{kb' dt}}{1-\lambda^g}}} \frac{\left(\sum_{kb' \in \mathcal{J}_{dt}} e^{\frac{\delta_{kb' dt}}{1-\lambda^g}}\right)^{1-\lambda^g}}{1 + \left(\sum_{kb' \in \mathcal{J}_{dt}} e^{\frac{\delta_{kb' dt}}{1-\lambda^g}}\right)^{1-\lambda^g}}, \quad (3)$$

and consumer surplus across all products is given by:

$$CS_{dt}(\mathcal{J}_t) = Q_{dt} \frac{1}{\theta^p} \ln \left(1 + \left(\sum_{jb \in \mathcal{J}_{dt}} e^{\frac{\delta_{jbd t}}{1-\lambda^g}} \right)^{1-\lambda^g} \right) - \underbrace{\sum_{jb \in \mathcal{J}_{dt}} q_{jbd t} \left(\frac{\theta^m}{\theta^p} 1_{\{m_{jbd t} > 0\}} \right)}_{\text{adjustment for meal "bias"}}, \quad (4)$$

which is the standard formula derived by [McFadden \(1978\)](#), minus an adjustment for the fact that potential bias due to meals affects demand, but not patients' utility. An equivalent interpretation would be that physicians maximize a sum of physician (chooser) and patient (consumer) utility, and all terms but $\theta^m 1_{\{m_{jbd t} > 0\}}$ represent patient utility.

We next characterize how prices are set in equilibrium. Let the supplier's profit be: $\pi(p_{jbt}^{pos}) = \sum_d q_{jbd t} (p_{jbt}^{pos} - mc_{jbt})$, where mc_{jbt} captures the cost of manufacturing and dis-

⁴²Several recent papers (e.g. [Dubois et al. \(2018\)](#); [Shapiro \(2018\)](#); [Sinkinson and Starc \(2018\)](#)) focused on television advertising have explicitly focused on the possibility that such ads can have spillover effects across brands in a category. We have used the fact that we allow for different controls across molecule-brands to attempt to estimate such cross-effects, but they have always been small and noisy (recall our identification strategy based on regional spillovers does not provide strong explicit firm-specific variation).

tributing the marginal unit of molecule j . p_{jbt}^{pos} is the point-of-sale price insurers pay for the drug, which we assume is constant across providers. We link the negotiated point-of-sale price and out-of-pocket price paid by enrollees via $p_{jbd}^{oop} = cs_{jbd} p_{jbt}^{pos}$, where cs_{jbd} is an exogenous cost-sharing parameter that can vary across markets and years, depending on product mix and insurer mix (discussed in detail in Appendix A.2).

We assume that prices of substitute drugs in the market are determined in a simultaneous Nash Equilibrium of Nash Bargaining between suppliers (manufacturers/wholesalers/pharmacies) and buyers (PBMs/insurers).⁴³ This captures the primary forces relevant to our research question, abstracting from some of the details of the upstream interactions between suppliers, and from insurer competition and insurance plan structure.⁴⁴ In the model, each price maximizes the Nash Product of the gains from trade for each supplier and buyer pair, taking other prices as given.⁴⁵ The first-order condition on each price is:

$$\begin{aligned} p_{jbt}^{pos} &= \arg \max \left(\pi(p_{jbt}^{pos}, p_{jbt}^{oop}, m_{jbd}) \right)^{b_{jbt}} \left(\widetilde{CS}_t(\mathcal{J}_t) - \widetilde{CS}_t(\mathcal{J}_t \setminus jb) \right)^{1-b_{jbt}} \\ &= mc_{jbt} + b_{jbt} \left[\left(1 + \frac{\partial q_{jbt}}{\partial p_{jbt}^{oop}} \frac{p_{jbt}^{oop} - mc_{jbt}}{q_{jbt}} \right) \frac{\widetilde{CS}_t(\mathcal{J}_t) - \widetilde{CS}_t(\mathcal{J}_t \setminus jb)}{q_{jbt}} + p_{jbt}^{pos} - mc_{jbt} \right] \end{aligned} \quad (5)$$

where $q_{jbt} := \sum_d q_{jbd}$ denotes the sum over physicians. The term b_{jbt} is a bargaining ability parameter, weighting the extent to which the optimal price depends on supplier profits vs. the expected additional buyer surplus in the case that a contract is agreed to for product jb : $\widetilde{CS}_t(\mathcal{J}_t) - \widetilde{CS}_t(\mathcal{J}_t \setminus jb)$. Note that quantities and thus elasticities are driven by physician/enrollee decision-making based on out-of-pocket price under insurance coverage p^{oop} , but the insurer and supplier negotiate over point of sale price p^{pos} . In contrast to some recent papers on insurer-hospital bargaining (e.g., [Gowrisankaran et al. 2015](#); [Ho and Lee 2017](#)), we model pricing of a single product class (statins) rather than a bundle of products. Also, given that we do not observe how plan enrollment might respond to disagreement, we proxy for plan surplus by using a parameter $\alpha \in [0, 1]$ to capture the relative weight insurers place on consumer surplus, and subtracting plan costs: $\widetilde{CS}_t(\mathcal{J}_t) := \alpha CS_t(\mathcal{J}_t) - \sum_{jb} q_{jbt} (p_{jbt}^{pos} - p_{jbt}^{oop})$.

⁴³As discussed by [Starc and Swanson \(2018a\)](#), both pharmacies and pharmaceutical manufacturers have market power, but relative market power of different suppliers varies by drug. Pharmacies make larger margins on generic drugs than on branded drugs, while branded manufacturers command higher markups (even net of rebates) than generic manufacturers.

⁴⁴These details are captured in a reduced form sense by the bargaining and cost-sharing parameters. Our counterfactual analysis will hold these fixed. This assumes that banning meals to physicians does not change the fundamental supply chain of the pharmaceutical industry or the general treatment of branded and generic therapies in insurance plan formularies.

⁴⁵We can also model the supply side of meals; see Appendix C.2.1. It is not necessary for our counterfactuals to estimate the parameters underlying meal provision, but the likelihood of a physician receiving meals depends on the marginal benefit and marginal cost of meals for that physician. This cost-benefit analysis is the motivation for our AMSA-based identification strategy.

4.2 Identification and Estimation

We follow [Berry \(1994\)](#), setting choice probabilities implied by the demand model equal to observed market shares, and inverting the system to yield a linear specification:

$$\ln(s_{jbd t}/s_{0dt}) = \lambda^{g_j} \ln(s_{jb|g dt}) + \theta^m 1_{\{m_{jbd t} > 0\}} - \theta^p p_{jbd t} + \theta_j + \theta_b + \theta_t + x'_{jbd t} \theta_{jb}^x + \xi_{jbd t} \quad (6)$$

where $s_{jbd t}$ is jb 's overall market share, s_{0dt} is the market share of the outside good (non-statin treatments), and $s_{jb|g dt}$ is jb 's market share within nest g .

Theory suggests that $\ln(s_{jb|g dt})$, $p_{jbd t}$, and $1_{\{m_{jbd t} > 0\}}$ are all correlated with the unobservable term $\xi_{jbd t}$. We take an instrumental variables approach to solving this identification problem, again using regional spillovers from AMC CoI policies to identify the meal effect, and leveraging the variation induced by the introduction of generic atorvastatin and the regional variation in prices introduced by the timing of formulary changes across insurers to disentangle price elasticities.

To use the variation induced by the introduction of generic atorvastatin to the choice set, we follow the suggestion of [Gandhi and Houde \(2016\)](#), constructing an instrument that interacts the post-entry period with indicators for Lipitor, Crestor, and the other generics, $z^a = 1_{\{t=2012\}} \cdot [1_{\{jb=Lipitor\}}, 1_{\{jb=Crestor\}}, 1_{\{b=0\}}]$. With our molecule and time fixed effects, this instrument captures difference-in-differences variation, allowing the effect of the treatment on substitution patterns to differ by whether a drug is Lipitor (branded atorvastatin – the same molecule), Crestor, or a generic moderate statin, such as simvastatin. These instruments help identify substitution patterns broadly, and in theory are valid for both $\ln(s_{|g})$ and p .

We further leverage the heterogeneity in insurer responses to atorvastatin entry across geography, as described in [Appendix A.2](#). When Lipitor's patent expired, some insurers instantly added generic atorvastatin to their preferred drug lists and/or removed Lipitor from their formularies, while others took more than a year. The variation in penetration of these insurers across geography generated large variation in the relative prices consumers faced for Lipitor and generic atorvastatin. To utilize this variation, we create instruments for each plan-drug-year-region as the average out-of-pocket price for that drug-year-insurer across *other* regions. We then average across plans to create an instrument for physician d 's region. We also create an analogous instrument based on an average dummy for formulary coverage, alone and interacted with the Lipitor dummy. The instrument set is then: $z^p = [p_{jbd t}^{oop, IV}, \bar{1}_{\{jb \in form_{dt}^{IV}\}}, \bar{1}_{\{jb \in form_{dt}^{IV}\}} \cdot 1_{\{jb=Lipitor\}}]$. These are similar to the bargaining ability instruments in [Grennan \(2013, 2014\)](#) and [Dickstein \(2016\)](#), with the added benefit of a clear mechanism driving their variation. As such, they are also valid for both $\ln(s_{|g})$ and p .⁴⁶

⁴⁶We are sensitive to the fact that firm activities besides the prices and meals we measure may change

In addition to the instruments linked to generic atorvastatin entry, we also follow much of the literature (e.g., [Berry and Waldfogel 1999](#)) in using a polynomial in the size of the set of generic statins prescribed $z^J = [1/|\mathcal{J}_{dt}^{gs}|, |\mathcal{J}_{dt}^{gs}|, |\mathcal{J}_{dt}^{gs}|^2]$ as an instrument. This leverages the fact that more variety will mechanically affect within-group shares.⁴⁷

Finally, we use the same identification strategy for meals $1_{\{m_{jbt}>0\}}$ detailed previously. The strategy from [Belloni et al. \(2017\)](#) is built to work in any generalized method of moments (GMM) framework, so we implement it here as well. We allow the LASSO-selected sets of instruments z and controls x to vary flexibly across molecule-brand (now including generics as well). We then construct the residuals and orthogonal instruments as before, for each endogenous variable in turn. In our preferred implementation, we jointly estimate the above (linearized) demand model with the supply model using GMM. This enables us to simultaneously recover the demand, bargaining, and marginal cost parameters.⁴⁸

4.3 Parameter Estimation Results

The top panel of [Table 7](#) shows the demand parameter estimates. The nesting parameters on atorvastatin and other statins both indicate more substitution within statins than to the outside good, and further, generic atorvastatin is a stronger substitute for Lipitor (the identical molecule) than for other statins. The price coefficient is small, as we would expect given the muted incentives provided by insurance, and the related own-price elasticity $\eta_p = \frac{\partial s}{\partial p} \frac{p}{s}$ has an average of -0.06 , which is also consistent with insurer negotiating power preventing manufacturers from fully exercising their market power. Product fixed effect parameters follow a pattern that matches clinical evidence on the quality of the different molecules, and the brand dummy has a small positive (but noisy) coefficient (see [Appendix Table A12](#) for these coefficients, elasticities, and other results, for this and alternative demand specifications).

The relative size of the meal and price coefficients suggest that a meal has an equivalent impact to a \$454 decrease ($= \theta^m / \theta^p$) in out-of-pocket price. While this seems like a large effect, it is in part driven by the observed lack of price sensitivity. Perhaps more enlightening is the implied semi-elasticity $\eta_m = \frac{\partial s}{\partial m} \frac{1}{s}$, which measures the percent change in market share of the focal drug associated with a meal. The average of 125 percent suggests this payment effect is indeed substantial.⁴⁹ The meal coefficient of 0.88 is about three quarters of a standard around the period of patent expiration. See [Appendix C.6](#) for a discussion and results along these lines. In particular, our results remain unchanged if we allow selection and estimation of $x\theta^x$ for Lipitor to vary across years.

⁴⁷This is also closely related to the intuition behind [Sinkinson and Starc \(2018\)](#), who use managed care penetration to proxy for restricted choice sets in the statin market in an earlier time period.

⁴⁸It also imposes the constraints of the supply model that $mc_{jbt} \in [0, p_{jbt}]$, and $\frac{\partial s_{jbt}}{\partial p_{jbt}} \frac{p_{jbt} - mc_{jbt}}{s_{jbt}} \in [-1, 0]$ (though these constraints do not bind in our preferred model when demand is estimated separately).

