Welfare Effects of Physician-industry Interactions: Evidence From Patent Expiration

(PRELIMINARY — do not cite without permission)

Aaron Chatterji*, Matthew Grennan†, Kyle Myers‡, Ashley Swanson†

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Abstract

Many transactions occur via expert advisors, especially in the healthcare sector, and as such, firms frequently implement strategies to influence these advisors. The efficiency of these interactions is an empirical question. Using data on physician-industry interactions and prescribing behavior during the entry of a major generic statin drug, we examine the causal effect and welfare implications of the most common type of interaction: meals. Guided by a theoretical model of endogenous meals, we develop an instrumental variables identification strategy and document reduced form evidence that these meals directly influence prescribing decisions. We find a significant degree of negative selection and primarily extensive margin effects, whereby firms target small meals to prescribers with an otherwise low propensity to use the target drug. Given this evidence, we estimate a structural model of drug choice that allows us to predict counterfactual outcomes in a world where these meals are banned. Results from these counterfactuals are in line with theoretical predictions that these interactions can offset efficiency losses due to pricing with market power. However, this offset does not appear large enough to justify their existence in this particular market.

*Duke University, The Fuqua School & NBER

†University of Pennsylvania, The Wharton School & NBER

‡NBER

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Interactions between firms and consumers often occur via expert advisers. In health care and financial services for example, consumers often select a product in conjunction with an intermediary, typically a physician or a certified financial adviser. These experts can theoretically provide valuable information about complex products, helping to increase market efficiency. However, because these experts also can receive renumeration from firms selling in the market, their advice could also be potentially biased. Whether this bias is economically significant and reduces efficiency is a contentious and important policy question, animating debates over recent legislative and regulatory initiatives to address conflicts of interest in the United States, including the Physician Payment Sunshine Act in 2010 and Department of Labor's Fiduciary Rule, first proposed in 2015.

Economists have long been interested in studying these types of conflicts and tradeoffs across various settings, where information is imperfect and contracts are costly to enforce. Inderst and Ottaviani (2012) provide a cogent summary of the prior literature on interactions between firms and expert advisers and develop a useful theoretical model of interactions between firms, advisers, and customers, yielding several testable and sometimes counterintuitive predictions. For example, they find that increased disclosure of payments could actually reduce market share for higher-quality firms, possibly reducing welfare.

The pharmaceutical industry offers an ideal setting to study the costs and benefits of expert-industry collaboration and to quantify the tradeoffs involved. In the U.S. pharmaceutical industry, physicians can receive payments and other in-kind compensation, such as meals, from companies that produce products they can prescribe, implant, or recommend. Even if these payments bias physician decisions toward the sponsoring firm, the implications for efficiency are unclear. With sufficient market power, this bias may increase efficiency, though at a cost to patients and/or payers. Moreover, if patients are consuming "too little" of a high-quality product due to countervailing payments from competitors or to other behavioral or market frictions, these kinds of payments to physicians might also increase efficiency.

Many empirical studies to date observe positive correlations between payments and physician prescription behavior.[1] While these cross-sectional results provide some insight into these issues, it is empirically challenging to estimate the *causal* impact of these payments to physicians on prescribing behavior and to determine the ultimate welfare implications. The key challenge is that payments are not randomly allocated, and thus identifying the causal

[1] Kremer et al. (2008) provides a review of early research on this topic, noting that physician responsiveness appears to vary across drugs and that studies accounting for the endogeneity of interactions find lower responsiveness. The vast majority of this initial work lacked data on the specific value of payments exchanged. Yeh et al. (2016) and DeJong et al. (2016) provide more recent cross-sectional evidence documenting this relationship using the actual dollar amounts involved.

impact of payments on physician behavior and other related outcomes is not straightforward. Physicians who receive payments may be different than those who do not in their preferences, access to information, and flexibility in prescribing. A second key challenge has been that researchers rarely have access to detailed data on payments from specific firms to specific physicians and prices and quantities chosen by consumers.

The contribution of the current paper is twofold. First, we employ new instrumental variables approaches based on cross-sectional exposure of physicians to payments *and* on differential changes in payments to differentially-exposed physicians pre- vs. post-patent expiration for the focal drug. Crucially, we define "exposure" primarily based on practice- and market-level characteristics, making it unlikely that our results are primarily driven by correlation between exposure and physicians' responsiveness to payments. Second, we use structural demand estimates to shed light on the welfare effects of demand inducement in the presence of market power.

To motivate our analyses, we develop a model of physician prescribing and firm payment behavior in the spirit of Inderst and Ottaviani (2012). The model allows for prescribing decisions to be a function of preferences over products, patent protection, prices, and meals (a popular variety of in-kind payment from pharmaceutical firms to physicians, and the one we focus on in our study). We then model supply side decisions regarding drug prices and payments to physicians before and after patent expiration. The first order conditions in the firm's problem demonstrate how prices respond optimally to patent expiration, and how variables that shift the costs of interacting with physicians impact the likelihood of observing meals between physician-firm pairs. We use the first-order conditions to motivate our causal identification strategy, which we use in several reduced form analyses. We build a comprehensive dataset on firm-physician payments based on early firm disclosures of payments, and prices and quantities observed in a large market – physicians prescribing to Medicare Part D enrollees. To our knowledge, this level of richness has only been used in one other study: Carey et al. (2017) also examined causal responses and quality effects of physician-industry interactions.

We use the above first-order condition for meals to argue that, due to economies of scale in setting marketing strategy to a particular physician market, a set of market, hospital, and physician-level variables are highly predictive of meals but plausibly unrelated to physicians' latent responsiveness to payments. We generate a large set of such variables and utilize a LASSO regression to select the most important predictors of payments in the pre-patent-expiration period. We also perform robustness checks in which only market (hospital referral region)-level variables are used and results are similar. For both tractability and identification purposes, we limit our investigation to cardiologists and the statin market in 2011 and 2012,

an empirical setting discussed in further detail below.

Our IV strategy is similar in spirit to Finkelstein (2007), in which the effects of Medicare were identified by differential changes in utilization pre- and post-Medicare introduction in regions with high vs. low pre-Medicare insurance rates.[2] In our setting, we find a set of physician-practice and market characteristics that have a large effect on practices' propensity to receive payments from pharmaceutical firms – we use those characteristics to generate variation in exposure to Lipitor's patent expiration. The richness of our data allows us to examine extensive margin, intensive margin, and nonlinear effects of payments on utilization – most interactions are small $20-50 meals. We also examine heterogeneity in treatment effects with respect to physicians' age and the quality of their educations.

Our 2SLS regression results indicate meal receipt caused a 100 percent increase in cardiovascular share (2.5 p.p.). We also demonstrate that this result is substantially larger than simple panel analyses would indicate, that the effects are not driven by physician-level instruments, and that effects are smaller for physicians with repeat interactions than for physicians that were newly paid.

We then contribute to bridging the gap between reduced form evidence and the theoretical literature on the effects of kickbacks in markets with advisors. Inderst and Ottaviani (2012) highlight the interesting interaction between competition and kickbacks in such settings. They note in particular that hidden kickbacks allow firms to expand market share without having to lower their prices at the same time.[3] In the market for branded drugs, in which most consumption is insured and prices are perhaps even higher than monopoly power alone would confer, kickbacks or payments may increase utilization closer to efficient levels.[4]

In order to examine the interactions between market power and payments to physicians, we estimate a nested logit model to show how patent expiration effects are mediated by price changes and changes in payments in the presence of both branded and generic substitutes. In our setting, patent expiration results in generic entry, pricing changes, and changes in payments for a substitute branded statin (Crestor). These structural estimates indicate that a meal has an equivalent impact to a $118 in out of pocket price. This magnitude is partially driven by the lack of price sensitivity we observe. The primary result of the

[2]Intuitively, the low-insurance areas were more "exposed" to the introduction of Medicare.

[3]They then go on to evaluate the role of kickback disclosure in such settings. Several nice recent papers have come out on this very topic. For example, Pham-Kanter et al. (2012) studied the experience of two states, Maine and West Virginia, that previously implemented similar disclosure laws. Focusing on statins and SSRIs, they show that the disclosure laws in the two states examined had a negligible to small effect on physicians switching from branded therapies to generics and no effect on reducing prescription costs.

[4]Indeed, Huckfeldt and Knittel (2011) show that when a drug's patent expires, the total number of units of that molecule consumed, both brand and generic, falls noticeably around patent expiration, which is not what an ordinary model of demand would predict.

model is that while payments do increase prescribing, on average they do so in a way that offsets the underprovision of statins due to market power keeping prices above marginal cost. The model estimates that, if payments were banned, Lipitor would be under-utilized in 2011—utilization would be on the order of 1.36 million prescriptions vs. 2.21 million at the efficient allocation. The observed payments raise Lipitor utilization to 1.52 million, only partially closing the gap. Even after the sharp price drop upon generic introduction in 2012, the model estimates that atorvastatin is still underprovided. The spillovers from prior Lipitor payments raise prescribing to 2.35 million, which almost closes the gap. On net, all payments (including payments for Crestor) result in underprovision of statins in 2011, but overprovision in 2012 once the price of atorvastatin has declined.

In sum, our results highlight several of the issues motivated by Inderst and Ottaviani (2012). The extent to which payments distort the efficient allocation depends upon their scale relative to that of the distortion due to market power maintaining high prices, and to the prices, payments, and quality of close substitutes. In the market studied here, payments move the market closer to the efficient allocation. However, banning payments results in an increase of $37.1M (1.8 percent) in consumer surplus. These consumer surplus losses outweigh producer gains, resulting in payments being inefficient in terms of total surplus, in spite of moving closer to the aggregate efficient allocation on the extensive margin. Banning payments results in an increase of $8.9M (0.4 percent) in total surplus in the retail market for statins.

These effects hint towards a value in banning payments; however, they omit any welfare effects of physician-industry interactions related to information provision on new drugs, and a full reckoning of their effects on point-of-sale prices and insurance premiums. We leave these issues as fruitful areas to explore in future work.