⁴⁹This is larger than our 2SLS estimates from [Section 3.2](#) since it is averaged here across all physicians

deviation of the variation across product-physician observable ($sd(\theta_j + x'_{jbd} \theta_{jb}^x) = 1.13$) and unobservable ($sd(\theta_j + x'_{jbd} \theta_{jb}^x + \xi_{jd}) = 1.22$) dimensions.

Table 7: Demand and Supply Parameter Estimates

Demand:	θ^m	θ^p	$\lambda^{ator=1}$	$\lambda^{ator=0}$		
	0.88 (0.09)	-0.0019 (0.0006)	0.50 (0.02)	0.45 (0.03)		
Supply:	Atorvastatin	Lipitor	Lovastatin	Pravastatin	Crestor	Simvastatin
<i>mc</i>	0	0	0	0	0	0
<i>B</i> ₂₀₁₁	-	0.26 (0.01)	0.04 (0.00)	0.05 (0.00)	0.44 (0.01)	0.03 (0.00)
<i>B</i> ₂₀₁₂	0.07 (0.00)	0.50 (0.01)	0.03 (0.00)	0.03 (0.00)	0.51 (0.01)	0.02 (0.00)

N = 124,876 doctor-drug-brand-year observations with standard errors clustered at the doctor level (*N*_{*d*} = 15,063) via delete-120 jackknife.

The bottom panel of Table 7 summarizes supply side parameter estimates.⁵⁰ The most striking feature is the high bargaining parameter estimates for the branded drugs relative to generics. Because the generic sales are aggregated over firms, the bargaining parameters also capture within-molecule competitiveness. This can also be seen in the slightly larger bargaining parameter for generic atorvastatin, where only two manufacturers compete during the first six months of 2012, before 11 enter. The larger bargaining parameters for Lipitor and Crestor in 2012 reflect the fact that POS prices remain high in many regions for much of 2012 as insurers are slow to adjust formularies, despite the improved outside option with generic atorvastatin entry.⁵¹ Finally, we estimate that the weight insurers place on enrollee surplus in negotiations is equivalent to the weight they place on net costs: $\alpha = 1$. This may reflect that enrollees are sensitive at the plan choice stage to formulary inclusion of important drugs, and/or downward bias in our revealed preference measure of enrollees' valuation of statins relative to their true health value (a possibility we address in the next Section).

These demand and supply estimates cannot by themselves speak to the effect of payments on pharmaceutical markets. By construction, they measure the effect "holding all else equal", but both prices and quantities may adjust to any policy change. And with the oligopoly structure of the market, these strategic reactions will depend on one another in equilibrium.

and drugs (including generics and physicians with very low prescribing that receive no meals in practice).

⁵⁰For simplicity, we set marginal costs to zero. The small number of negotiated drug-year prices p^{pos} makes it difficult to estimate marginal costs as a statistical exercise. As the generic margins are quite small, any reasonable marginal cost assumption would give very similar numbers in our counterfactual analyses.

⁵¹One potential caveat to this approach is that we do not observe confidential rebates between plans and manufacturers. To the extent that realized net-of-rebate prices to plans are much lower than observed point-of-sale prices for branded pharmaceuticals, our estimates of b for Pfizer and AstraZeneca may be biased upward. These unobserved potential rebates are an endemic challenge to research on pharmaceutical pricing. Our counterfactuals should be interpreted as holding fixed these rebate incentives (conditional on changes to demand induced by a meal ban).

4.4 Welfare: Status Quo vs. Meal Ban vs. Efficient Benchmark

To better understand the effects of payments to physicians on market welfare, we consider two counterfactual scenarios banning meals/payments from pharmaceutical firms to physicians. The first scenario bans payments and allows all prices and quantities to adjust to a new equilibrium. The second scenario computes equilibrium quantities and prices with banned payments and OOP prices at marginal cost: an efficient static allocation benchmark.⁵²

In each scenario, we also calculate several functionals of the equilibrium prices and quantities: retail producer surplus PS_{retail} , which is equivalent to out-of-pocket spending; consumer surplus CS_{retail} implied by the utility model of demand; the component of revealed preference “consumer surplus” driven by meals $CS_{meals} = \sum_{jb \in \mathcal{J}_t} q_{jbd} \frac{\theta^m}{\theta^p} 1_{\{m_{jbd} > 0\}}$ (which we assume to be pure bias in our overall consumer and total surplus calculations); and total point-of-sale transfers from insurers to manufacturers/distributors $POS_{transfers}$. We summarize the welfare implications in two ways: First, we calculate “total surplus” $TS = CS_{retail} - CS_{meals} + PS_{retail} - PS_{meals}$. Here, we net out PS_{meals} (the dollar value of meals to physicians) as a lower bound on the firms’ costs of physician interactions, so that TS represents an upper bound on true surplus. Second, we compute an alternative measure of consumer welfare based on estimated health impacts from studies of statin efficacy.

Table 8 displays the results, under the observed data and counterfactual regimes (and separately for each year in order to show how the results depend on market structure). Focusing first on quantities, the primary result is that payments offset the underprovision of statins due to (market power keeping) prices above marginal cost, but in such a way that quantity of statins consumed with payments (column (1)) overshoots the efficient benchmark (column (3)). The model estimates that Lipitor is under-utilized with payments banned in 2011 at 1.18 million prescriptions, vs. 1.26 million at the efficient allocation, whereas the observed payments raise Lipitor to 1.38 million. Thus, payments cause Lipitor quantity to overshoot the efficient benchmark, by slightly more than the shortfall with payments banned. By contrast, the model predicts that payments cause Crestor utilization to overshoot the efficient benchmark by much more than the shortfall in the “no payments” scenario: quantity under the meal ban is 0.47 million prescriptions, slightly less than the efficient 0.53 million, whereas payments cause Crestor to jump to 0.97 million prescriptions.

Comparing 2011 and 2012 shows the importance of modeling strategic interaction and substitution across drugs. In 2012, after the entry of generic atorvastatin, Pfizer almost completely stopped providing meals, and both Lipitor and generic atorvastatin were utilized less

⁵²This is efficient in the sense that it removes any meal or market power pricing distortions. It does not speak to other potential distortions in patient/physician choice or insurer weighting of the implied consumer surplus in price negotiations.

Table 8: Welfare and Counterfactual Estimates

	2011			2012		
	(1) Obs	(2) Ban	(3) Eff	(4) Obs	(5) Ban	(6) Eff
$Q_{statins}$ (millions)	6.05	5.63	5.77	6.43	6.17	6.31
$Q_{atorvastatin}$	1.38	1.18	1.26	2.03	2.04	2.13
$Q_{Crestor}$	0.97	0.47	0.53	0.99	0.46	0.52
$\bar{p}_{statins}$ (\$, OOP)	19	15	0	16	13	0
$\bar{p}_{atorvastatin}$	39	39	0	24	23	0
$\bar{p}_{Crestor}$	42	41	0	38	37	0
PS_{retail} (\$ millions)	113.7	86.0	0	100.0	79.2	0
PS_{meals}	-0.5	0	0	-0.9	0	0
CS_{retail} (\$ millions)	3452.0	3190.6	3279.5	3685.8	3542.2	3608.6
CS_{meals}	-451.6	0	0	-312.2	0	0
TS (\$ millions)	3113.6	3276.5	3279.5	3472.5	3603.4	3608.6
$POS_{transfers}$	254.1	187.1	290.1	195.6	130.6	230.4
CS_{LYG}	17,325.1	16,150.3	16,551.5	18,685.0	17,948.6	18,389.5

Welfare estimates using data (Obs) and counterfactual equilibrium (Ban and Eff) quantities and prices. All parameters different from zero at 1% level (clustered via delete-120 jackknife at the doctor level). Table with standard errors in Appendix.

with meals than without because meals drove substitution to Crestor. Also, since payments were limited to Crestor in 2012, we observe that payments moved total quantity *closer* to the efficient benchmark in 2012 ($|Q_{(3)} - Q_{(1)}| < |Q_{(3)} - Q_{(2)}|$), but payments moved total quantity *further* from the efficient benchmark in 2011 ($|Q_{(3)} - Q_{(1)}| > |Q_{(3)} - Q_{(2)}|$).

These quantity effects highlight several of the issues motivated in Section 4.1 and [Inderst and Ottaviani \(2012\)](#). The extent to which payments affect allocative efficiency depends upon their scale relative to the distortion due to high prices, and upon their affect on strategic interactions between the firms. In the market studied here, payments may move total quantity closer to the efficient allocation, but these aggregate effects also play out very differently across individual products in the market. Moreover, translating these quantity effects into surplus measures requires further analysis, depending on the extent to which meals affect prices and/or better align consumption with the true quality/cost tradeoffs of the various drugs in the market and vs. the outside option.

Equilibrium prices in the meal ban counterfactual indicate that meals have little effect on out-of-pocket prices. The reason is that the combination of low price sensitivity of demand, and prices that are often below firm profit maximizing levels due to insurer-firm bargaining, mean that the price decreases we might expect with a meal ban are small in this market.

Regarding the efficient allocation of consumers to specific products, the direct effect of payments is to move quantity towards the paying firm’s drug in cases where it otherwise would not have been used. This results in a loss of consumer surplus of $CS_{meals} = -\$452$ million in 2011, vs. $-\$312$ million in 2012. This could be offset to the extent that payments

steer patients towards better treatments – in particular, since two firms have patented drugs in 2011, payments could in principle better align their market shares with their qualities – but the comparison of $CS_{retail} + CS_{meals}$ across columns (1) and (2) shows that this is not the case here. Banning payments results in an increase of \$190M (6.3 percent) in consumer surplus in 2011 via allocation. These distortions are stronger in 2011 than in 2012: the distortion for Crestor alone is stronger than for Lipitor alone in 2011, but the total distortion for Crestor+Lipitor in 2011 is stronger than for Crestor alone in 2012.

Even if consumer surplus is harmed, total surplus need not be. To the extent the market expands to allocate more statins to patients who should receive them at marginal cost, this may increase surplus in an efficient manner. Here, we see that consumer surplus losses outweigh producer gains in both 2011 and 2012, resulting in payments being inefficient in terms of total surplus in both 2011 and 2012, in spite of moving closer to the aggregate efficient allocation on the extensive margin in 2012. Banning payments results in an increase of \$163M (5.2 percent) in total surplus in the retail market for statins in 2011.

While the above effects in the retail market all hint towards a value in banning payments, they leave out at least three potentially important features of the market. The first is the valuable information that may be provided via the interactions the meals facilitate. This has been assumed to be zero in the context we study here, due to the late stage of the statin market, but it could be large in other contexts. If we were to instead interpret changes in prescribing due to meals as utility enhancing, this would imply that a ban reduces consumer surplus by \$261M (7.6 percent) in 2011 (the break-even number for consumer surplus is if 42 percent of the meal effect is associated with true utility vs. pure persuasive bias).

The second is the point of sale price p^{pos} that insurers pay, which is split among pharmaceutical manufacturers, distributors, and pharmacies. This number is difficult to compare with the others as it is a cost shared by enrollees and, in the case of Part D, the government, and so it is not easily translatable into a per person effect on premiums, let alone welfare. With that caveat, however, the calculations under $POS_{transfers}$ suggest that these drug cost effects are meaningful. Because payments steer patients toward much more expensive drugs, they increase spending on statins by \$67M (26 percent) in 2011 and \$65M (33 percent) in 2012 relative to our counterfactual where payments are banned.

Finally, there is the fact that our consumer welfare measurements are based on revealed preference estimates of a utility function that represents doctor/patient choice for statin treatment. While this estimated function has a straightforward interpretation in terms of the choice process driving market demand, it could deviate from a measure of true consumer surplus due to physician agency or physician/patient biases. In light of this, we construct an alternative measure of consumer welfare, CS_{LYG} , by combining our equilibrium quantity

predictions with estimates of the health value of statins from the clinical literature valuing “life years gained.”⁵³ Because the health value of statins is so large in the clinical literature, the calculations in the final row of Table 8 would indicate a large welfare loss from a meal ban relative to any spending considerations.⁵⁴

5 Conclusion

In many industries, firms reach consumers through expert intermediaries. Interactions between firms and these experts, which can involve direct payments and other kinds of remuneration, risk creating conflicts of interest that can hinder efficiency. However, these interactions can also facilitate valuable information flows, enhancing welfare, and they often take place in conjunction with other distortions due to agency, market power, and strategic interactions between firms. While recent theoretical work (Inderst and Ottaviani 2012) has shed new light on these tradeoffs, it has remained challenging to identify these relationships empirically. This gap in the literature is particularly important, given recent debates over conflicts of interest and disclosure in the US health care and financial services industries.

We address this gap by proposing an instrumental variables strategy to overcome the challenges of empirically estimating these effects in the health care industry. We show that local academic medical center conflict of interest policies influence the probability of payments from pharmaceutical companies for unaffiliated doctors in the same region. We also exploit variation in market structure over time using the Lipitor patent expiration and ensuing generic entry to disentangle market power effects. Leveraging this approach with detailed data on prescriptions, prices, and payments, we are able to identify the impact of these interactions on prescribing behavior and overall welfare.

Overall, we find the IV estimates for the effect of meals on prescribing to be positive and

⁵³Here, we assume that all prescribing is “appropriate” – i.e., marginal patients are indicated for treatment – up to the efficient quantities with no price or meal distortion. For usage above this, we assign utility value zero. This assumption seems appropriate for drugs like statins, which are generally thought to be underutilized even absent price distortions (see Baicker et al. (2015)). We would urge caution in applying this assumption for drugs that are prone to overutilization, such as opioids (Hadland et al. (2018)).

⁵⁴Our life-years gained calculation is as follows. First, there are approximately 6.93 claims per beneficiary year in the 2013 Medicare Part D Data, the first year that days supply and beneficiary counts are publicly reported; we therefore divide our claim counts by 6.93 in each year. Second, statins are intended to treat chronic conditions and effectiveness will depend on medication adherence; we apply the minimum 37 percent adherence rate from hyperlipidemia trials (adherence rates range from 37 percent to 80 percent) (Deichmann et al. 2006). Third, among our estimated count of adherent beneficiaries choosing moderate statins, we apply the life year gain of 0.69 for Medicare-age enrollees estimated by the Heart Protection Study Collaborative Group (Heart Protection Study Collaborative Group 2009); and for the incremental benefit of the “strong statins” atorvastatin/rosuvastatin, we apply the additional 0.09 life year gain from high-dose atorvastatin vs. low-dose atorvastatin from the TNT study (Wagner et al. 2009). Finally, we apply a conservative value of \$75,000 per life year gained (Cutler 2004).

significant, both statistically and economically. The IV estimates are also larger than OLS estimates, consistent with firms targeting payments to physicians who would otherwise have prescribed the focal drug with low probability. These effects appear to be highly nonlinear within and across physicians. In particular, larger or more frequent payments make little difference compared to the event of having any payment at all.

Our counterfactual welfare analysis of banning payments indicates that such a ban would have a positive effect on consumer and total surplus as measured from our estimated demand and supply models. This is the result of two conflicting forces. High prices due to market power keep statin consumption – overall and of the powerful branded molecules – inefficiently low, and increased consumption due to payments partially offsets this, bringing the market closer to the efficient allocation. However, this comes at the cost of higher prices, which outweighs the extensive margin gains. This result is sensitive to the consumer welfare measure, though – an alternative measure based on estimates of life-year gains from statins suggests that consumer health gains are sufficient to justify the increased costs.

There are limitations in our approach. We focus on a particular market, cardiologists and statin prescriptions, during a two-year time period near the expiration of the Lipitor patent. The dynamics of this market could differ in important ways from other drug and device markets in health care, and other industries where expert intermediaries play an important role, such as financial services. Future research can address these limitations, perhaps by building on our identification strategy for payments, which is quite general, or by providing alternative approaches to identify causal effects and model market responses.

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A Additional Institutional Background

A.1 Medicare Part D

37 million people, or 70 percent of eligible Medicare beneficiaries, enrolled in Part D plans in 2014.⁵⁵ Medicare-eligible individuals can acquire prescription drug coverage through standalone Part D plans or bundled with medical and hospital coverage in the form of “Medicare Advantage” plans. Utilization of drugs in the Part D program is a function of physicians’ prescribing decisions. These in turn may be impacted by: prescribers’ training and knowledge, interactions with pharmaceutical firms, and preferences over cost control; the relevant drugs’ effectiveness, side effects, and out-of-pocket costs; and Part D insurers’ coverage policies.

Part D plans are offered by private insurers, but the federal Centers for Medicare and Medicaid Services mandates coverage generosity of plans in terms of actuarial value, types of drugs covered, and pharmacy network breadth. Enrollees are entitled to basic coverage of prescription drugs by a plan with equal or greater actuarial value to a standard Part D plan.⁵⁶

The majority of Part D enrollees are not enrolled in standard plans, but rather in actuarially equivalent or “enhanced” plans with non-standard deductibles and tiered copays where enrollees’ out-of-pocket costs vary across drugs and pharmacies. Branded drugs with close generic substitutes (e.g., Lipitor and Crestor vs. simvastatin and pravastatin prior to Lipitor’s patent expiration) generally have higher copays than generics, while branded drugs with generic equivalents (e.g., Lipitor after patent expiration) have even higher copays or may not be covered by plans at all. On the other hand, approximately 30 percent of Part D enrollees qualify for low-income subsidies (LIS), which entitles them to substantial reductions in premiums and out-of-pocket costs on covered drugs; maximum copays for LIS enrollees are low or zero.⁵⁷

Part D issuers receive premiums from enrollees and a variety of subsidy payments from

⁵⁵ Hoadley, J., Summer, L., Hargrave, E., Cubanski, J., and Neuman, T. (2014). *Medicare part d in its ninth year: The 2014 marketplace and key trends, 2006-2014. Technical report, Kaiser Family Foundation.*

⁵⁶ In 2011, the standard plan covered: none of the first \$310 in drug costs each year (the deductible); 75 percent of costs for the next \$2,530 of drug spending (up to \$2,840 total; the “initial coverage region”); 50 percent of branded costs for the next \$3,607 of drug spending (up to \$6,447 total; the “donut hole”); and 95 percent of costs above \$6,447 in total drug spending (the “catastrophic region”).

⁵⁷ Partial subsidies are available at 150 percent of the federal poverty level (FPL); full subsidies are available at 100 percent of FPL. LIS enrollees can enroll premium-free in “benchmark plans” or enroll in a non-benchmark plan and pay the difference between the chosen plan’s premium and the benchmark premium out-of-pocket.

CMS: risk-adjusted direct subsidies for each enrollee, additional subsidies to cover LIS premiums and cost-sharing, and reinsurance for particularly high-cost enrollees. They also receive or pay “risk corridor” transfers such that the issuers’ profits/losses are within certain bounds.⁵⁸ Although issuers’ strategies and profits are heavily regulated by CMS, they can constrain costs through formulary design (drugs’ coverage and placement on tiers, which determine patients’ access to those drugs and out-of-pocket costs), negotiations with drug manufacturers, and negotiations with pharmacies.

A.2 Regional Pricing/Formulary Variation in 2012

In our structural analyses in Section 4, we identify the price sensitivity of demand using panel variation in out-of-pocket prices faced by Medicare enrollees. This variation is driven by Lipitor’s patent expiration and by regional variation in insurers’ responses to Lipitor’s patent expiration.

Out-of-pocket prices are generally determined using insurance plan-specific formulas as a function of drug coverage, placement on tiers, point-of-sale price, and benefit phase. If a drug is covered, the out-of-pocket price will be *either* the tier-phase-specific copay *or* the product of the tier-phase-specific coinsurance and the point-of-sale price of the drug. In our analyses, we focus on prices per one-month supply of the relevant drug in the initial coverage phase of the Medicare Part D plan – most claims are filled in the initial coverage phase as opposed to the deductible, donut, or catastrophic phase.

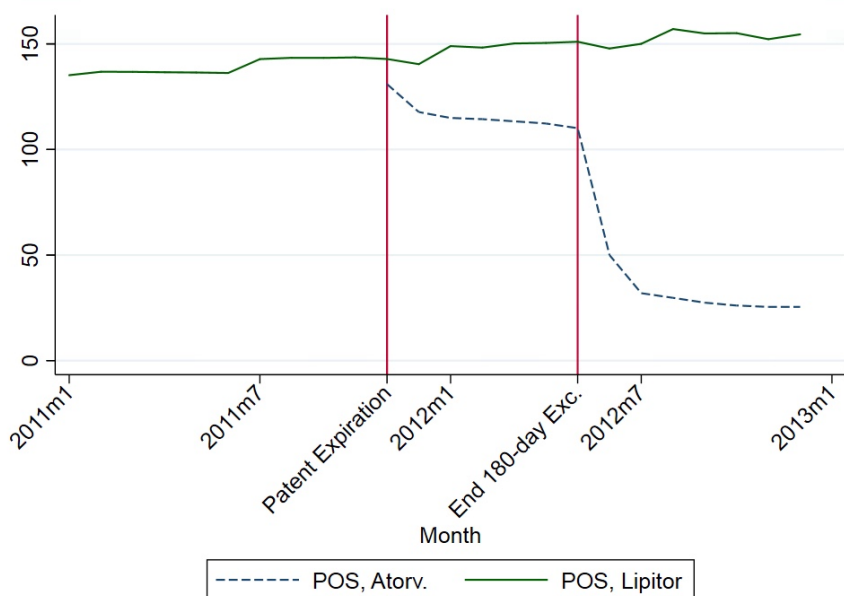
Figure A1 shows the trend in point-of-sale prices for Lipitor and generic atorvastatin over 2011-2012. After Lipitor’s patent expired in November 2011, generic atorvastatin was introduced by two generic manufacturers – the “authorized” generic firm Watson Pharmaceuticals and the paragraph IV challenger Ranbaxy Laboratories – that were afforded 180 days of exclusivity from other generic competition. Prices for generics remained high, near \$115, for the 180-day generic exclusivity period, then dropped dramatically and leveled out near \$25. Branded Lipitor’s price remained high, increasing slightly from \$135 in early 2011 to \$155 during 2012.⁵⁹

Figure A2 shows the percent of Medicare Part D plans covering atorvastatin and Lipitor during 2011 and 2012. When Lipitor’s patent expired in November 2011, there was an immediate jump from 0 percent to about 80 percent of plans covering atorvastatin. Conversely,

⁵⁸Insurers bear all upside/downside risk within a 5 percent band of zero profit; outside this risk corridor, the plan absorbs 20-25 percent of profits and losses.

⁵⁹The observed point-of-sale prices are the basis to which enrollees’ coinsurances are applied, but they are not net of rebates, and thus do not accurately represent the prices that pharmaceutical manufacturers receive per claim. Rebates are known to be an important strategic variable for branded manufacturers (though not for generic manufacturers). We return to this issue in our discussion of the structural results.

Figure A1: Point-of-Sale Price of Atorvastatin/Lipitor, 2011-2012



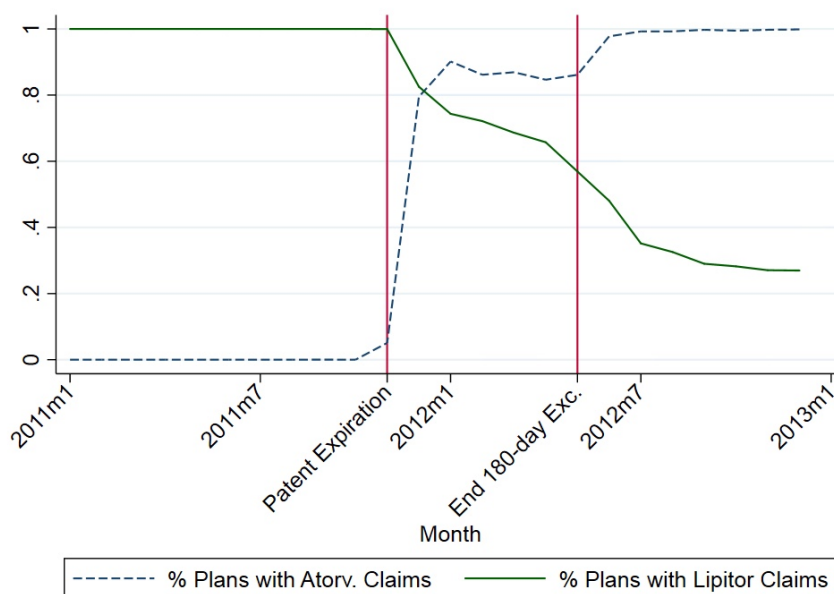
Note: Reproduced from [Starc and Swanson \(2018b\)](#). Average point-of-sale price of Lipitor/atorvastatin observed in monthly prescription drug event data. Claims made by non-LIS enrollees for 30-day supply in the initial coverage phase of the drug benefit only.

the trend downward in plans' coverage of branded Lipitor is much flatter, as many plans did not remove Lipitor from their formularies until well after patent expiration. As of December 2012, 27 percent of plans still covered Lipitor.