1 Related Literature

Our study fits into a rich literature on marketing behavior of pharmaceutical manufacturers. See Scott Morton and Kyle (2012) for a review. As the authors highlight, promotion of pharmaceuticals embodies both potential inducements to use firms' products and some scientific information.[5] In our study, we focus on payments from manufacturers to physicians, which is just one component of firms' promotional strategies. These generally include

[5]It is worth noting that such promotion will likely expand the market overall and include some business-stealing; in some cases, firms would like to commit not to advertise for this reason. As noted in Scott Morton and Kyle (2012), 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.

direct-to-consumer advertising, detailing, advertisements in venues targeted to physicians, and payments.

Assessing the actual risk of potential conflicts of interest has traditionally been difficult due to the lack of systematic, longitudinal data on physician payments. Recently however, new data on physician payments has become available following the Physician Payments Sunshine Act, facilitating a number of new studies on this topic (e.g. DeJong et al. 2016) and spurring much commentary (Drazen 2015; Rosenbaum 2015a,b,c; Steinbrook et al. 2015; the entire May 2017 issue of the *Journal of the American Medical Association*). Most of the empirical studies to date document positive correlations between payments and prescription outcomes. For example, Yeh et al. (2016) found an association between industry payments to physicians and the prescribing of brand-name statins; they also found that this association was no longer significant when the analysis was limited to physicians who received $2,000 or less in total payments. In contrast, DeJong et al. (2016) found that physicians who received a single meal promoting the drug of interest had higher rates of prescribing promoted statins, beta-blockers, ACE inhibitors and ARBs, and SSRIs and SNRIs.

A number of studies have moved beyond documenting correlations to present causal evidence of the effects of marketing efforts on prescribing. For example, Shapiro (2016) uses a discontinuity in advertising along the borders of television markets to estimate significant positive effects of television advertising on use of prescription antidepressants; Sinkinson and Starc (2015) exploit shocks to local advertising markets generated by the political advertising cycle to show significant positive effects of statin advertising on demand, as well as positive spillovers from drug advertisements to non-advertised competitors in the same class; and Alpert et al. (2015) examine a large and plausibly exogenous shock to advertising driven by the introduction of Medicare Part D in 2006 and find substantial differential increases in drug utilization that mirror the increases in DTCA after Part D. While the literature on advertising is somewhat more developed, there is also compelling recent evidence on the effects of sales visits to physicians (known as detailing): Larkin et al. (2017) examine the effects of changes in US academic medical centers' policies restricting detailing between 2006 and 2012. They find that restricted detailing was associated with modest but significant reductions in prescribing of detailed drugs across 6 of 8 major drug classes, including statins and antidepressants. Finally, the study that is closest to ours is Carey et al. (2017): in this work, the authors use unprecedentedly rich prescribing data linked to physicians to examine the effects of pharmaceutical manufacturer payments to physicians on prescribing behavior. They show that payments from a drug firm raise expenditures on the firm's products, even controlling for physician selection using physician fixed effects; the results also hold when focusing on patients who changed prescribers. They further analyze the impact of payments

on four major patent expirations – Lipitor, Singulair, Seroquel, and Lexapro – finding that prescribers who had received payments transitioned their patients to the generic equivalents just as quickly as prescribers with no payments.[6]

2 Setting & Data: Cardiologists and the Statin Market, 2011-2012

The pharmaceutical industry is a setting where a large number of firms produce many differentiated products, and physicians specialized in many different fields influence the treatment of a wide range of diseases. To reduce the complexity surrounding why a particular firm might interact with a particular physician while still identifying empirically useful variation, we focus on cardiologists and the Medicare Part D statin market during 2011 and 2012. This timeframe of this particular market is ideal for empirical anlaysis.

The usefulness of this sample in terms of data are as follows: (1) as detailed below, we have physician-firm interaction data for the two major on-brand statin-producing firms at this time, Pfizer (who produced Lipitor) and Astrazeneca (who produced Crestor), accounting for 53% and 28% of branded statin revenue from Medicare Part D in 2011, respectively; and (2) these statins were each the chief source of revenue from cardiologists for these two firms with Lipitor accounting for 84% of Pfizer's cardiologist-based revenues and Crestor similarly accounting for 80% of Astrazeneca's cardiologist-based revenues. That is to say, if a Pfizer or Astrazenca representative was taking a cardiologist out to lunch in this time period, it was very likely that statins were the focus of any drug-related discussions.

Econometrically speaking, this sample includes variation useful for identification purposes because the generic version of Lipitor (Atorvastatin) became available at the very end of 2011. The entry of this generic drug created the customary shocks to absolute and relative prices that follow the loss of exclusivity, and at a very large scale: the total Part D expenditures associated with Lipitor dropped nearly five fold from $2.5 billion (13 million claims) in 2011 to $591 million (2.8 million claims) in 2012. We leverage this large change in our empirical approaches below. Section 2.3 provides a more detailed view of the focal cardiologists and the statin market at this time.

One additional advantage of examining behavior around the end of Lipitor's (atorvastatin's) exclusivity period relates to the role of information. A classic difficulty of identifying the welfare implications of any firm-intermediary interaction is that it is often unclear if the interaction may have included the transfer of useful information regarding the product.

[6] At least two other published studies we are aware of incorporate physician-level fixed effects: Mizik et al. (2004) and Datta et al. (2015).

In healthcare, it is certainly common for physicians to learn about a drug's features (e.g. indications, side-effects, dosage guidelines) via firm-sponsored interactions. Thus, even if reduced form regressions that account for the endogeneity of these interactions identifies a treatment effect, it is not immediately clear that this effect is driven by classical notions of "bias," or rather information transmission changing optimal choices.

However, we contend that by 2011 there was very little information regarding the atorvastatin molecule that cardiologists did not have. By this time, it had been nearly 24 years since the first statin had come to the market and roughly 15 years since Lipitor's FDA approval (and 7 years since Crestor's approval). And most importantly, by definition, the generic version of atorvastatin is chemically identical to its branded predecessor. Given this, we argue that if we document reduced form evidence of a causal effect of interactions on prescribing outcomes, it is unlikely to be due to the physicians becoming any more or less informed as to this particular set of drugs.

2.1 Data on Provider Characteristics and Prescribing Behavior

We obtain data on physician demographics, specialties and affiliations from the Physician Compare database from the Centers for Medicare and Medicaid Services (CMS), which contains all physicians enrolled in Medicare[7]. Given the size of Medicare Part D, this population of physicians is worthy of study in its own right, even if they are not completely representative of all physicians.[8] Each physician's reported location is matched to the Hospital-Referral-Regions (HRR) of the Dartmouth Atlas, which identify market segments driven by patient flows rather than sometimes arbitrary geographical boundaries such as zip codes or counties.[9]

Prescribing behavior is based on the publicly available CMS Part D claims data for 2011 and 2012[10]. This claims data contains prescriber-drug-year rows. The prescriber information includes the National Provider Identifier, which allows us to link claims data to the Physician Compare database as well as the industry-interaction data. The drug information includes the brand and molecule name (if the drug is "generic" these two are equivalent), and the total number of claims and expenses associated with the prescriber-drug-year triplet. Loosely

[7] Available at https://data.medicare.gov/data/physician-compare.

[8] As with the numerous other studies that rely on Medicare Part D data, our study may not correctly estimate the relationship between payments and prescriptions for all physicians and all patients (i.e., including those outside the Medicare population). In terms of generalizability, we might expect physicians treating the elderly to receive higher *levels* of payments from industry given that this population accounts for a majority of prescription drug spending. However, we contend that there are no strong reasons to believe that physicians would *respond* differently to industry payments across payers; e.g., we have no reason to believe that doctors who receive large payments from industry primarily respond by increasing their prescribing of branded drugs to the non-Medicare population.

[9] See www.dartmouthatlas.org.

[10] Available at https://goo.gl/4NhfCZ

speaking, claims approximate prescriptions. More detailed information on prescriptions such as drug dosages and formulation would not necessarily be useful because of differences in molecule-specific prescribing practices, so we are confident that by following prior research (Einav et al. 2015) and studying claims as a primary outcome we are not obfuscating any important variation in therapeutic decisions.

This data also contains the total expenditures associated with the focal claims, which we scale by the claim quantity to arrive at a proxy for price. These expenditures reported include "ingredient cost, dispensing fee, sales tax, and any applicable vaccine administration fees and is based on the amounts paid by the Part D plan, Medicare beneficiary, government subsidies, and any other third- party payers." Thus, these values capture the total amount transferred between all parties involved in each claim. These numbers closely track the average wholesale cost for each drug-year from the Medicaid Drug Utilization Review files. We also obtain the plan-enrollment-weighted average copay for each drug-year from the Medicare Part D Public Use Files, which we use in our demand models in order to more closely track the price relevant to patients and to focus on price variation due to generic introduction as a source of identification.

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 as well as all drugs to their WHO Anatomical Therapeutic Classification (ATC) codes. The ATC codes provide a well-defined hierarchy of drug categories organized to 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 three measures of prescribing outcomes: the share of a focal drug claims for (1) all drugs prescribed by the physician that year, (2) all cardiovascular drugs prescribed that year (ATC code = "C"), and (3) all statins prescribed that year (ATC code = "C10AA").

2.2 Data on Manufacturer Payments to Providers

Although federally mandated reporting of manufacturer-provider payments did not begin until 2013, nationwide interest in these dealings had been growing for some time. By 2010, states had begun to institute their own payment limitations and/or public reporting rules;[11] a number of high-profile lawsuits found conflicts of interest between physicians and manufacturers to be a punishable offense;[12] and calls from politicians and patient advocacy groups

[11] Massachusetts, Minnesota and Vermont each had instituted a mandatory public-reporting system for physician-firm payments.