Finally, Figure A3 shows the trend in out-of-pocket prices for Lipitor and atorvastatin in 2011-2012, conditional on Lipitor being on-formulary. Generic copays for atorvastatin dropped from about \$25 to about \$9 after 180-day exclusivity. Lipitor copays were fairly flat, declining from about \$38 to \$32 over 2011-2012, implying that the primary incentives plans used to induce enrollees to switch from Lipitor to atorvastatin were to drop Lipitor from their formularies and/or reduce copays for atorvastatin.

For our structural model estimation, we use point-of-sale and out-of-pocket prices from the CMS Part D public use files for Q2 2011 and Q3 2012. Prices are collected at the plan-drug-year level. Given that our prescription drug claims data cannot be linked to plans, we aggregate up to the Part D region-drug-year level (Part D regions are 39 supersets of states) using plan enrollment data to construct weighted averages. Cross-sectional variation in prices is generated by plan-pharmacy negotiations over point-of-sale prices and by plan-specific decisions regarding drug coverage and tiering. The coefficients of variation for the point-of-sale (out-of-pocket) price across Part D regions in 2011 were 0.03 (0.18) for Crestor, 0.03

Figure A2: Coverage of Atorvastatin/Lipitor, 2011-2012



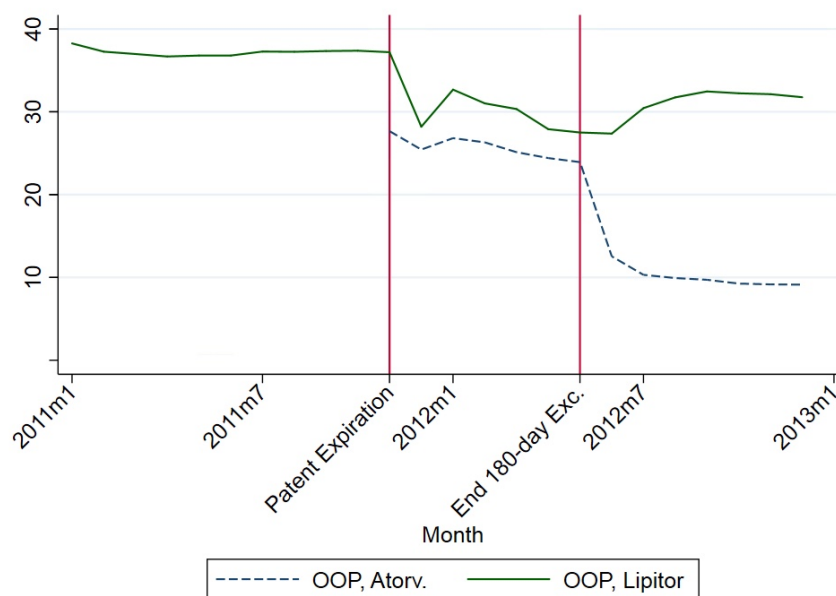
Note: Reproduced from [Starc and Swanson \(2018b\)](#). Average formulary coverage of Lipitor/atorvastatin observed in monthly prescription drug event data.

(0.13) for Lipitor, and 0.33 (0.22) for simvastatin. The coefficients of variation for point-of-sale price for Lipitor and Crestor were similar in 2012; however, the coefficient of variation on out-of-pocket price increased to 0.19 for Lipitor, and there was substantial variation in 2012 in terms of both point-of-sale ($CV = 0.27$) and out-of-pocket price ($CV = 0.28$) for generic atorvastatin. This price variation is presented for our focal drugs in Table A1 below.⁶⁰

Many of the determinants of both point-of-sale and out-of-pocket prices across regions at a point in time are likely driven by insurer-specific factors that are correlated across regions. These might include management, contracts with prescription benefit managers, and costs. Given this, we introduce another source of identifying variation – for each plan-drug-region-year, we calculate the average price for that plan-drug-year in *other* regions, and we aggregate that instrument across plans within each region to generate a region-drug-year-specific instrument. The logic is as follows: if (for instance) United HealthCare were

⁶⁰The primary distinctions between Table A1 and Figures A1 and A3 are (1) that the prices in the Figures are claims-weighted, while the prices in the Table are enrollment-weighted across plans; and (2) that the Figures are from claims data and are thus conditional on drugs being covered on plans' formularies. We set out-of-pocket price equal to average point-of-sale price in the relevant region when Lipitor is excluded from a plan's formulary. This results in Lipitor's out-of-pocket price increasing from \$38 to \$84 between 2011 and 2012. To the extent that some enrollees whose plans dropped Lipitor from the formulary were motivated to purchase Lipitor in cash (in which case the claim would not be recorded in the Medicare Part D data), this will bias our estimates of price sensitivity upward in magnitude.

Figure A3: Out-of-Pocket Price of Atorvastatin/Lipitor, 2011-2012



Note: Reproduced from [Starc and Swanson \(2018b\)](#). Average out-of-pocket price of Lipitor/atorvastatin observed in monthly prescription drug event data. Claims made by non-LIS enrollees for 30-day supply in the initial coverage phase of the drug benefit only. Prices are from claims and are thus conditional on drugs’ formulary inclusion.

Table A1: Lipitor, Atorvastatin, and Crestor Prices – 2011-2012

		Price 2011		First Stage			Price 2012		First stage		Panel	
		Mean	SD	25th	75th	Mean	SD	25th	75th	First stage	First Stage w/ Doc FE	
Lipitor	OOP	38.10	5.09	34.02	41.16	0.702***	84.21	16.16	77.54	95.01	1.197***	1.265***
	POS	114.92	3.62	113.34	113.86	0.378***	136.21	4.60	134.35	134.83	0.356***	1.210***
Atorvastatin	OOP						11.66	3.23	9.77	11.99	0.839***	
	POS						31.18	8.31	28.51	31.62	1.352***	
Crestor	OOP	40.96	7.28	37.09	45.67	0.932***	38.85	6.89	35.31	41.78	1.109***	0.822***
	POS	138.32	4.15	136.55	137.12	0.360***	161.66	4.93	159.60	160.45	0.312***	1.732***

particularly slow to remove Lipitor from its formularies, then Lipitor prices in 2012 would be higher in regions dominated by United HealthCare for reasons unrelated to those regions’ latent price-sensitivity or willingness to substitute to generic equivalents. The association between the point-of-sale and out-of-pocket prices within and across time is in the “first stage” columns in Table A1. There is a strong positive association between the pricing policies of the dominant firms in each region and their pricing policies in other regions – this holds within each year and across years, which we can see in the “first stage” result in the final column that pools years and controls for physician fixed effects.

B Appendix: Payment Data, Construction and Context

B.1 Building the Dataset

The payment data is based on publicly available data released by firms prior to the Sunshine Act-required reporting that began in 2013. When posting these reports, each firm adopted its own standards for specificity,⁶¹ categorization approach,⁶² and accuracy. Physician-level identifiers were ambiguous and often limited to a name, city of address and perhaps a specialty. Furthermore, many of these documents have since been removed from easily accessible websites. During the period that these payments were still posted on the firms' websites, the enterprise software company Kyruus collected these reports as a part of their initiative to analyze physician-firm interactions.⁶³ In order to create a disambiguated physician-level dataset using the unstandardized reports, Kyruus utilized their proprietary machine-learning algorithms to match each individual-firm data point with the physician most likely to be the true recipient. The resulting dataset, generously provided to us by Kyruus, connects each firm-physician-payment to the most probable unique National Provider Identifier – a variable enabling us to merge this data to a number of other datasets.

There is significant heterogeneity in the nature of payments as they relate to the potential for conflict of interest. For example, a physician may receive a royalty payment for an invention sold by a company or a consulting payment for advice on product development. Other payments might not be related to a product at all. We construct two main categories of payments: “research” and “general” (all non-research payments). This scheme closely follows that of Open Payments and excludes all royalty payments. Within general payments we identify three sub-categories: “meals,” “travel or lodging,” and “consulting, speaking or education.” Table A2 summarizes interactions levels for all of the firms, active physicians⁶⁴ and years of data we observe. In the focal analysis, we utilize only payments from Pfizer (which owns Lipitor) and AstraZeneca (which owns Crestor) to active Cardiologists.

The concern for misreporting, and in particular underreporting, in the early years of these documents led us to remove certain firm-year outliers.⁶⁵ To identify those firm-years most

⁶¹For example, while many firms reported whole dollar amounts, Allergan reported payments in large bins uninformative for analyses (e.g. \$1-\$1,000, \$1,001-\$10,000, etc.)

⁶²Some firms utilized three mutually exclusive categories (e.g., consulting, meals, research), while others utilized non-exclusive labels (e.g., meals; meals, consulting; consulting, teaching and education).

⁶³E.g., *Rose, S. L., Sanghani, R. M., Schmidt, C., Karafa, M. T., Kodish, E., and Chisolm, G. M. (2015). Gender differences in physicians' financial ties to industry: A study of national disclosure data. PlosOne.*

⁶⁴Active prescribers here defined as being above the bottom 10th percentile of total annual claims in the Medicare Part D data.

⁶⁵For anecdotes related to the inaccuracies of these early reports, see: *Ornstein, C. and Weber, T. (2010). In Minnesota, drug company reports of payments to doctors arrive riddled with mistakes. Technical report,*

likely to suffer from significant misreporting, we collapsed each firm’s annual total number of payments and payment amounts and dropped any firm-year for which either of these variables were an order of magnitude smaller than the most recent year’s data. Given the relative stability in payment behaviors across firms and over time, we assume these sharp discontinuities were the result of misreporting and not any dramatic change in firm policies.

Table A2: Firm-wide Total Interaction Amounts

Firm	Years	Avg. total, \$M		Avg. total, n	
		General	Research	General	Research
AstraZeneca	2011-2013	\$31.8	\$0.95	115,490	119
Cephalon	2010-2013	\$6.43	\$10.5	27,736	258
EMD-Serono	2011-2013	\$1.81	N.R.	7,070	N.R.
Forest	2012-2013	\$39.8	\$7.66	222,308	422
GlaxoSmithKline	2012-2013	\$9.26	N.R.	40,989	N.R.
Eli Lilly	2011-2013	\$35.8	\$148	85,403	3,079
Merck	2012-2013	\$22.3	\$174	19,038	4,256
Novartis	2012-2013	\$49.9	\$74.4	99,129	2,853
Pfizer	2010-2012	\$39.1	\$93.9	137,012	1,855
Valeant	2010-2013	\$1.78	N.R.	19,549	N.R.

Note: Expenditures and number of payments per year, dollars in millions. General and research payments are defined in text. N.R. indicates type was not reported.

B.2 Comparing the Dataset to Post-Sunshine Act Data

As outlined in the main text, the Kyruus-developed physician-industry interaction data we analyze was available due to the fact that Pfizer and AstraZeneca, among other drug firms, released this information prior to the mandatory reporting regulations of the Sunshine Act, which began reporting in late 2013. Because these disclosures prior to the Sunshine Act occurred on an ad hoc basis without any standardized reporting agency (the interaction files were typically posted on each firm’s website), it is important to provide evidence that this pre-Sunshine Act data is relatively accurate, e.g. it is not censored or biased in any way that would alter our conclusions. To investigate this, we explored post-Sunshine Act data made available by ProPublica⁶⁶, examining trends and distributions under the working assumption that firm-level annual trends in physician payments should be smooth, and within-year distributions of payments should be relatively stable.

Like our Kyruus-developed data, the ProPublica version of the official Sunshine Act data, (available at <https://openpaymentsdata.cms.gov>), is matched to National Provider

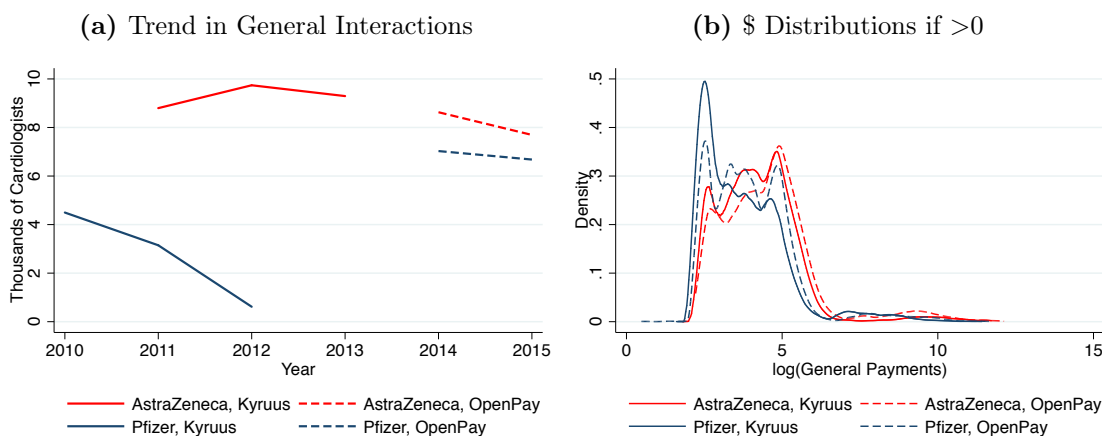
Dollars for Doctors.