[12] For example, in 2009 Eli Lilly paid a $1.4 billion fine following allegations of the off-label promotion of its drug Zyprexa (See: https://goo.gl/77xApj). In 2010, Allergan paid a similar fine of $600 million following the illegal promotion of Botox (See: https://goo.gl/g1q1RP).

were gaining significant momentum in the press.[13] Amidst this growing concern, a number of pharmaceutical firms, most importantly Pfizer and Astrazeneca, began to publicly release data on payments to physicians, some preemptively, others due to legal settlements. These documents are the basis of our payments data. Because these were internally generated documents, the disclosures came 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 to create a disambiguated physician-level dataset of payments from Pfizer and Astrazeneca in 2011 and 2012.

Our analyses primarily focus on two variables: (1) a dummy that equals one if a physician is reported to received a general (non-research) payment from a firm in a given year, and (2) the dollar-value reported. As is shown in the summary statistics below (Table 1), the vast majority of these "general" payments are in the form of a meal. Therefore, all analyses will focus only on meals since (1) this is by far the most common type of interaction, and (2) amongst the types of interactions reported, these are the most likely to be related to pure persuasion in contrast to, for example, payments associated with consulting or speaking activities, which are much more likely to be due to services rendered. That is not to say these other forms could influence physicians, nor that meals could be venues for information transfer that can ultimately improve patient and/or social welfare. In fact it is precisely this latter point that motivates the structural analyses in Section 6.

2.3 Data Set Construction and Summary Statistics

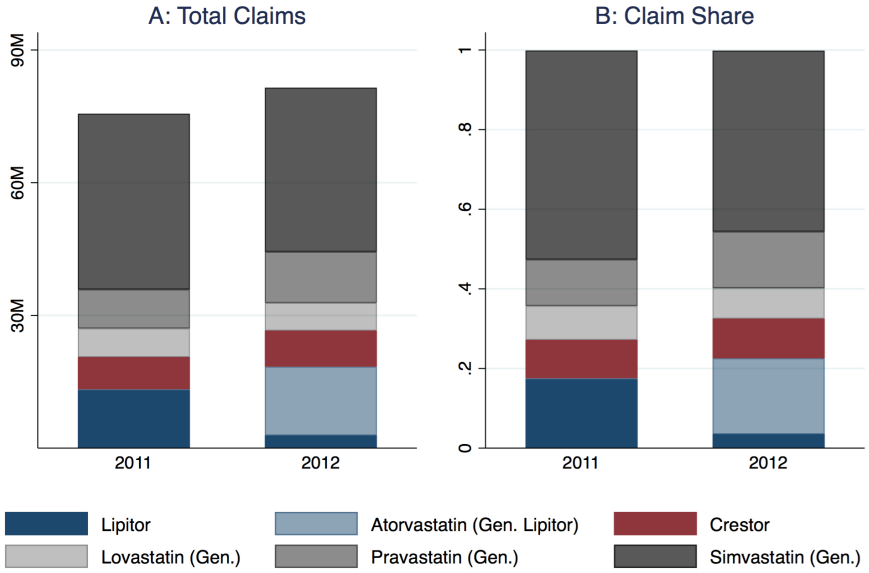
Starting with the full sample of cardiologists in the Medicare Physician Compare database, per their self-reported primary specialty, we restrict our sample to "active" Medicare prescribers by keeping only cardiologists who are responsible for a non-zero number of Part D claims in both 2011 and 2012. The final sample used in our analyses contains 35,676 observations of 17,838 cardiologists.

To first get a sense of the statin prescribing behavior for this set of cardiologists, Figure 1 plots the total annual number and share of claims for the six major statins (2 brand, 4 generic). Together, these six drugs account for more than 99% of the statin claims and total expenditures in this period.

The entry of generic atorvastatin is clear - in its first full year of availability, this new alternative accounted for roughly 19% of statin claims while Lipitor's share dropped from 17% in 2011 to about 3.5% in 2012. During this time, the average (imputed) price of Lipitor increased from 219.58 (s.d. = 54.51) in 2011 to 234.43 (s.d. = 60.50) in 2012, where the

[13] See Senator Chuck Grassley's call for reform here: https://goo.gl/GIPPzF.

Figure 1: Medicare Part D Cardiologist Statin Market, 2011-2012

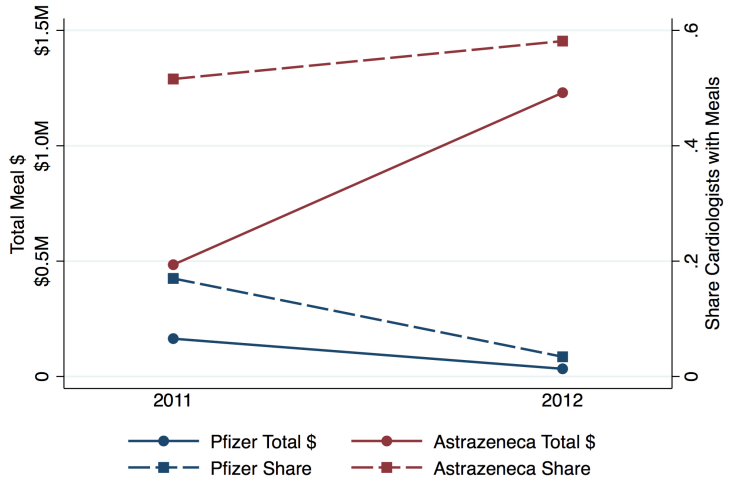


Note: Includes the 6 major statin brands per Medicare utilization, with these 6 drugs accounting for more than 99% of both quantities and expenditures on statins amongst Part D beneficiaries. 2012 was the first full year of generic Atorvastatin's availability. Drugs denoted "Gen." are the generic versions of those molecules, sold by a large number of different companies. The two brand-name statins are Lipitor (Atorvastatin) which is manufactured by Pfizer, and Crestor which is manufactured by Astrazeneca.

average 2012 price of generic atorvastatin was 92.85 (s.d. = 27.04). For comparison, Crestor's average price also increased from 198.28 (s.d. = 50.63) to 228.67 (s.d. = 57.90). Figure 2 highlights the trend in meal-related payments during this period where Pfizer reduced its meal rate for cardiologists from roughly 17% to 3.5%, while on the other hand Astrazeneca increased both its meal rate and value of meal conditional on receipt.

Table 1 describes the average payment amounts (all and the meal-related subset) from Pfizer and Astrazeneca, and the shares of each firm's statin per the cardiologists claim counts. For both firms, meal-related payments account for more than 90% of these interactions, with the vast majority of these meals being valued at less than $200. The table also includes the percentiles of the non-zero distributions for each variable, which for the payment amounts, highlights the extremely skewed nature of these interactions. It is clear that Pfizer and Astrazeneca implement different strategies in this timeframe: cardiologists are nearly 5 times as likely to receive a meal from Astrazeneca compared to Pfizer (50% vs. 10%), and conditional on receiving a meal Astrazeneca's total meal value per cardiologist are twice as large ($48 vs. $24). And in terms of prescribing behavior, the two focal statins account for a roughly similar share of claims when scaled by total annual claims, just claims for cardiovascular drugs,

Figure 2: Firm-wide Meal Expenditures for Cardiologist Sample, 2011-2012



Note: Plots the share of cardiologists included in our sample (N=17,838) that receive a meal from either firm (Right Axis) and the total dollar value of these meals (Left Axis).

and just claims for statins. But as evidenced in Figures 1 and 2, these sample averages mask the large changes in prescribing and payments that occurred from 2011 to 2012.

3 Model: Demand, Supply, and Endogenous Interactions

The goal of this Section is to motivate the identification strategy for our reduced form analysis, and to present a model of supply and demand that allows us to: (1) disentangle own molecule and competitor molecule effects, and (2) compute counterfactual prices and quantities to better understand the welfare effects of industry interactions with physicians.

The utility of molecule $j \in \mathcal{J} = \{1, ..., J\}$ (b denoting branded or generic version) for use case i (a doctor/patient/visit combination) under doctor d in year t is

$$u_{ijbdt} = \delta_{jbdt} + \varepsilon_{ijbdt}. \tag{1}$$

The use-specific i.i.d. unobservable $\varepsilon_{ijbdt} = \epsilon_{idt} + (1 - \lambda)\epsilon_{ijbdt}$ is the random coefficients representation (from Cardell 1997) of the nested logit model where ϵ_{idt} is a random component common to all statins vs. the outside good; and ϵ_{ijbdt} is the standard type I extreme value error term (with scale normalized to one). As the nesting parameter $\lambda \in [0, 1]$ approaches 1, there is less substitution to the outside good.

Table 1: Summary Statistics, Focal Firms & Drugs

	Mean	% > 0	Percentiles if > 0					
			10	25	50	75	90	Max
Panel A: Pfizer & Lipitor								
All Non-research $	40.970 (1116.1)	0.109 (0.312)	11	13	27	90	165	97640
Meal $	5.542 (39.478)	0.102 (0.303)	11	12	24	59	124	4640
Q. Share, Year	0.020 (0.028)	0.727 (0.445)	.0056	.0097	.01952	.0362	.0569	1
Q. Share, Cardio.	0.025 (0.036)	0.727 (0.445)	.0072	.0125	.0248	.0459	.0717	1
Q. Share, Statins	0.136 (0.150)	0.727 (0.445)	.0414	.0689	.1436	.2542	.3661	1
Panel B: Astrazeneca & Crestor								
All Non-research $	277.771 (3567.673)	0.555 (0.497)	14	24	51	114	199	123175
Meal $	48.059 (115.179)	0.549 (0.498)	13	23	48	106	178	3054
Q. Share, Year	0.022 (0.027)	0.775 (0.417)	.0080	.0131	.0219	.0358	.0544	1
Q. Share, Cardio.	0.028 (0.032)	0.775 (0.417)	.0106	.0169	.0279	.0448	.0674	1
Q. Share, Statins	0.146 (0.130)	0.775 (0.417)	.0661	.1007	.1562	.2318	.3251	1

Note: N=35,676 cardiologist-year observations during 2011-2012. Non-research interactions include, for example, speaking fees, consulting payments, reimbursements for travel, meals - all payments disclosed by the firm not explicitly labeled as pertaining to research activities. "Q. Share" are the quantity share of claims for the focal drug (Lipitor/Crestor) as a fraction of the cardiologist total annual claims (Year), total annual claims for any cardiovascular drug (Cardio.) or total annual claims for any Statin.

The mean utility across use cases is specified as

$$\delta_{jbdt} = \theta_{jd} + \theta^m 1_{\{m_{jdt}>0\}} - \theta^p p_{jbdt} + \theta_t + \xi_{jbdt}, \tag{2}$$

where θ_{jd} are molecule-provider specific dummy variables and their utility weights; $\theta^m 1_{\{m_{jdt}>0\}}$ is an indicator for whether provider d received a meal from the manufacturer of molecule j and its weight; and $\theta^p p_{jbdt}$ is the average price paid by patients and its utility weight. θ_t are year-specific dummy variables and their utility weights to capture general market trends; and ξ^{jbdt} is a product-physician-year unobservable preference heterogeneity term.

Given a set of products $\mathcal{J}_t$ 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:

$$q_{jbdt} = Q_{dt} Pr[u_{ijbdt} > u_{ikbdt}, \forall k \in \mathcal{J}_t] = Q_{dt} \frac{e^{\frac{\delta_{jbdt}}{1-\lambda}}}{1 + \left(\sum_{k \in \mathcal{J}_t} e^{\frac{\delta_{kbdt}}{1-\lambda}}\right)^{1-\lambda}}, \tag{3}$$

and consumer surplus across all products is given by:

$$CS_{dt}(\mathcal{J}_t) = Q_{dt} \frac{1}{\theta^p} \ln\left(1 + \left(\sum_{jb \in \mathcal{J}_t} e^{\frac{\delta_{jbdt}}{1-\lambda}}\right)^{1-\lambda}\right) - \sum_{jb} q_{jbdt} \frac{\theta^m}{\theta^p} 1_{\{m_{jdt}>0\}}, \tag{4}$$

which is the standard formula derived by McFadden (1978), minus $\sum_{jb} q_{jbdt} \frac{\theta^m}{\theta^p} 1_{\{m_{jdt}>0\}}$ to adjust for the fact that potential bias due to meals affects decisions, but not utility directly. An equivalent interpretation would be that physicians maximize a sum of physician (chooser) and patient (consumer) utility, and that all terms but $\theta^m 1_{\{m_{jdt}>0\}}$ represent consumer utility.

We next characterize how prices and meals are determined in equilibrium. In the foregoing, the lack of a d subscript on price indicates that all prices are held fixed across providers in each market. Let the supplier's profit function be given by:

$$\pi(p^{pos}_{jbt}, m_{jbdt}) = \sum_d \left(q_{jbdt}(p^{pos}_{jbt} - mc_{jbt}) - (\bar{m} + c^m_{dt}) 1_{\{m_{jbdt}>0\}}\right) \tag{5}$$

where mc_{jbt} is a function capturing the costs of manufacturing and distributing the marginal unit of molecule j, $\bar{m}$ is the fixed and exogenous cost of a meal, and c^m_{dt} captures the fixed cost of interacting with physician d (roughly equivalent to a "marketing" cost over and above the dollar value of the meal). We assume that prices of the substitute drugs in the market are determined in a Nash Equilibrium of Nash Bargaining between suppliers (manufacturers/wholesalers/pharmacies) and buyers (PBMs/insurers). This captures the primary forces relevant to our research question, abstracting from some of the details of

the upstream interactions between manufacturers/wholesalers/pharmacies, and from insurer competition and tiering/formulary details. 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. We assume that meals and prices are determined simultaneously. The first-order condition on each price is (here $q_{jbt} := \sum_d q_{jbdt}$ denotes the sum over physicians):

$$\begin{aligned} p_{jbt} &= \arg\max \left(\pi(p^{pos}_{jbt}, p^{oop}_{jbt}, m_{jbdt})\right)^{b_{jbt}} \left(\frac{\widetilde{CS}_t(\mathcal{J}_t) - \widetilde{CS}_t(\mathcal{J}_t \setminus jb)}{q_{jbt}}\right)^{1-b_{jbt}} \\ &= mc_{jbt} + b_{jbt}\left[\left(1 + \frac{\partial q_{jbt}}{\partial p^{oop}_{jbt}} \frac{p^{oop}_{jbt} - mc_{jbt}}{q_{jbt}}\right) \frac{\widetilde{CS}_t(\mathcal{J}_t) - \widetilde{CS}_t(\mathcal{J}_t \setminus jb)}{q_{jbt}} + p^{pos}_{jbt} - mc_{jbt}\right] \end{aligned} \tag{6}$$

Here, the term $b_{jbt} = \beta_{jb}\nu_{jbt}$ 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 the model captures the important feature of health insurance negotiations that quantities and thus elasticities are driven by consumer 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}, so that the buyer surplus term differs from consumer surplus $\widetilde{CS}_t(\mathcal{J}_t) := CS_t(\mathcal{J}_t) - \sum_{jb} q_{jbt}(p^{pos}_{jbt} - p^{oop}_{jbt})$.

The first-order condition on meals will be:

$$m^*_{jbdt} > 0 \quad \Leftrightarrow \quad (q_{jbdt,\{m_{jbdt}>0\}} - q_{jbdt,\{m_{jbdt}=0\}})(p^{pos}_{jbt} - mc_{jbt}) > \bar{m} + c^m_{dt}. \tag{7}$$

Firms give meals to any physician when the meal-induced shift in revenues is greater than the total costs of interacting with that physician.

Intuitively, the implications of our model, in which prices and interactions are jointly determined and demand depends on both, can be summarized as follows:

- The quantity consumed of the drug will depend on the availability of generic substitutes, relative prices, and meals.
- The marginal return to meals m^* for a given doctor will depend on the provider's panel size Q_{dt}, their responsiveness $q_{jbdt,\{m_{jbdt}>0\}} - q_{jbdt,\{m_{jbdt}=0\}}$, and the unit margin $p^{pos}_{jbt} - mc_{jbt}$.
- For reasonable parameters, generic entry will decrease the marginal return to meals, though there are parameter combinations for which generic entry *increases* the marginal return to meals.
- The likelihood of a physician receiving meals will depend on the marginal return for that physician, but also on the physician-specific marginal cost of interaction c^m_{dt}. 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.

4 Identification Strategy: Approximating the Physician-Targeting Function

The model suggests an identification strategy based on variables that shift the marginal costs (c^m_{dt}) and/or benefits (i.e. Q_{dt}) of interacting with physicians but are plausibly exogenous to those physicians' latent responsiveness to these interactions (θ^m).

A traditional reduced form econometric approach in this vein would be to choose a single variable, such as c^m_{dt}, identify a reasonable proxy in the data, and then assume that the optimal meal level is linear in this proxy (i.e. $\frac{\partial m^*_{jbdt}}{\partial \text{proxy}}$ is constant) and estimate a 2SLS model where interactions are instrumented with the proxy. However, as will be made clear below, there are an enormous number of potential proxies for both the marginal costs and benefits of interactions and the functional form of how any particular variable influences the optimal interaction level is far from obvious.

Instead, our approach will be to identify a large number of potential instruments that would be observable to firms and useful in predicting the costs and benefits of interacting with a particular physician, and then employ LASSO regression to select the final instrument set with most predictive power. In other words, we seek to approximate the function by which firms target their meals to physicians, but only using variables orthogonal to each physician's latent responsiveness.

As evidence of this "physician-targeting function", consider the 2014 civil case levied against the DaVita dialysis company. Charges filed on behalf of the U.S. to the District Court of Colorado claimed that DaVita had violated the False Claims Act, and in support of their charges presented internal documents from DaVita that indicated how the company was explicitly choosing to pursue interactions with physicians located in regions with dense populations of patients who could be referred to their dialysis clinics (See Figure 3). In this particular case, the interactions appear to have largely revolved around the prospects of joint venture agreements between physicians and the dialysis clinics. Although this involves a different level of commitment than the $20-50 meals we study here, the underlying premise is clear: firms have access to patient- and physician-level data for their relevant populations and use it to allocate their resources accordingly.

Figure 3: Physician Targeting Example: DaVita Dialysis Clinics

Note: Internal DaVita documents reveal how the firm utilized data on physicians and patient populations to direct their interactions. Quoting the text of the lawsuit: "The following excerpt from an internal DaVita powerpoint describing the IMS deal shows the precision with which DaVita tracked the potential physician partners' patients and patient locations ... As shown above, in areas where the targeted IMS physicians had patients, DaVita decided to offer a joint venture" - Source: U.S. v. DaVita Inc., and Total Renal Care, Inc., Civil Action No. 09-cv-02175-WJM-KMT.

4.1 Potential Instrument Set

This section outlines our inclusion of two major sets of potential instrumental variables - "volume" and "attributes" - that are plausibly exogenous to each physician's underlying responsiveness to industry interactions, but are still likely to be inputs to the kind of targeting activities highlighted by Figure 3. Together, these variables form the basis of our approximation to the physician-targeting function.

We identify each physician's drug-class-specific patient volumes using the 2013-2014 Medicare Part D claims data. Unlike earlier years, this data includes the unique number of beneficiaries that each physician treats with a particular drug. With this count, we can construct, for example, the annual number of patients each physician treats with cardiovascular drugs. Using the full sample of Medicare physicians (including all specialties), we construct average volume metrics at two levels of ATC drug classes: Cardiovascular and Lipid-Modifiers. These two ATC drug classes correspond to where statins sit in the ATC hierarchy - statins are a subset of lipid-modifiers, which are a subset of cardiovascular drugs. We utilize these broader classes to minimize the extent to which the volume measures might have endogenously been affected by industry interactions. We contend that the average number of patients that a physician treats with cardiovascular drugs is driven by latent

characteristics of their local patient population. As evidence to the stability of these volume metrics, the year-to-year correlation of these metrics are all above 0.90.