⁶⁶<https://www.propublica.org/article/about-the-dollars-for-docs-data>

Identifiers. This enables us to hold fixed our set of cardiologists from the main analyses, and compare payments from Pfizer and AstraZeneca in 2011-2012 (from Kyruus) to those in 2014-2015 (from ProPublica).⁶⁷

Figure A4 Panel (a) plots the total number of our cardiologists (out of roughly 15,000) that receive any general (non-research) payment from the two firms in each year, based on either data source. In the case of AstraZeneca, the trend is clearly smooth between the two data sources, supporting our assumption that the self-reported data is not notably censored in any way. Although the Pfizer trend line appears to be dramatically different across the two data sources, the spike in 2014 can be explained by the fact that this year marked the approval of Eliquis, a joint venture between Pfizer and Bristol Myers Squibb. Eliquis is an anticoagulant for the treatment and prevention of deep vein thrombosis and pulmonary embolisms, thus cardiologists are the most relevant specialty, and in the OpenPayments data – where, unlike in the Kyruus data, the specific drug associated with each interaction is reported – Eliquis accounts for roughly 60% of the interactions with cardiologists and 78% of total spending on cardiologists. Figure A4 Panel (b) indicates very little variation in the distribution of payment dollar values across the data years/data sources, further supporting the notion that our data is not censored or biased in any significant way.

Figure A4: Kyruus vs. OpenPayment Comparison



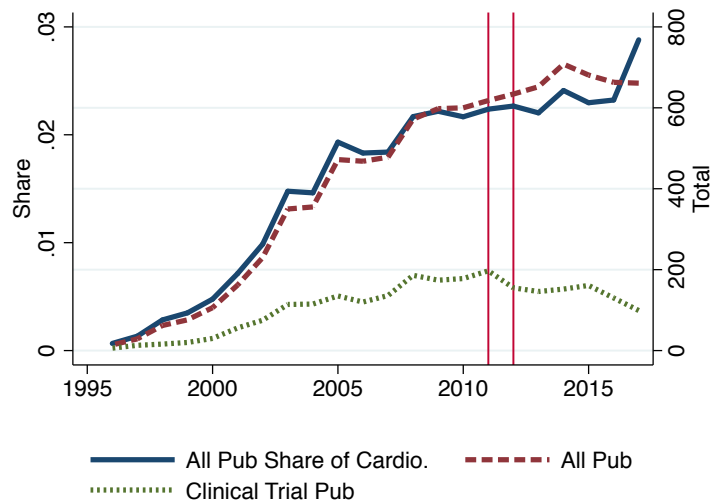
B.3 The State of Science of Statins

A key assumption we make when interpreting our estimates is that the effect we observe is not (significantly) due to a transfer of information from the firms to the physicians. This

⁶⁷2013 is omitted because OpenPayments reporting only includes the last quarter of the year.

would not be an ideal assumption in the early years of a drug’s release, when information surrounding a drug’s mechanism, formulation, indications, etc., would likely not yet be disseminated. However, the statin market was arguably quite mature by 2011-12, 15 and 8 years after Lipitor’s and Crestor’s FDA approvals, respectively. To further support this claim, Figure A5 plots the trend in scientific publications that contain Lipitor or Crestor’s brand or generic (molecule) name in the abstract or title. First examining the full set of publications, we see that in sum or as a share of all cardiovascular-related publications, there was considerable growth in study during the 2000’s; however, the rate of publications stabilized starting around 2008. Examining the subset of publications that were directly linked to clinical trials (the green dashed line), we see further evidence that the rate of study of these statins has been roughly constant since 2008, even declining in recent years. Thus, we interpret these plateauing trends as evidence that the major scientific studies that developed the key pieces of information surrounding these drugs had been completed and their results largely disseminated by 2011-12.

Figure A5: Trends in Science of Statins



Note: Data from *PubMed*, per searches for any journal article with Lipitor, Atorvastatin, Crestor, or Rosuvastatin in either the abstract or title, showing the total count of these publications (right-axis) scaled by the total number of cardiovascular publications (left-axis), or including only those flagged by *PubMed* as being connected to a Clinical Trial (right-axis). Sample years, 2011-12, flagged with vertical lines.

C Additional Tables and Figures

Table A3: Summary Statistics, X and Z Variables

Variable	Mean (s.d.)	AstraZeneca 2012 If \$ Diff.	Variable	Mean (s.d.)	AstraZeneca 2012 If \$ Diff.
X Variables			X Variables		
NPI '10 Claims, Cardio.	2355 (2145)	524.3***	HSA N Cardiol.	49 (86)	-7.3***
NPI '10 Claims, Statins	402 (359)	78.8***	HSA N Docs.	1575 (2365)	-267.1***
NPI '10 Claims, Year	3068 (3073)	686.9***	HSA N AMCs	8.1 (11.5)	-1.16***
NPI Is Faculty	.089 (.285)	-.0919***	HSA N Faculty	275 (610)	-84.1***
NPI N Hosp. Affls.	3.6 (1.4)	.21***	HSA Teach. Hosp. Bed Shr.	.109 (.19)	-.0263***
NPI N Practice Affls.	1.5 (.8)	.08***	HSA Teach. Hosp. Admit Shr.	.119 (.212)	-.0276***
NPI N Specialties	1.4 (.60)	-.02**	HRR-Card. '10 Claims, Cardio.	679 (10572)	-443.2**
NPI N Zip Affls.	1.7 (1.9)	.01	HRR-Card. '10 Claims, Statin	113 (1829)	-77.2**
Hosp-Card. '10 Claims, Cardio.	2085 (1709)	425.8***	HRR-Card. '10 Claims, Year	888 (13921)	-554.2**
Hosp-Card. '10 Claims, Statin	355 (276)	63.1***	HRR Medicare Advnt. N Elgbl.	98694 (181413)	561.6
Hosp-Card. '10 Claims, Year	2712 (2355)	559.9***	HRR Medicare Advnt. % Penet.	21 (13)	-.60***
Hosp. Faculty Shr.	.077 (.146)	-.0555***	HRR Pop. %Uninsured	11 (5)	1.4***
Hosp. N Beds	353 (345)	-17.5***	HRR Pop. %Medicaid	23 (7)	.3***
Hosp. N Admits.	17455 (17333)	-955.8***	HRR Faculty Shr.	.029 (.018)	-.0015***
Hosp. N Cardiol.	18 (19)	-2.5***	HRR N Cardiol.	142 (151)	-5.6**
Hosp. N Docs.	380 (349)	-76.7***	HRR N Docs.	4630 (4613)	-484.4***
Hosp. N AMC Affls.	4.3 (3.6)	-.48***	HRR N AMCs	16 (15)	-.4*
Hosp. N Faculty	57 (163)	-57.3***	HRR N Faculty	423 (843)	-60.1***
HSA-Card. '10 Claims, Cardio.	796 (4577)	47.2	HRR Teach. Hosp. Bed Shr.	.145 (.151)	-.0026
HSA-Card. '10 Claims, Statin	140 (769)	11.7	HRR Teach. Hosp. Admit Shr.	.166 (.178)	-.0025
HSA-Card. '10 Claims, Year	1045 (5895)	71	Z Variables		
HSA Medicare Advnt. N Elgbl.	118814 (198980)	-2207.9	HSA AMSA CoI	26 (2)	-.2***
HSA Medicare Advnt. % Penet.	24 (14)	-1***	HSA AMSA CoI, Wgt.	3.5 (1.9)	-.25***
HSA Pop. %Uninsured	11 (4)	1.3***	HRR AMSA CoI	26 (3)	-.1**
HSA Pop. %Medicaid	22 (8)	-.2	HRR AMSA CoI, Wgt.	2.4 (1.2)	-.14***
HSA Faculty Shr.	.036 (.023)	-.0044***			

Note: NPI-level means (standard deviations in parentheses) for the 46 baseline X controls and 4 Z instruments. As cross-sectional variables, these do not vary over time. The third columns present unconditional t-tests of means across physicians that do and do not receive a meal from AstraZeneca in 2012 (i.e. for variable v , $\text{mean}(v^{meal}) - \text{mean}(v^{nomeal})$). Consistent with what is observed in the first-stage analyses of our instruments, paid physicians have lower AMSA CoI scores on average.

C.1 Preventing Overfitting via LASSO

This and all LASSO regressions use common machine learning practices of: 10-fold cross-validation (split data set into 10 equal parts, and use each in turn as the holdout sample on which the model trained on the other 9 is tested) at 100 potential penalty parameters to select

the simplest model (i.e., the largest penalty) within one standard deviation of the penalty that minimizes the mean RMSE in the hold-out samples of the 10-fold cross validation runs.

The 100 potential penalty parameters range up to a maximum of $MaxPenaltyGuess = 2 \times \max(\tilde{x}'y)$, where \tilde{x} is the pre-standardized regressor matrix and y is the vector of the outcome variable, from a minimum of $[MaxPenaltyGuess/1000]$; the 100 potential penalties are evenly spaced between the minimum and maximum penalty guess values over a log scale. This appears to be the most common approach in machine learning to addressing overfitting problems. We have also used the combined cross-validation, estimation, and averaging approach discussed in Chernozhukov et al. (2017), and found similar results. With our relatively large data and regressor sets, especially in the demand estimation with all statins pooled, the computational savings of separating cross-validation and estimation are significant, so we proceed with this approach in the paper.

C.2 Instruments

As described above, we rely on spillovers from local hospitals' conflict of interest policies to generate identifying variation in meal receipt. We have four candidate instruments: HSA-level average AMSA score across all AMC faculty not affiliated with the focal cardiologist's hospital; HSA-level faculty-weighted average AMSA score across all cardiologists not affiliated with the focal cardiologist's hospital; HRR-level average AMSA score across all AMC faculty not affiliated with hospitals in the focal cardiologist's HSA; and HRR-level faculty-weighted average AMSA score across all cardiologists not affiliated with hospitals in the focal cardiologist's HSA.⁶⁸ In theory, each variable could contribute independent identifying variation: the first and third describe strictness of local AMCs' conflict of interest policies; the second and fourth add information on how many local physicians are faculty; and the different levels of geography may have distinct, additive effects on sales force allocations.

The first four panels of Table A4 show the independent effects of the faculty-weighted hospital-, HSA-, and HRR-level AMSA scores on meal receipt, separately for each firm-year. Column (1) indicates that hospital-level AMSA scores have a strong negative association with meals, but this relationship is not robust to the inclusion of controls in column (2). Columns (3) and (5) show negative associations between HSA- and HRR-level AMSA scores and meals for AstraZeneca and Pfizer 2011. This relationship remains large and significant when controls are included in column (4) and (6). As expected, the first stage is much weaker for Pfizer in 2012, after Pfizer significantly scaled back marketing of Lipitor. Finally, the last two panels in the Table pool firm-years, with and without Pfizer 2012. They indicate

⁶⁸In generating each of the "faculty-weighted" instruments, we assign AMSA scores of zero to non-faculty.

that both HSA- and HRR-level AMSA scores have a meaningful effect on meal receipt, conditional on controls. These results emphasize the importance of including rich controls for physician-, hospital-, and market-level characteristics, and highlight the different effects of hospital-level AMSA scores vs. market-level AMSA scores.