The volume metrics are calculated at three levels of observation (Hospital Referral Region (HRR); Hospital; Individual) and separately for cardiologists and all other specialties. The HRR- and Hospital-level metrics are calculated using jack-knife procedures where each physician's hospital (per reported affiliation) are excluded from the HRR-level value, and each physician's own volume are excluded from the Hospital-level value. Thus, for example, an HRR-level metric for cardiologist i would indicate the average annual number of beneficiaries a physician in i's HRR, but not in i's hospital, treats with a particular class of drugs. This procedure is an effort to minimize the degree of collinearity across the set of metrics, and is intended to mimic a firm's allocation procedure whereby resources are targeted to regions, hospitals and physicians with larger patient pools at risk of using the firm's drug.

We supplement the HRR volume metrics with data from the Behavioral Risk Factor Surveillance System (BRFSS). Using the 2011 BRFSS we identify three additional HRR-specific variables: (1) the average rate of cardiovascular-related hospitalizations for Medicare beneficiaries, (2) the uninsurance rate, and (3) the percent of individuals enrolled in Medicaid. Together these variables likely capture first-order variation in disease prevalence, health insurance coverage and incomes.

In an attempt to introduce proxies for the marginal costs of these interactions, we identify a number of affiliation and density related metrics. Our intuition here is based on the likelihood of returns to scale - when there are many hospitals or physicians clustered geographically, fixed costs to the firm of setting up a meals program can be spread over more interactions. Per the hospital and organizational (e.g. practice) affiliations reported in the CMS Physician Compare data, we construct a set of "attribute" variables at the three levels of observation as follows: (1) HRR: number of hospitals, number of organizations, average number of hospital/organization affiliations per physician; average number of physicians per hospital/organization; (2) Hospital: number of physicians, number of organizations, average number of hospital/organization affiliations per physician; and (3) Individual: number of hospital/organization affiliations. Again, as in the case of the volume metrics, these metrics are calculated separately for cardiologists and all other specialties, and HRR- and Hospital-level averages are calculated using the jack-knife procedure.

Beginning with these raw volume and attribute variables, we introduce a number of transformations and interactions: (1) percentiles (National for HRRs; HRR for Hospitals; Hospital for individuals); (2) jack-knife counts of individuals above the national median; (3) squared terms; (4) volume-attribute interactions using the same level of observation (i.e. interacting all HRR-level volume metrics with all HRR-level attribute metrics). The final set

of potential instruments amounts to 382 variables (186 HRR, 136 Hospital, 60 Individual).

Obviously, there are a vast number of ways one could compress and/or transform this set of variables to implement as instruments. Instead of relying on any ad hoc protocols to select subsets or individual variables, we utilize LASSO regression. The procedure is as follows: (1) perform a LASSO regression using the full set of potential instruments to predict the likelihood that Pfizer (or, separately, Astrazeneca) gave a meal to the cardiologist in 2011; (2) use the subset of variables that the LASSO does not shrink to zero to estimate a linear probability model (LPM), again predicting the likelihood of a meal; and (3) generate the physician-specific (and time-invariant) linear prediction based on the LPM coefficient estimates. This identifies each physician's instrumental variable predicted probability of receiving a meal, or what we will term their "IV Index".

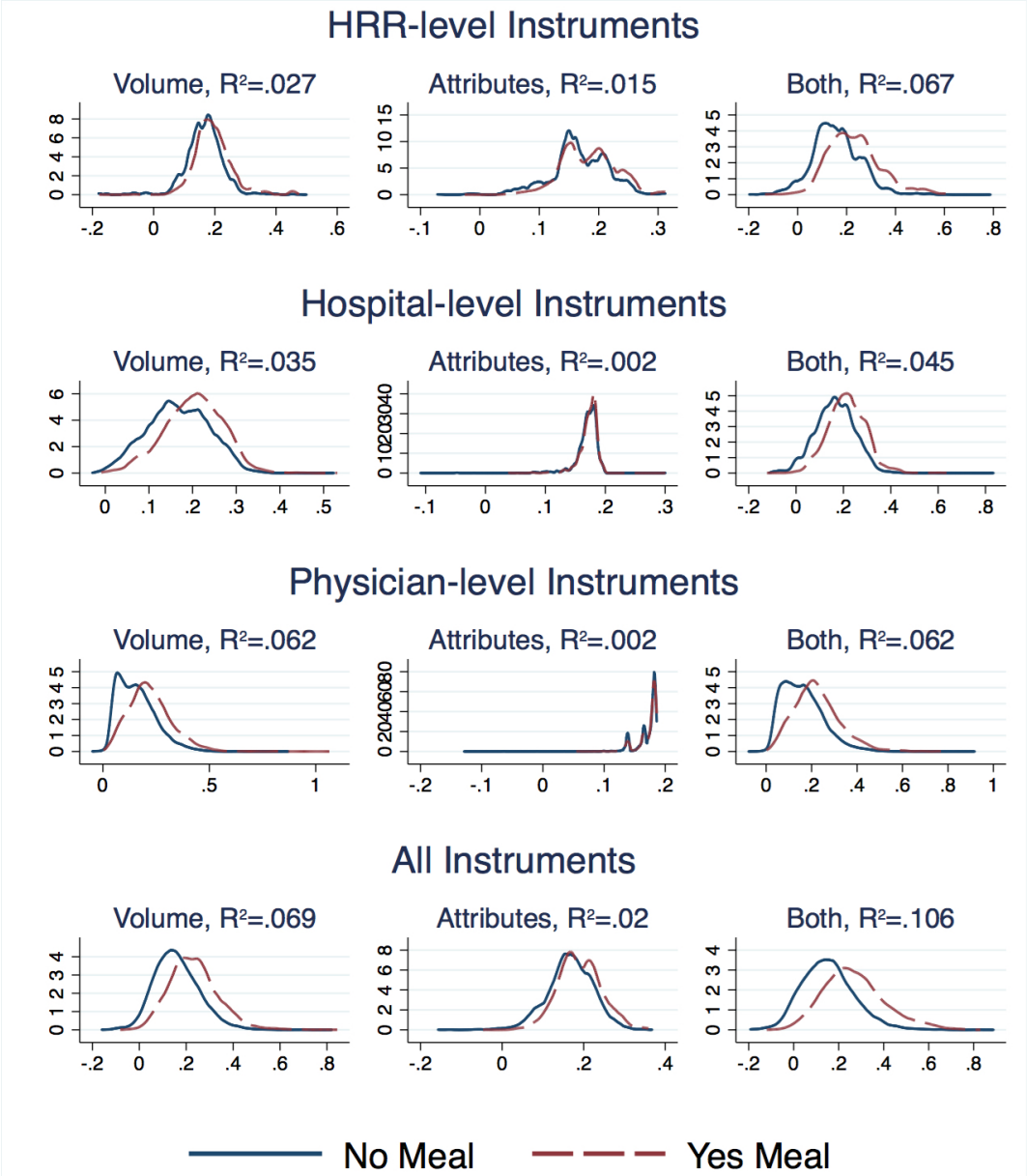
4.2 Instrument Selection Results

Figure 4 shows the performance of each instrument set at the different levels of observation by printing the R^2 from the LPMs above the distribution of predicted meals for physicians with and without realized meals. For reference, when using the full set of 384 instruments, the LASSO regression shrinks 196 variables to non-zero values.

Across the different instrument sets, the LASSO-selected variables can explain roughly 4-10% of the cross-sectional variation in meals. As a comparison point, regressions including HRR-fixed effects or Hospital-fixed effects have R^2 values of 0.09 and 0.20, respectively. Thus, it appears we are capturing important sources of variation in meal payments.

When including the full set of potential instruments the R^2 equals 0.106, which is greater than the sum R^2 values for the full set of volume metrics (0.069) and attributes (0.020) indicating that these two sets are in fact capturing separate sources of variation. However, there does appear to be some collinearity across the three levels of observations given the non-additivity of the R^2 values. For this reason, we will also report regressions where only the HRR-level instrument sets are utilized. Since the HRR set of instruments is purged of each physician's own values, a comparison of 2SLS estimates from all instruments versus the HRR set only can also shed light on the possibility of individual-level endogeneity. But as we will show, the coefficients identified using either set are very similar in magnitude.

Figure 4: Instrument Set Predictions, by Level of Variable



Note: Each plot displays the distribution of predicted meal probabilities (the IV Index) per LPM using the LASSO-selected subsets of each potential instrumental variable set. The R^2 are from the LPM.

5 Reduced Form Evidence on the Returns to Meals

Our statistical analyses in this section consist of regressions of the form

$$\text{Outcome}_{djt} = \text{Payment}_{df(j)t}\alpha + \text{Controls}_{djt}\beta + \epsilon_{djt} \tag{8}$$

where our focal outcomes (share-based measures of quantity) for a cardiologist d and molecule j in year t depend on whether or not the firm who manufactures the molecule $f(j)$ provides payment to the cardiologist (i.e. in the form of a meal), a set of controls (i.e. doctor- or year-fixed effects) and an i.i.d. error term (ϵ_{djt}). The focal parameter α describes the effect of industry-interaction on the physicians treatment decisions, where payment may equal a dummy variable indicating any interaction, or the dollar value of the reported interactions.

Because these interactions do not randomly occur, we are concerned that the receipt of a payment is correlated with the error term. Depending on the direction and magnitude of this correlation, simple OLS regressions of Equation 8 will over- or under-estimate the true causal payment treatment effect, which motivates are instrumental variable approach described previously.

Should we expect positive or negative selection, or in other words, do firms attempt to persuade physicians with inherently larger or smaller relative (share-based) utilization? Note that the degree of this selection will depend on the shape of what one might call the "persuasion function" as it behaves across the outcome distribution. If there are increasing costs to responding to meals and or physicians place a decreasing value on marginal meals, then this would imply decreasing returns to persuasion (the persuasion function is concave) and, in turn, negative selection. In short, because it is easier to influence physicians with inherently low utilization to begin with, firms will target these individuals.

We posit that a concave persuasion function is very likely in this setting. Physicians are constrained in the number of patients they treat and face both organizational and legal forces (e.g. electronic prescribing recommendations, threat of lawsuit) that impose limits on the degree to which they can influence their prescriptions. As initial evidence to this proposition, consider the distribution of cardiovascular claim shares for Lipitor and and Crestor in Table 1 - there are very few "high-share" cardiologists. The reported maximum of 1 (=100% of the focal drug) is driven entirely by low-volume prescribers. For cardiologists with more than the 25th percentile of cardiovascular-related claims each year ($\approx$ 800), no cardiologist prescribes more than 30% of their claims on the focal drug. If there were linear or increasing returns to persuasion, we would have expected to see larger shares across the full support of this distribution. In the empirical analyses below, we provide direct evidence for negative selection made possible with the instrumental variables.

The instrumental variables approach will amount to estimating the following first-stage equation

$$\text{Payment}_{df(j)t} = \text{Predicted Pr(Payment)}_{df(j)}\gamma + \text{Controls}_{djt}\delta + \varepsilon_{djt} \tag{9}$$

where Predicted $\text{Pr(Payment)}_{df(j)}$ is the linear prediction of a physician's probability of receiving a meal in 2011 per the instrumental variable selection procedure described in the

previous section.

Since Predicted Pr(Payment)$_{df(j)}$ does not vary over time within each cardiologist-firm pair, it can only be implemented in a cross-sectional manner. However, we can introduce within-physician variation to this instrument by interacting it with a dummy variable that equals one in 2012. This approach leverages the fact that the entry of generic atorvastatin in 2012 likely changed the incentive for each firm to interact with a cardiologist given the new statin choice set. When estimating these difference-in-difference models both physician- and year-fixed effects will be introduced as controls.

5.1 LATE Clarification

Equations 8 and 9 will be estimated via 2SLS and, given the exogeneity assumptions, will identify the LATE of receiving a meal from a firm on the share of claims a physician prescribes for the firm's focal drug. Which physicians are the compliers here, given the set of variables included in the instrument selection process? As discussed in Section 4, we interpret these variables as proxies for the firm's net value of interacting with each physician *without considering the weight that any particular physician places on the receipt of meals*. That is to say, we ask what is the causal effect of meals on physicians who look like they would be valuable to a firm?

In this vein, we interpret our LATEs as being in the same vein as the traditional notion of average treatment effects for the treated (ATET). In this particular setting, ATET estimates are very policy-relevant. Since these interactions have traditionally been unregulated, all policy discussions to date have been with respect to the impact of restricting or prohibiting such meals, which is precisely the counterfactual exercise we conduct in Section 6. Thus, while our estimates are less generalizable to the effects of *increased* physician-firm interactions, the particular LATE we do identify is much more informative for existing policy proposals.

The cross-sectional version of our I.V. approach will allow us to identify year-specific LATEs - which we would expect to differ in 2011 and 2012 given the entry of generic atorvastatin. However, this approach convolutes two versions of what might be considered the "meal-effect": (1) the "ever-meal" effect - the causal effect of *ever* receiving a meal from a firm, and (2) the "meal-this-year" effect - the causal effect of receiving a meal from a firm *in a particular year*.

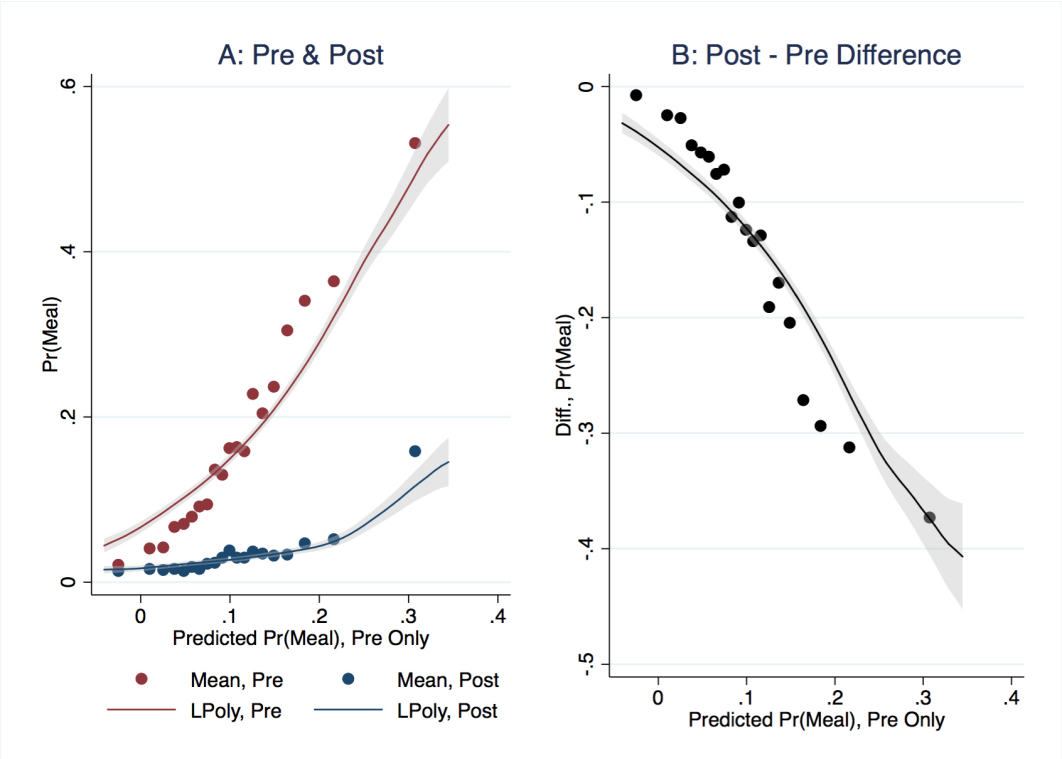
The panel version of our I.V. approach permits the inclusion of physician-fixed effects, which will remove the ever-meal effect, and all other stable attributes between each physician-firm pair. By exploring year-to-year variation in treatment and outcomes this estimate will

represent a weighted average of the meal-this-year effect across 2011 and 2012.

5.2 Main Reduced Form Results

Figure 5 makes clear the first-stage relationships for Pfizer's payment behavior in both cross-sectional and panel dimensions. There is a clear positive relationship between the predicted probabilities of meals and realized probability of meal receipt in both 2011 and 2012 (Panel A), and the magnitude of this relationship is significantly less positive in 2012 relative to 2011 (Panel B).

Figure 5: First Stage: Pfizer's Meal Payments



Note: Panel A displays the realized probability of receiving a meal from Pfizer in each year as a function of the predicted meal probabilities (the IV Index), which is generated using pre (2011) data only. Binned scatter plots are overlaid by kernel-weighted local polynomials. Panel B plots the difference in realized payment probabilities from 2011-2012 as a function of the IV Index.

Table 2 reproduces the Figure as regressions, including results for both Pfizer and Astrazeneca, displaying cross-sectional results in pre (2011) and post (2012) periods, as well as the difference-in-difference estimates that utilize both years and include both physician- and year-fixed effects. The predicted meal index is a significant predictor of meal receipt in the cross section for both firms. However, the difference-in-difference estimates indicate that

in 2012 while Pfizer reduced payments moreso for high-index cardiologists (driven mostly by an across the board decline of payments to near-zero), Astrazeneca increased both meal rates (Pr(Meal)) and meal sizes ($) for the cardiologists that our instruments identified as most at-risk of interaction.

Table 2: First Stage: Payments

	y =Any Meal			y =Meal $		
	Pre	Post	D-D	Pre	Post	D-D
Panel A: Pfizer						
IV Index	1.620***	0.380***		116.0***	22.07***	
	(0.0430)	(0.0300)		(11.78)	(3.346)	
Post × IV Index			-1.239***			-93.92***
			(0.0497)			(11.35)
N	16140	16140	32280	16140	16140	32280
adj. R^2	0.108	0.0260	0.220	0.0276	0.00958	0.142
F-stat	1422.0	161.1	620.7	97.00	43.52	68.52
avg(y)	0.173	0.0339	0.104	9.237	1.870	5.554
Panel B: Astrazeneca						
IV Index	0.982***	1.018***		59.71***	138.0***	
	(0.0146)	(0.0142)		(1.531)	(5.709)	
Post × IV Index			0.0351**			78.29***
			(0.0146)			(5.210)
N	16140	16140	32280	16140	16140	32280
adj. R^2	0.158	0.175	0.567	0.0606	0.0322	0.310
F-stat	4548.9	5124.3	5.781	1520.2	584.3	225.8
avg(y)	0.522	0.589	0.555	27.69	70.77	49.23

Note: Pre and Post subsets utilize cross-sectional variation only. D-D includes physician- and year-fixed effects and interacts the IV with a post-dummy. IV Index is the firm-specific linear prediction of meal receipt per the LASSO-selected instrumental variable set.

The reduced form regressions where the share outcomes are regressed directly on our instrument are presented in Table 3. In line with the cross-sectional results of Table 2, physicians with larger predicted probabilities of meal receipt commit a larger share of their claims to the firm's focal statin drug. The difference-in-difference estimates are more imprecise, statistically speaking.