Table A4: “First Stage”: Meals vs. Faculty-Weighted AMSA Scores

Dependent Variable: $1_{\{m_{jdt}>0\}}$						
	Hospital AMSA Scores		HSA AMSA Scores		HRR AMSA Scores	
	(1)	(2)	(3)	(4)	(5)	(6)
AstraZeneca 2011:	-0.0824*** (0.00387)	-0.0238*** (0.00709)	-0.0187*** (0.00431)	-0.0203*** (0.00743)	-0.0122*** (0.00431)	-0.0276*** (0.00871)
N	13274	13274	13274	13274	13274	13274
R ²	0.0281	0.124	0.00145	0.123	0.000612	0.124
Mean(Meal)	0.590					
AstraZeneca 2012:	-0.0907*** (0.00386)	-0.0141* (0.00751)	-0.0310*** (0.00415)	-0.0147** (0.00736)	-0.0283*** (0.00415)	-0.0519*** (0.00844)
N	13762	13762	13762	13762	13762	13762
R ²	0.0358	0.140	0.00417	0.140	0.00347	0.142
Mean(Meal)	0.642					
Pfizer 2011:	-0.0192*** (0.00275)	0.0136*** (0.00519)	-0.00276 (0.00314)	-0.0123** (0.00550)	-0.00704** (0.00316)	-0.0507*** (0.00642)
N	14699	14699	14699	14699	14699	14699
R ²	0.00230	0.0977	0.0000476	0.0977	0.000310	0.101
Mean(Meal)	0.199					
Pfizer 2012:	-0.000734 (0.00187)	0.00244 (0.00188)	0.00684*** (0.00206)	0.00891*** (0.00205)	0.000606 (0.00196)	0.00346* (0.00197)
N	10684	10684	10684	10684	10684	10684
R ²	0.0000131	0.0282	0.00113	0.0300	0.00000889	0.0284
Mean(Meal)	0.0430					
Pooled All Firm-Years:	-0.0507*** (0.00176)	-0.00231 (0.00219)	-0.0134*** (0.00211)	-0.00444* (0.00232)	-0.0145*** (0.00209)	-0.0178*** (0.00230)
N	52419	52419	52419	52419	52419	52419
R ²	0.0109	0.347	0.000761	0.347	0.000885	0.348
Mean(Meal)	0.383					
Pooled exc. Pfizer-2012:	-0.0640*** (0.00208)	-0.00664* (0.00376)	-0.0169*** (0.00244)	-0.0156*** (0.00389)	-0.0160*** (0.00242)	-0.0437*** (0.00450)
N	41735	41735	41735	41735	41735	41735
R ²	0.0164	0.264	0.00115	0.264	0.00102	0.265
Mean(Meal)	0.470					
Incl. $L(X)$		Y		Y	Y	Y
Jackknife			Y	Y	Y	Y

Note: Each estimate and standard error from a separate regression of meal dummy on standardized AMSA variables, with and without LASSO-selected controls. Standard errors clustered at hospital level. Jackknife formulations exclude the AMSA scores from the focal units at the sub-level of aggregation (i.e. the HSA-level measure for doctor i at hospital h and HSA s is the average score for all other doctors in s but not at hospital h).

C.2.1 Meals Model

Here, we modify the supply side to endogenize meals as well as prices. Let the supplier’s profit be:

$$\pi(p_{jbt}^{pos}, m_{jbt}) = \sum_d (q_{jbt}(p_{jbt}^{pos} - mc_{jbt}) - (\bar{m} + c_{dt}^m)1_{\{m_{jbt}>0\}}) \quad (7)$$

where mc_{jbt} captures the cost of manufacturing and distributing the marginal unit of molecule j , \bar{m} is the fixed and exogenous cost of a meal, and c_{dt}^m captures the fixed cost of interacting with physician d (roughly equivalent to a “marketing” cost over and above the dollar value of the meal). Intuitively, the quantity consumed of each drug will depend on the availability of substitutes, relative prices, and meals.

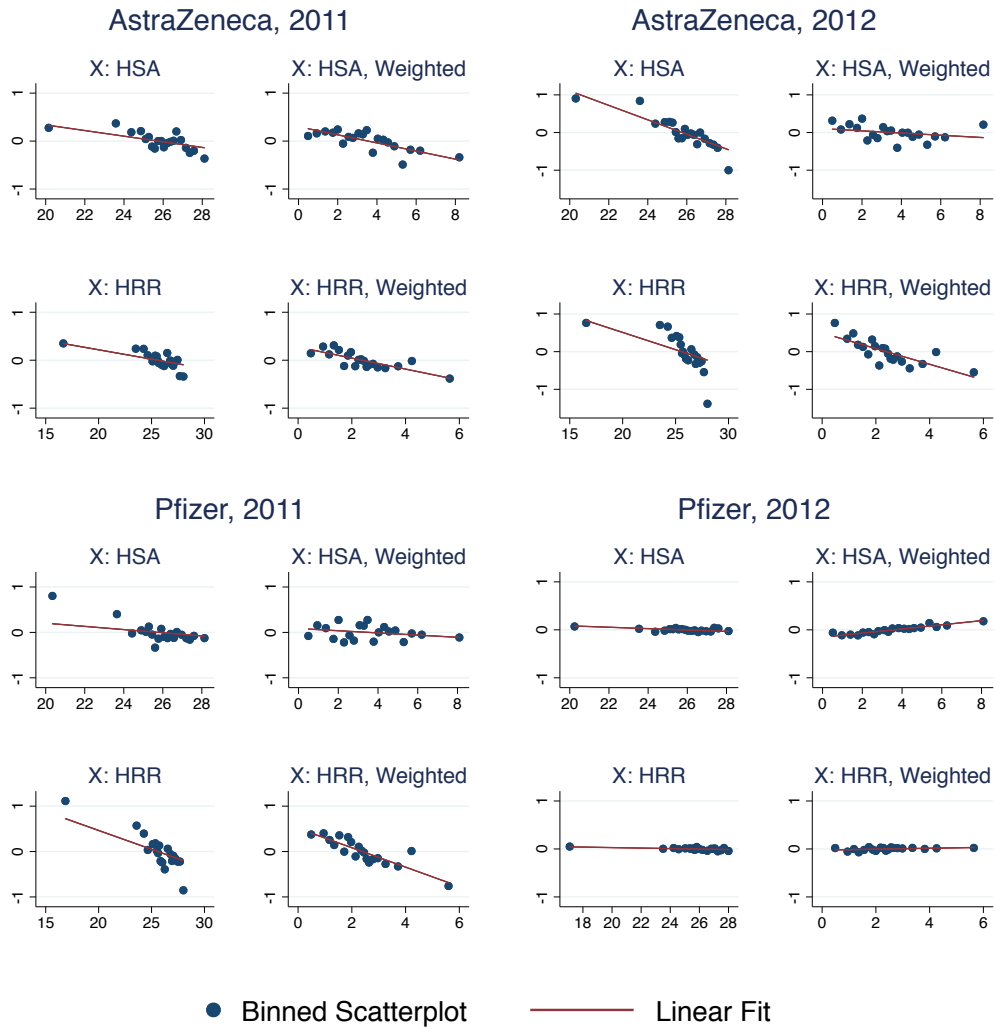
Given our assumption that the point-of-sale price insurers pay for the drug is constant across providers, the firm’s first-order condition on price will be as in the main text, and the first-order condition on meals will be:

$$m_{jbd}^* > 0 \Leftrightarrow (q_{jbd, \{m_{jbd} > 0\}} - q_{jbd, \{m_{jbd} = 0\}})(p_{jbt}^{pos} - mc_{jbt}) > \bar{m} + c_{dt}^m, \quad (8)$$

where \bar{m} is the dollar value of a meal and c_{dt}^m is the physician-specific marginal cost of interaction. Firms give meals to any physician when the meal-induced shift in revenues is greater than the total costs of interacting with that physician. While it is not necessary for our counterfactuals to estimate the parameters underlying this optimality condition, it is worth noting that this condition motivates our identification strategy. The likelihood of a physician receiving meals will depend on the marginal benefit and marginal cost of meals for that physician. For example, if there are lower costs of interacting with physicians that are geographically close to the firm’s headquarters, or of interacting with physicians in large practices, then physicians’ propensity to receive meals will vary in geographic space and in practice size. More to the point, the conflict of interest policies embodied in our instruments are exogenous shocks to the marginal value of interactions.

In the instrumental variables (IV) results in the following Section, we show results both for simple 2SLS on all HSA- and HRR-level instruments, and for our preferred specification that uses the “orthogonal 2SLS approach” described above. To fix ideas, Figure A6 below shows the relationship between the combined, orthogonalized AMSA instrument and each component instrument: jackknifed HSA- and HRR-level AMSA averages, unweighted and faculty-weighted. With the exception of Pfizer 2012, the orthogonalized instrument is significantly negatively associated with each individual instrument in each firm-year, after conditioning out controls for cardiologist, hospital, and market covariates.

Figure A6: Orthogonal 2SLS Instrument vs. Individual AMSA Instruments



Note: Each figure plots standardized values of the orthogonalized instrument \tilde{z} as described in text, vs. each AMSA component instrument.

C.3 Firm-Year-Level LASSO Importance Tables

Table A5: Important Variables, Pfizer 2012

Utilization Equation ($y: \log(Claims_{jdt})$)		Meal Equation ($y: 1_{\{m_{jdt}>0\}}$)	
X Var.	Flexible ΔR^2	X Var.	Flexible ΔR^2
NPI '10 Claims, Statins	-.207	NPI '10 Claims, Year	-.95
HRR N Cardiol.	-.02	HSA Pop. %Medicaid	-.95
HSA Pop. %Medicaid	-.019	Hosp. N Admits.	0
HRR N Docs.	-.014	HSA Medicare Advnt. Penet.	0
HRR Medicare Advnt. N Elgbl.	-.01	HRR Faculty Shr.	0
HSA Medicare Advnt. N Elgbl.	-.01	HRR-Card. '10 Claims, Cardio.	0
HRR Teach. Hosp. Bed Shr.	-.01	HRR Medicare Advnt. Penet.	0
HSA Teach. Hosp. Bed Shr.	-.009	HRR N Docs.	0
HRR Faculty Shr.	-.009	Hosp-Card. '10 Claims, Cardio.	0
HSA Medicare Advnt. Penet.	-.009	HSA Medicare Advnt. N Elgbl.	0
		Z Var.	Flexible ΔR^2
		HSA AMSA CoI, Wgt.	-.042
		HSA AMSA CoI	-.003
		HRR AMSA CoI, Wgt.	-.002
		HRR AMSA CoI	-.002

Table A6: Important Variables, AstraZeneca 2011

Utilization Equation ($y: \log(Claims_{jdt})$)		Meal Equation ($y: 1_{\{m_{jdt}>0\}}$)	
X Var.	Flexible ΔR^2	X Var.	Flexible ΔR^2
NPI '10 Claims, Statins	-.313	HRR Pop. %Uninsured	-.043
NPI '10 Claims, Year	-.023	NPI Is Faculty	-.037
HRR Faculty Shr.	-.006	NPI N Hosp. Affls.	-.037
HSA Medicare Advnt. N Elgbl.	-.005	HRR Teach. Hosp. Bed Shr.	-.033
HRR Pop. %Uninsured	-.004	HRR Medicare Advnt. Penet.	-.03
HRR N Faculty	-.003	HRR Faculty Shr.	-.03
Hosp. N Admits.	-.003	Hosp. N Faculty	-.029
Hosp. N Cardiol.	-.003	Hosp. N Admits.	-.028
HRR Medicare Advnt. Penet.	-.002	Hosp-Card. '10 Claims, Statin	-.026
Hosp-Card. '10 Claims, Statin	-.002	HSA Medicare Advnt. Penet.	-.026
		Z Var.	Flexible ΔR^2
		HRR AMSA CoI	-.007
		HSA AMSA CoI, Wgt.	-.005
		HRR AMSA CoI, Wgt.	-.002
		HSA AMSA CoI	-.001

Table A7: Important Variables, AstraZeneca 2012

Utilization Equation ($y: \log(Claims_{jdt})$)		Meal Equation ($y: 1_{\{m_{jdt}>0\}}$)	
<i>X</i> Var.	Flexible ΔR^2	<i>X</i> Var.	Flexible ΔR^2
NPI '10 Claims, Statins	-.265	HRR Faculty Shr.	-.045
NPI '10 Claims, Year	-.027	NPI N Hosp. Affls.	-.038
HRR N Cardiol.	-.013	NPI '10 Claims, Cardio.	-.032
HRR Faculty Shr.	-.009	NPI Is Faculty	-.032
HSA Medicare Advnt. N Elgbl.	-.009	Hosp. N Admits.	-.032
NPI N Hosp. Affls.	-.008	NPI N Zip Affls.	-.029
Hosp-Card. '10 Claims, Statin	-.006	Hosp-Card. '10 Claims, Statin	-.025
HRR N Docs.	-.004	HRR N AMCs	-.025
NPI '10 Claims, Cardio.	-.004	NPI N Practice Affls.	-.025
HRR Medicare Advnt. Penet.	-.004	HSA Teach. Hosp. Bed Shr.	-.025
		<i>Z</i> Var.	Flexible ΔR^2
		HRR AMSA CoI	-.012
		HRR AMSA CoI, Wgt.	-.009
		HSA AMSA CoI	-.007
		HSA AMSA CoI, Wgt.	-.001

C.4 Firm-Year-Level Table Replications

In the below, we show each of our main 2SLS results separately for each firm-year combination. For each firm-year, we cannot reject (at the 95 percent level) the null hypothesis that the firm-year meal effects are identical to the pooled effect. The effect for Pfizer in 2011 is larger, as might be the case if Pfizer maintains meals where they have the largest impact, even as it allocates less resources to them with the impending patent expiry. The effect for AstraZeneca 2012 is smaller, as might be the case if additional meals provided in 2012 are allocated to more marginal physicians, or if all meal relationships have less of an impact in the presence of generic atorvastatin. Pfizer 2012 has a weak instruments problem where our identification strategy has difficulty predicting the remaining few cardiologists receiving Pfizer payments in 2012. However, as shown in the final panel of each Table, re-running the pooled analysis with Pfizer 2012 excluded provides similar results to our preferred estimates, so Pfizer 2012 does not affect our pooled inference on the meal effect substantially.