Tables 4 and 5 present the naive results of estimating Equation 8 via OLS alongside the 2SLS estimates, separately for 2011 and 2012 using cross-sectional variation and for the full sample using the difference-in-difference approach. The tables present results for the focal statin's share of cardiovascular and statin drugs, respectively. Consider first the Pfizer-based "Pre-OLS" estimate in Table 4 that identifies the correlation between meal receipt and share of cardiovasculars the physician commits to Lipitor in 2011. The coefficient of 0.00824 indicates that physicians receiving a meal from Pfizer in 2011 had about a 20% larger share of their cardiovascular claims for Lipitor. In contrast, the IV estimate for

Table 3: First Stage: Share of Claims

	y =Q. Share, Year			y =Q. Share, Cardio.			y =Q. Share, Statins		
	Pre	Post	D-D	Pre	Post	D-D	Pre	Post	D-D
Panel A: Lipitor & Pfizer									
IV Index	0.0262***	0.0131***		0.0398***	0.0181***		0.0857***	0.0713***	
	(0.00157)	(0.000500)		(0.00198)	(0.000627)		(0.00907)	(0.00292)	
Post × IV Index			-0.0131***			-0.0216***			-0.0172**
			(0.00131)			(0.00166)			(0.00778)
N	14558	14558	29116	14538	14550	29062	14309	14425	28492
adj. R^2	0.0167	0.0402	0.539	0.0231	0.0491	0.525	0.00601	0.0358	0.613
F-stat	278.9	683.2	100.1	404.8	837.7	170.6	89.37	596.5	4.863
avg(y)	0.0321	0.00725	0.0197	0.0407	0.00918	0.0249	0.225	0.0465	0.136
Panel B: Crestor & Astrazeneca									
IV Index	0.00457***	0.00311**		0.00760***	0.00718***		0.0618***	0.0494***	
	(0.00136)	(0.00141)		(0.00161)	(0.00158)		(0.00633)	(0.00635)	
Post × IV Index			-0.00146			-0.000180			-0.0091**
			(0.00106)			(0.00126)			(0.00447)
N	16140	16140	32280	16093	16119	32156	15622	15737	30874
adj. R^2	0.00107	0.000471	0.678	0.00196	0.00202	0.716	0.00830	0.00573	0.753
F-stat	11.26	4.862	1.882	22.17	20.61	0.0205	95.36	60.68	7.156
avg(y)	0.0221	0.0228	0.0225	0.0278	0.0284	0.0280	0.146	0.147	0.147

Note: Pre and Post subsets utilize cross-sectional variation only. D-D includes physician- and year-fixed effects and interacts the IV with a post-dummy. IV Index is the firm-specific linear prediction of meal receipt per the LASSO-selected instrumental variable set.

this same sample identifies a treatment effect nearly 5 times larger, where a meal increases Lipitor's share of cardiovasculars by 95% (3.9 p.p.). Examining Astrazeneca in this same period, the OLS and IV estimates are very similar, implying that a meal increases Crestor's share of cardiovasculars by roughly 40% (1 p.p.).

A similar pattern emerges when examining only 2012, except the relative difference between the OLS and IV estimates is much more dramatic, especially for Pfizer and Astrazeneca where the difference between the coefficients is more than an order of magnitude. Although a dramatic result, this is in line with a strategy whereby Pfizer reduces Lipitor-related interactions across the board to cardiologists, except for only the most persuadable (i.e. cardiologists with a large θ^m). Under this logic, the difference in 2011 and 2012 estimates for Pfizer suggest an apparently large heterogeneity with respect to the meal treatment effect. In contrast, the Astrazeneca IV-identified meal treatment effect is similar in both time periods, indicating that the generic entry of atorvatistin did not significantly alter Astrazeneca's ability to influence physician's use of Crestor.

The difference-in-difference estimates for Pfizer and Lipitor also reveal a rather large difference between OLS and 2SLS estimates. The IV indicates that meal receipt causes a 100% increase in cardiovascular share (2.5 p.p.). That this is smaller than both the cross-

Table 4: OLS v 2SLS: Quantity Shares, Cardiovasculars

	Pre		Post		D-D	
	OLS	2SLS	OLS	2SLS	OLS	2SLS
Panel A: Lipitor & Pfizer						
Any Meal	0.00824***	0.0387***	0.00324***	0.108***	0.00360***	0.0252***
	(0.000691)	(0.00211)	(0.000507)	(0.0114)	(0.000589)	(0.00206)
N	16046	14538	16058	14550	32074	29062
$\frac{\%}{100}$ per Meal	0.204	0.952	0.357	11.79	0.145	1.011
F-stat		1558.9		104.4		922.3
Panel B: Crestor & Astrazeneca						
Any Meal	0.00579***	0.0116***	0.00387***	0.0123***	0.000174	0.00168
	(0.000456)	(0.00139)	(0.000454)	(0.00146)	(0.000319)	(0.00886)
N	16046	14538	16058	14550	32074	29062
$\frac{\%}{100}$ per Meal	0.210	0.420	0.138	0.436	0.00627	0.0602
F-stat		3300.9		2459.9		28.84

Note: Dependent variable is the share of the physicians annual claims for all cardiovascular drugs attributed to the firm's focal statin. OLS estimates are per Equation 8, 2SLS are per jointly estimating Eqs. 8 and 9 using the IV Index directly (Pre and Post) or the IV Index interacted with a dummy variable equaling one in the Post period (D-D). Pre and Post subsets utilize cross-sectional variation only. D-D includes physician- and year-fixed effects. Standard errors are robust and clustered at the physician-level; $^{*}p < 0.1$,** $p < 0.05$,*** $p < 0.01$.

Table 5: OLS v 2SLS: Quantity Shares, Statins

	Pre		Post		D-D	
	OLS	2SLS	OLS	2SLS	OLS	2SLS
Panel A: Lipitor & Pfizer						
Any Meal	0.0299***	0.0830***	0.0135***	0.423***	0.0109***	0.0199**
	(0.00263)	(0.00888)	(0.00208)	(0.0453)	(0.00219)	(0.00900)
N	15789	14309	15918	14425	31434	28492
$\frac{\%}{100}$ per Meal	0.134	0.369	0.293	9.079	0.0809	0.146
F-stat		1543.3		104.1		902.7
Panel B: Crestor & Astrazeneca						
Any Meal	0.0325***	0.0608***	0.0245***	0.0581***	0.00128	0.0918*
	(0.00197)	(0.00625)	(0.00188)	(0.00632)	(0.00139)	(0.0484)
N	15789	14309	15918	14425	31434	28492
$\frac{\%}{100}$ per Meal	0.220	0.410	0.167	0.394	0.00866	0.621
F-stat		3158.0		2425.5		26.55

Note: Dependent variable is the share of the physicians annual claims for all statins attributed to the firm's focal statin. OLS estimates are per Equation 8, 2SLS are per jointly estimating Eqs. 8 and 9 using the IV Index directly (Pre and Post) or the IV Index interacted with a dummy variable equaling one in the Post period (D-D). Pre and Post subsets utilize cross-sectional variation only. D-D includes physician- and year-fixed effects. Standard errors are robust and clustered at the physician-level; $^{*}p < 0.1$,** $p < 0.05$,*** $p < 0.01$.

section IV estimates suggests that the "meal-this-year" effect cannot explain the full effect observed in the cross-section. To further explore this temporal dimension of the meal effect, we utilize additional data on Pfizer payments to these physicians in 2010, and separately estimate the difference-in-difference 2SLS model for those cardiologists paid or not paid in 2010. These regressions (not shown) reveal a relative meal treatment effect on Lipitor's share of cardiovasculars of 43% and 215%, for those that did and did not get paid in 2010, respectively. That we observe larger relative effects for "new" meal recipients is suggestive of decreasing returns to meals over time; however, our brief panel of data prohibits us from examining these effects in more detail. Thus, we treat all interactions as static and leave future research to explore these dynamics of physician-firm interactions.

Overall, comparing Tables 4 and 5 can reveal whether the firm's meals have any differential effect on physician's decisions when considering all cardiovascular drugs versus just statins. In the case of Lipitor, meals from Pfizer appear to be more effective inducing Lipitor prescriptions in the place of other non-statins relative to statins given the relatively larger effects of meals on cardiovascular shares. In contrast, Astrazeneca's margin of influence for Crestor appears roughly equivalent at both levels (except for the noisy estimates identified in the difference-in-difference models).

5.3 I.V. Robustness & Selection

The sizable differences in the magnitudes of the OLS and 2SLS coefficients is in line with a strong negative selection strategy by these firms. However, it also could be explained by our instrumental variables be weak or endogenous and positively correlated with physicians meal-responsiveness. To the first critique, the F-statistics in all 2SLS models are well above conventional standards for I.V. strength, and all models reject the null of under-identification tests. To the second point, we generate an IV Index that utilizes only the HRR-level variables and re-esitmate the 2SLS models with this predicted meal probability. The intuition here is that if our set of potential instruments is in fact endogenous (which would invalidate our approach), then the variables most likely to be significantly and positively correlated with each physician's responsiveness to meals are the physician-level variables. If this in fact the case, then utilizing only the HRR-level variables (which do not include any physician-specific data) should identify smaller point estimates. However, these models (not shown) all reveal LATE magnitudes that are very similar to the magnitudes identified with the full set of instruments.[14]

[14]For Pfizer and Lipitor, the "Any Meal" treatment effect for cardiovascular shares is 0.032, 0.124, and 0.021 (all significant at $p < 0.01$) for the pre-only, post-only and difference-in-difference estimates, respectively. Compare this to the results in Table 4 which are 0.039, 0.108, and 0.025 for the same models,

Is there any statistical evidence for the negative selection story then? First, examine the Pfizer Panel of Table 4, where from 2011 to 2012, the OLS point estimates decrease while the 2SLS estimates increase. This is directly in line with a strategy where, amidst the large generic entry, Pfizer scaled back their meals beginning with the least responsive cardiologists (leading to a larger 2SLS point estimate), which in a world of negative selection would target payments to those with lower shares (leading to a smaller OLS point estimate). To examine this more directly, Table 6 presents a variety of two-stage estimates of Equations 8 and 9 using both linear and non-linear models. By separately estimating the two equations we can directly identify the correlation between the two error terms. Across all specifications, there is evidence of a strong negative correlation between the payment and prescribing error terms - firms target meals to physicians with otherwise low levels of their utilization of their drugs.[15] The significance and relatively large negative magnitudes of this correlation suggest that the large differences in OLS and 2SLS results may be due in large part to firms' strategies. This negative selection is even apparent within the difference-in-difference models, which include physician-fixed effects, indicating that this negative selection occurs not only across physicians, but also within-physicians over time.