It is worth noting that the meal effects within year for AstraZeneca are often just on the margin of significance at conventional levels. Significance levels vary slightly by sample and included controls (compare Tables [A8](#), [A9](#), and [A11](#)). However, point estimates are fairly consistent across these decisions and we consider our baseline specification to be fairly conservative. Accordingly, we do not focus on these small variations in p-values.

Table A8: Main Results, Firm-Year Subsets, $\log(\text{claims})$

	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Pfizer 2011						
β^m	0.454*** (0.0166)	0.172*** (0.0123)	0.974*** (0.160) [27.18]	1.264*** (0.293) [35.98]	1.369*** (0.306) [35.91]	1.101*** (0.254) [44.27]
N_{obs}	14699	14699	14699	14699	14699	14699
N_X		46	46	417.33	252.67	582.33
N_Z			4	9	6	11
Panel B: Pfizer 2012						
β^m	0.368*** (0.0365)	0.125*** (0.0280)	5.104 (3.168) [0.82]	-0.862 (1.971) [2.51]	1.273** (0.631) [15.46]	4.675 (7.840) [0.47]
N_{obs}	10684	10684	10684	10684	10684	10684
N_X		46	46	257.33	49.33	394.33
N_Z			4	4	4	6
Panel C: AstraZeneca 2011						
β^m	0.300*** (0.0150)	0.159*** (0.0120)	0.866*** (0.314) [6.07]	0.805 (0.543) [7.69]	1.363** (0.656) [7.71]	0.457 (0.399) [13.67]
N_{obs}	13274	13274	13274	13274	13274	13274
N_X		46	46	320.67	154	436.67
N_Z			4	9	6	9
Panel D: AstraZeneca 2012						
β^m	0.266*** (0.0151)	0.149*** (0.0126)	0.597*** (0.198) [15.31]	0.341 (0.227) [41.45]	0.496** (0.208) [51.43]	0.466** (0.186) [58.66]
N_{obs}	13762	13762	13762	13762	13762	13762
N_X		46	46	379.67	229	503.67
N_Z			4	9	8	14
Panel E: Pooled, Excl. Pfizer-'12						
β^m	0.186*** (0.00988)	0.159*** (0.00832)	0.820*** (0.143) [12.58]	0.722*** (0.192) [61.31]	0.910*** (0.188) [69.19]	0.649*** (0.156) [93.85]
N_{obs}	41735	41735	41735	41735	41735	41735
N_X		138	138	1117.67	635.67	1522.67
N_Z			12	27	20	34
Spec.	OLS	OLS	2SLS	O-2SLS	O-2SLS	O-2SLS
Incl. X		X	X	$L(X)$	$L^+(X)$	$L^{min}(X)$
X Set		B	B	E	E	E

Variable sets: “B” = baseline, totaling 46 X and 4 Z variables; “E” = exploded baseline set via interactions, logs, and squares, totaling 1173 X and 18 Z variables; all models with X controls also include firm-year fixed effects. The preferred LASSO penalty λ (Col. 4) is chosen via cross-validation (“CV”) as the largest λ within 1 s.e. of the out-of-sample MSE-minimizing λ ; alternative control sets $L^+(X)$ and $L^{min}(X)$ formed using penalties that are one log point higher than the 1 s.e. λ and the MSE-minimizing λ , respectively. $N_{X/Z}$ indicates the total number of control/instrumental variables selected across firm-years, averaging the number of X variables selected across the 3 selection routines within each firm-year. Standard errors clustered at the physician-level are shown in parentheses. F-statistics are shown in brackets.

Table A9: Specification & Robustness Tests, $\log(Claims)$

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Panel A: Pfizer 2011										
β^m	1.264*** (0.294) [35.77]	1.271*** (0.286) [37.75]	1.307*** (0.311) [32.68]	1.262*** (0.327) [28.25]	1.412** (0.576) [10.67]	1.568* (0.951) [5.04]	1.143*** (0.268) [39.13]	1.154*** (0.275) [72.73]	1.126*** (0.302) [34.17]	0.916*** (0.236) [34.6]
N	14699	14699	13553	10112	6429	2818	14694	14699	14699	14699
Mean(Claims)	102.2	102.2	104.3	103.4	101.9	100.8	102.2	102.2	102.2	102.2
Mean(Meal)	0.199	0.199	0.204	0.201	0.195	0.186	0.199	0.199	0.199	0.199
Panel B: Pfizer 2012										
β^m	-0.858 (1.971) [2.51]	-0.574 (1.943) [2.46]	-0.119 (1.365) [4.81]	0.0538 (2.974) [0.91]	0.401 (1.483) [2.75]	0.167 (0.943) [8.87]	0.693 (2.091) [2.04]	0.189 (0.505) [40.17]	-1.861 (2.395) [2.35]	-0.617 (1.782) [2.45]
N	10684	10684	9915	7238	4516	1945	10679	10684	10684	10684
Mean(Claims)	37.50	37.50	38	37.50	37	37.40	37.50	37.50	37.50	37.50
Mean(Meal)	0.0430	0.0430	0.0430	0.0410	0.0370	0.0340	0.0430	0.0430	0.0430	0.0430
Panel C: AstraZeneca 2011										
β^m	0.796 (0.544) [7.63]	0.893 (0.581) [7.05]	0.807 (0.593) [6.44]	2.316 (2.494) [1.02]	1.356 (1.058) [2.98]	0.875 (0.641) [5.91]	0.676 (0.507) [8.23]	0.504 (0.493) [15.46]	0.540 (0.517) [9.15]	0.805 (0.543) [7.69]
N	13274	13274	12292	9163	5837	2528	13269	13274	13274	13274
Mean(Claims)	82.20	82.20	83.90	85.70	88.20	90	82.20	82.20	82.20	82.20
Mean(Meal)	0.590	0.590	0.610	0.618	0.604	0.583	0.590	0.590	0.590	0.590
Panel D: AstraZeneca 2012										
β^m	0.339 (0.227) [41.72]	0.376 (0.244) [36.37]	0.293 (0.220) [44.29]	0.415* (0.235) [39.46]	0.516* (0.292) [25.64]	0.489 (0.469) [10.82]	0.266 (0.230) [40.14]	0.603* (0.335) [36.36]	0.171 (0.245) [41.72]	0.340 (0.226) [41.92]
N	13762	13762	12709	9476	6027	2599	13757	13762	13762	13762
Mean(Claims)	86.90	86.90	88.70	89.90	92.20	93.60	86.90	86.90	86.90	86.90
Mean(Meal)	0.642	0.642	0.664	0.671	0.663	0.652	0.642	0.642	0.642	0.642
Panel E: Pooled										
β^m	0.734*** (0.195) [60.25]	0.772*** (0.199) [59.31]	0.715*** (0.194) [59.78]	0.816*** (0.223) [46.92]	0.827*** (0.279) [31.4]	0.811** (0.379) [19.66]	0.630*** (0.187) [62.47]	0.786*** (0.225) [109.29]	0.560*** (0.209) [61.01]	0.651*** (0.190) [60.14]
N	52419	52419	48469	35989	22809	9890	52399	52419	52419	52419
Mean(Claims)	79.90	79.90	81.50	82.10	83	83.70	79.90	79.90	79.90	79.90
Mean(Meal)	0.383	0.383	0.395	0.399	0.392	0.380	0.383	0.383	0.383	0.383
Panel F: Pooled, Excl. Pfizer-'12										
β^m	0.722*** (0.192) [61.22]	0.778*** (0.198) [59.13]	0.708*** (0.191) [60.82]	0.812*** (0.221) [47.33]	0.835*** (0.282) [30.26]	0.829** (0.389) [18.63]	0.628*** (0.184) [63.62]	0.838*** (0.238) [94.34]	0.544*** (0.206) [61.94]	0.660*** (0.187) [61.22]
N	41735	41735	38554	28751	18293	7945	41720	41735	41735	41735
Mean(Claims)	90.80	90.80	92.70	93.30	94.30	95	90.80	90.80	90.80	90.80
Mean(Meal)	0.470	0.470	0.485	0.489	0.480	0.465	0.470	0.470	0.470	0.470
Own AMSA	Y	Y								
Hosp. AMSA		Y								
Drop Faculty			Y	Y	Y	Y				
No Fac Hosp.				P-P	P-A	A-A				
Census Div FE							Y			
Vol. Controls								None	y,c	y,c,s,d

All models estimated via orthogonalized 2SLS using only LASSO-selected variables from the exploded set, selected separately at the firm-year level, all including firm-year-fixed effects. "No Faculty at Hospital" columns drop any physician whose own primary (P) hospital is also the primary (P) hospital of any faculty (P-P), whose own primary hospital is any (A) of a faculty's affiliated hospitals (P-A), etc. Volume controls legend: y-Year, c-Cardiovascular, s-Statins, d-Focal Drug; main specification in other tables is y,c,s.

Table A10: Main Results, Any Claims

	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Pfizer 2011						
β^m	0.0504*** (0.00314)	0.0225*** (0.00309)	0.116** (0.0570) [27.71]	0.0516 (0.0913) [38.79]	0.00123 (0.0912) [39.46]	0.0595 (0.0844) [45.99]
N_{obs}	15569	15569	15569	15569	15569	15569
N_X		46	46	301	187.33	462
N_Z			4	9	6	11
Panel B: Pfizer 2012						
β^m	0.138*** (0.0167)	0.0240 (0.0169)	4.172 (2.728) [.79]	-2.334 (2.096) [2.56]	0.584 (0.494) [19.19]	4.781 (4.064) [1.79]
N_{obs}	15569	15569	15569	15569	15569	15569
N_X		46	46	194	11.67	339
N_Z			4	4	4	6
Panel C: AstraZeneca 2011						
β^m	0.112*** (0.00585)	0.0781*** (0.00574)	0.289** (0.133) [7.23]	0.178 (0.241) [7.19]	0.335 (0.205) [10.33]	0.405* (0.222) [11.86]
N_{obs}	15569	15569	15569	15569	15569	15569
N_X		46	46	299.67	145.33	408.33
N_Z			4	9	6	9
Panel D: AstraZeneca 2012						
β^m	0.0879*** (0.00563)	0.0633*** (0.00554)	0.123* (0.0743) [19.08]	0.115 (0.0947) [45.94]	0.130 (0.0828) [58.07]	0.118 (0.0738) [66.52]
N_{obs}	15569	15569	15569	15569	15569	15569
N_X		46	46	354	215	489.67
N_Z			4	9	8	14
Panel E: Pooled, Excl. Pfizer-'12						
β^m	0.0497*** (0.00317)	0.0589*** (0.00356)	0.148*** (0.0559) [13.57]	0.102 (0.0784) [63.04]	0.120* (0.0674) [77.79]	0.131** (0.0638) [96.51]
N_{obs}	46707	46707	46707	46707	46707	46707
N_X		138	138	954.67	547.67	1360
N_Z			12	27	20	34
Spec.	OLS	OLS	2SLS	O-2SLS	O-2SLS	O-2SLS
Incl. X		X	X	$L(X)$	$L^+(X)$	$L^{min}(X)$
X Set		B	B	E	E	E

Variable sets: “B” = baseline, totaling 46 X and 4 Z variables; “E” = exploded baseline set via interactions, logs, and squares, totaling 1173 X and 18 Z variables; all models with X controls also include firm-year fixed effects. The preferred LASSO penalty λ (Col. 4) is chosen via cross-validation (“CV”) as the largest λ within 1 s.e. of the out-of-sample MSE-minimizing λ ; alternative control sets $L^+(X)$ and $L^{min}(X)$ formed using penalties that are one log point higher than the 1 s.e. λ and the MSE-minimizing λ , respectively. $N_{X/Z}$ indicates the total number of control/instrumental variables selected across firm-years, averaging the number of X variables selected across the 3 selection routines within each firm-year. Standard errors clustered at the physician-level are shown in parentheses. F-statistics are shown in brackets.

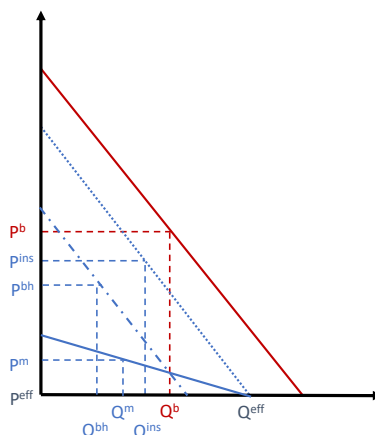
Table A11: Main Results across State Policies, Firm-Year Subsets, $\log(Claims)$

	No Rules		Restrict		Restrict / Report	
	RF	IV	RF	IV	RF	IV
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Pfizer 2011						
Orthog.-IV, or β^m	1.222*** (0.219)	1.288*** (0.309) [32.61]	1.804 (1.878)	57.00 (976.9) [0]	1.108 (0.964)	0.831 (0.790) [4.49]
N	14011	14011	176	176	688	688
Mean(Claims)		103.5		47.70		75
Mean(Meal)		0.204		0.006		0.102
Panel B: Pfizer 2012						
Orthog.-IV, or β^m	0.374 (1.361)	-0.443 (1.637) [3.8]	4.558 (10.83)	-2.640 (6.016) [4.59]	4.881 (4.472)	2.664 (4.765) [0.35]
N	10237	10237	71	71	447	447
Mean(Claims)		37.80		22.30		30.90
Mean(Meal)		0.0430		0.0140		0.0470
Panel C: AstraZeneca 2011						
Orthog.-IV, or β^m	0.695* (0.395)	1.120 (0.775) [4.61]	-2.135 (2.893)	3.411 (8.722) [0.19]	-0.0426 (1.725)	-0.0115 (0.464) [9.68]
N	12657	12657	161	161	617	617
Mean(Claims)		82.90		47.50		69.50
Mean(Meal)		0.604		0.0250		0.301
Panel D: AstraZeneca 2012						
Orthog.-IV, or β^m	0.502** (0.238)	0.465** (0.223) [44]	-2.512 (1.553)	-9.568 (25.76) [0.17]	-2.602** (1.056)	4.659 (5.502) [0.66]
N	13119	13119	166	166	643	643
Mean(Claims)		87.80		49.10		68.40
Mean(Meal)		0.657		0.0540		0.336
Panel E: Pooled, Excl. Pfizer-'12						
Orthog.-IV, or β^m	0.781*** (0.178)	0.817*** (0.203) [57.71]	-1.254 (1.168)	-2.661 (4.174) [0.57]	-0.951 (0.725)	-1.130 (1.222) [2.38]
N	39787	39787	503	503	1948	1948
Mean(Claims)		91.80		48.10		71.10
Mean(Meal)		0.481		0.0280		0.242

All IV models estimated via orthogonalized 2SLS, RF models using the orthogonal instrument as the independent variable, using only LASSO-selected variables from the exploded set, selected separately at the firm-year level, also including firm-year-fixed effects. Instruments and residuals are calculated on the full sample, and then the regressions are separately estimated on the state subsets. “Restrict” states had some sort of restriction imposed on industry interactions (i.e. could not take place at physician’s office); “Report” states had mandates that all industry interactions must be reported to authorities for public dissemination.

C.5 Alternative Graphical Theory

Figure A7: Welfare Analysis with Behavioral Hazard



C.6 Alternative Demand Specifications

Figure A12 displays the full set of parameter estimates and related statistics on elasticities and variation in prescribing for our main specification (column (1)) and several alternative specifications. Column (2) displays results where we allow Lipitor 2012 to have its own selected x and coefficients, and its own fixed effect. This is the strongest set of controls we can introduce for the variation in Lipitor prescribing/meals other than price as generic atorvastatin enters the market. The downside is that it also controls for the effect of generic atorvastatin on Lipitor as a source of demand parameter identification. As a result, the price coefficient becomes smaller, as does the nesting parameter on atorvastatin. Because we do not think anything has unobservably changed about Lipitor that we cannot control for, we prefer to use this as a credible source of demand identification – importantly, our results remain the same if we allow for Lipitor 2012 to have its own x and coefficients to control for regional changes in, say, other promotional activities.

Columns (3) and (4) allow for different nesting specifications. Column (3) nests all statins vs. the outside good. It gives results that are qualitatively similar to our preferred specification, but it doesn't account for the fact that Lipitor and generic atorvastatin are close substitutes, and so the meal elasticity increases, intuitively because the model is now in part using Crestor's meals in 2012 to explain some of the lack of substitution from Crestor to generic atorvastatin. Column (4) nests the "strong" statins (atorvastatin/Lipitor and rosuvastatin/Crestor), other statins, and the outside good. Again, the results are qualitatively

similar, but placing Crestor in the same nest as atorvastatin again increases the estimated meal effect. Thus we choose our preferred model (1) because it seems to respect the most important institutional details and their interaction with the identifying variation in the data.

Table A12: Alternative Demand Specifications

	(1)	(2)	(3)	(4)
θ_{post}	-0.0863*** (0.00244)	-0.0817*** (0.00235)	-0.00498* (0.00286)	-0.0768*** (0.00285)
θ^m	0.877*** (0.0909)	0.718*** (-0.0828)	0.633*** (0.0629)	1.773*** (0.123)
θ^p	-0.00193*** (0.000624)	-0.000974 (0.000611)	-0.00306*** (0.000296)	-0.00449*** (0.000688)
$\lambda^{Ator=1}$	0.497*** (0.0213)	0.187*** (0.0549)		
$\lambda^{Ator=0}$	0.453*** (0.0264)	0.481*** (0.0256)		
λ^{Statin}			0.724*** (0.0158)	
$\lambda^{SS=1}$				0.376*** (0.0202)
$\lambda^{SS=0}$				0.419*** (0.0234)
$\overline{\eta}_p$ (own)	-0.0649	-0.0294	-0.217	-0.138
$\overline{\eta}_m$ (own)	1.254	1.018	1.893	2.392
$sd(\theta_{jb} + x'_{jbd} \beta_{jb})$	1.134	1.183	1.610	1.040
$sd(\theta_{jb} + x'_{jbd} \beta_{jb} + \xi_{jd})$	1.216	1.243	1.621	1.300

$N = 124, 876$ doctor-drug-brand-year observations with standard errors clustered at the doctor level ($N_d = 15, 063$). Estimates for nested logit demand $\ln(s_{jbd}/s_{0dt}) = \lambda^{gj} \ln(s_{j|g} dt) + \theta^m 1_{\{m_{jbd} > 0\}} - \theta^p p_{jbd} + \theta_j + \theta_b + \theta_t + x'_{jbd} \beta_{jb} + \xi_{jbd}$, 2011-12.

C.7 Demand Instruments and Identification

The top panel of Table A13 shows the demand parameter estimates for several different specifications, to help to illustrate how our instrumental variables move coefficient estimates. Column (1) estimates the model with no excluded instruments – all variables are instruments for themselves. The nesting parameters are large while the parameters on price and meals are very small in magnitude. The approximately zero price coefficient is what we might expect if, for example, changes in prices with the introduction of generic atorvastatin are larger for products whose residual demand is more affected.

Column (2) adds our instruments $\{z^a, z^p, z^j\}$ discussed above, and removes the within-group share $\ln(s_{|g})$ and price p terms from the instrument set. The nesting parameters decrease, consistent with the identification strategy correcting for the mechanical correlation of these terms with the unobservable. The magnitude of the price coefficient increases in

magnitude, as expected if unobserved product attributes are positively correlated with price. Column (3) also replaces the meal indicator $1_{\{m>0\}}$ with z^m in the instrument set, to now instrument for all endogenous variables, and the coefficient on meals increases, similar to what we observed in the 2SLS regressions of quantities on meals.

Table A13: Demand and Supply Parameter Estimates

	(1)	(2)	(3)
θ^m	0.048 (0.004)	0.078 (0.005)	0.88 (0.09)
θ^p	0.0000 (0.000)	-0.0017 (0.001)	-0.0019 (0.0006)
λ^{g1}	0.65 (0.01)	0.59 (0.02)	0.50 (0.02)
λ^{g0}	0.95 (0.00)	0.55 (0.02)	0.45 (0.03)
$\overline{\eta}_p$ (own)	0.01	-0.07	-0.06
$\overline{\eta}_m$ (own)	0.46	0.13	1.25
$sd(\theta_j + x'_{jbd} \beta_{jb})$			1.13
$sd(\theta_j + x'_{jbd} \beta_{jb} + \xi_{jd})$			1.22
IV for:	—	$\ln(s _g), p$	$\ln(s _g), p, 1_{\{m>0\}}$

$N = 124,915$ doctor-drug-brand-year observations with standard errors clustered at the doctor level ($N_d = 15,074$). Estimates for nested logit demand $\ln(s_{jbd}/s_{0dt}) = \lambda^{gj} \ln(s_{jblgdt}) + \theta^m 1_{\{m_{jbd} > 0\}} - \theta^p p_{jbd} + \theta_j + \theta_b + \theta_t + x'_{jbd} \theta_{jb}^x + \xi_{jbd}$, 2011-12.

C.8 Standard Errors on Counterfactuals

Table A14: Welfare and Counterfactual Estimates

	2011			2012		
	(1) Obs	(2) Ban $m = 0$ p_{oop}^* p_{pos}^*	(3) Eff $m = 0$ p_{oop}^{mc} p_{pos}^*	(4) Obs	(5) Ban $m = 0$ p_{oop}^* p_{pos}^*	(6) Eff $m = 0$ p_{oop}^{mc} p_{pos}^*
$Q_{statins}$ (millions)	6.05 (0.01)	5.63 (0.01)	5.77 (0.01)	6.43 (0.01)	6.17 (0.01)	6.31 (0.01)
$Q_{atorvastatin}$	1.38 (0.00)	1.18 (0.00)	1.26 (0.01)	2.03 (0.00)	2.04 (0.00)	2.13 (0.01)
$Q_{Crestor}$	0.97 (0.00)	0.47 (0.00)	0.53 (0.01)	0.99 (0.00)	0.46 (0.00)	0.52 (0.01)
$\bar{p}_{statins}$ (\$, OOP)	19 (0.01)	15 (0.01)	0 (0.00)	16 (0.01)	13 (0.01)	0 (0.00)
$\bar{p}_{atorvastatin}$	39 (0.01)	39 (0.01)	0 (0.00)	24 (0.01)	23 (0.01)	0 (0.00)
$\bar{p}_{Crestor}$	42 (0.01)	41 (0.02)	0 (0.00)	38 (0.01)	37 (0.01)	0 (0.00)
PS_{retail} (\$ millions)	113.7 (0.15)	86.0 (0.24)	0 (0)	100.0 (0.14)	79.2 (0.16)	0 (0)
PS_{meals}	-0.5 (0)	0 (0)	0 (0)	-0.9 (0)	0 (0)	0 (0)
CS_{retail} (\$ millions)	3452 (194.1)	3190.6 (179.9)	3279.5 (179.7)	3685.8 (207.1)	3542.2 (198.5)	3608.6 (198.2)
CS_{meals}	-451.6 (26.7)	0 (0)	0 (0)	-312.2 (18.45)	0 (0)	0 (0)
TS (\$ millions)	3113.6 (169.0)	3276.5 (179.9)	3279.5 (179.7)	3472.5 (189.7)	3603.4 (198.5)	3608.6 (198.2)
$POS_{transfers}$	254.1 (0.3)	187.1 (0.6)	290.1 (1.2)	195.6 (0.3)	130.6 (0.5)	230.4 (1.2)

Welfare estimates using data (Obs) and counterfactual equilibrium (Ban and Eff) quantities and prices. $N = 124,876$ doctor-drug-brand-year observations with standard errors clustered at the doctor level ($N_d = 15,063$) via delete-120 jackknife.