Table 6: Two-Stage Models: Testing for Error Correlation & Selection

	Pre			Post			Both
	2SLS	IV-Tobit	Bi-Probit	2SLS	IV-Tobit	Bi-Probit	2SLS
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Panel A: Lipitor & Pfizer							
Any Meal	0.0387***	0.0504***	1.830***	0.108***	0.305***	2.149***	0.0252***
	(0.0040)	(0.0049)	(0.0360)	(0.0182)	(0.0114)	(0.0417)	(0.0037)
$\text{corr}(\epsilon^{\text{Meal}}, \epsilon^{\text{Share}})$	-0.193***	-0.318***	-0.928***	-0.857***	-0.946***	-0.967***	-0.104***
Panel B: Crestor & Astrazeneca							
Any Meal	0.0116***	0.0351***	1.699***	0.0123***	0.0326***	1.731***	0.00168
	(0.00244)	(0.00351)	(0.0273)	(0.00253)	(0.00330)	(0.0286)	(0.0088)
$\text{corr}(\epsilon^{\text{Meal}}, \epsilon^{\text{Share}})$	-0.053***	-0.285***	-0.869***	-0.098***	-0.326***	-0.908***	-0.027***
N	16093	16093	16093	16119	16119	16119	32212
IV	Index	Index	Index	Index	Index	Index	Index×Post
Year+Phys. F.E.							Y

Note: The dependent variable in all models is the focal statin's cardiovascular share (as in Table 4). $\text{corr}(\epsilon^{\text{Meal}}, \epsilon^{\text{Share}})$ is the correlation in error terms across the payment equation (Eq. 9; ϵ^{Meal}) and the prescription equation (Eq. 8; ϵ^{Share}). The IV-Tobit models assume that the dependent variable is censored at 0 and estimates the two-stage model via maximum likelihood; the Bi-Probit model jointly estimates both the payment and prescription equations as probit regressions. Standard errors are robust and clustered at the physician-level; $^{*}p < 0.1, ^{**}p < 0.05, ^{***}p < 0.01$.

respectively.

[15] Furthermore, the non-linear estimates provide evidence that our linear formulation of the payment and prescribing functions is not dramatically biasing our estimates in any way.

5.4 Extensive or Intensive Margin: Does the dollar value matter?

Because we can observe the dollar value associated with these meals, we can examine the extent to which these effects are driven differentially by the extensive or intensive margins of the interactions. Focusing just on Pfizer and Lipitor, Table 7 estimates the difference-in-difference specification using Lipitor's share of cardiovascular drugs, and presents coefficients when restricting the sample to different maximum payment amounts: \$25, \$60 and \$185, which correspond roughly to the 25th, 50th and 75th percentile of the non-zero meal value distribution.

Table 7: 2SLS Suggestive Non-Linearities: Quantity Shares, Cardiovasculars, Lipitor (Pfizer)

	(1) Pay < \$25	(2) Pay < \$60	(3) Pay < \$185	(4) Full Samp.
Panel A: Any Meal				
Any Meal	0.0407***	0.0283***	0.0252***	0.0252***
	(0.00393)	(0.00247)	(0.00210)	(0.00206)
N	26224	27662	28780	29062
$\frac{\%}{100}$ per Meal	1.672	1.150	1.013	1.011
$\frac{\%}{100}$ per \$	0.117	0.0530	0.0268	0.0199
F-stat	409.5	732.8	893.1	922.3
Panel B: Meal \$ Amt.				
Meal \$	0.00282***	0.00110***	0.000599***	0.000406***
	(0.000275)	(0.0000987)	(0.0000548)	(0.0000395)
N	26224	27662	28780	29062
$\frac{\%}{100}$ per \$	0.116	0.0446	0.0241	0.0163
F-stat	375.2	566.4	349.0	238.6

Note: All specifications estimate the difference-in-difference 2SLS model for Lipitor's share of cardiovascular drugs per Pfizer meal payments. The three censoring points correspond roughly to the 25th (\$25), 50th (\$60) and 75th (\$185) percentiles of the non-zero meal value distribution. Standard errors are robust and clustered at the physician-level; $^{*}p < 0.1$,** $p < 0.05$,*** $p < 0.01$.

Clearly, the extensive margin effect of receiving any meal leads to a larger absolute and relative increase in the share of cardiovasculars for Lipitor. As larger and larger meal values are included in the sample, the apparent returns to the marginal dollar decrease by upwards of 80%. Thus, it appears that the vast majority of 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 \$200. 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 observation of strong negative selection, this apparently large role of the extensive margin effect has important implications for the policy discussions surrounding physician-firm interactions. For example, much of the popular press coverage and political discourse to date is instigated with stories of physicians receiving exorbitant

payment amounts or prescribing extremely high rates of a firm's products. However, these results suggest that is quite the opposite. Firms seem to have the largest influence over what otherwise would have been low-volume prescribers, and this influence is largely driven by simply receiving a low-valued meal.

Why then do we not observe all physicians receiving these low-valued meals? Based on all of our reduced form evidence, it appears that this is likely because it is increasingly difficult to influence treatment decisions beyond a certain point across and within physicians - there are only so many physicians who are likely to be responsive to a meal, and amongst these physicians there are decreasing returns to interacting with them.

6 Welfare Analysis Using Supply and Demand Model

6.1 Identification and estimation

We follow the procedure in Berry (1994), setting choice probabilities implied by the demand model equal to market shares observed in the data, and inverting the system to yield a linear correspondence between a function of market shares and the mean utility for each product $\ln(s_{jbdt}/s_{0dt}) - \lambda \ln(s_{jbdt}/(1-s_{0dt})) = \delta_{jbdt}$, leading to the linear estimation problem:

$$\ln(s_{jbdt}/s_{0dt}) = \lambda \ln(s_{jbdt}/(1-s_{0dt})) + \theta_{jd} + \theta^m 1_{\{m_{jdt}>0\}} - \theta^p p_{jbt} + \theta_t + \xi_{jbdt}. \tag{10}$$

Estimating this equation faces two well-known challenges in that theory suggests $\ln(s_{jbdt}/(1-s_{0dt}))$, p_{jbdt}, and $1_{\{m_{jdt}>0\}}$ are all correlated with the unobservable term ξ_{jbdt}. We take an instrumental variables approach to solving this identification problem. For $\ln(s_{jbdt}/(1-s_{0dt}))$, we follow much of the literature (e.g., Berry and Waldfogel (1999); Sinkinson and Starc (2017)) in using the size of the set of products prescribed $\ln(|\mathcal{J}_{dt}|)$ as an instrument, which leverages the fact that more variety will on average affect substitution independent of the individual product's unobservable.

To find a source of variation in price p_{jbt} that is uncorrelated with unobservables ξ_{jbdt}, notice that with our provider-molecule fixed effects, the remaining variation in price is over time for the same molecule. This price variation is driven by the exogenous event of the Lipitor patent expiration. Thus we argue that our granular fixed effects plus the timing of our data surrounding this event provides exogenous variation in price to identify the slope of the demand curve.

Finally, our instrumental variables strategy for meals $1_{\{m_{jdt}>0\}}$ has been outlined in detail previously in our reduced form analyses. The explicit focus on the choice between substitutes in our discrete choice demand model here reveals a potential weakness in the simplest form

of the IV that uses only provider, practice, and HRR volume characteristics related to the statin market—these variables do not vary across molecules within the statin class. This can explain why some providers receive meals and others do not, but it cannot explain why some providers receive meals from Pfizer while others receive meals from AstraZeneca. For that reason we exploit the fact that Pfizer and AstraZeneca seem to follow different strategies—AstraZeneca provides more meals—and we flexibly allow for different first stage models to capture this fact and thus separately identify meal effects across these two firms.

In our preferred implementation, we jointly estimate the above (linearized) demand model with the supply model using a generalized method of moments approach. This enables us to simultaneously recover the demand parameters θ, bargaining parameters, and marginal costs. It also imposes the constraints of the supply model that $mc_{jbt} \in [0, p_{jbt}]$, and $\frac{\partial s_{jbt}}{\partial p_{jbt}} \frac{p_{jbt} - mc_j}{s_{jbt}} \in [-1, 0]$.

6.1.1 Parameter Estimation Results

Table 8 shows the parameter estimates for the full model described above, and for several intermediate models that help to illustrate the importance of the modeling choices and validity of the identification strategies. Column (1) estimates the model, but without molecule-provider fixed effects and without instruments for meals. The parameters on price and meals are very small in magnitude. The nesting parameter λ is significant, indicating that there is more substitution among statins than between statins and the outside good. Column (2) adds molecule-provider fixed effects, and all parameters change–the within variation is a more credible source of variation for all of our parameters. In particular, though, the price parameter becomes negative and economically significant, as it is now being identified by the demand response to the price drop for atorvastatin upon generic entry. Column (3) is our full model, using the volume targeting instruments for meals, which results in an order of magnitude increase in the impact of meals on prescribing. Column (4) estimates the same model, but imposes the supply side constraints as well, which has a minimal effect.

Table 8: Demand Parameter Estimates

	(1)	(2)	(3)	(4)
θ^m	0.0003	0.039*	0.413*	0.413
θ^p	0.0004*	-0.0032*	-0.0034*	-0.0035
λ	0.83*	0.49*	0.50*	0.49
FE	t	t , jd	t , jd	t , jd
IV	$\ln(\frac{s_{jbdt}}{(1-s_{0dt})})$	$\ln(\frac{s_{jbdt}}{(1-s_{0dt})})$	$\ln(\frac{s_{jbdt}}{(1-s_{0dt})})$, $1_{\{m_{jdt}>0\}}$	$\ln(\frac{s_{jbdt}}{(1-s_{0dt})})$, $1_{\{m_{jdt}>0\}}$
Supply	-	-	-	Y

While the utility function parameters are difficult to interpret directly in terms of usage

impact, the relative size of the meal and price coefficients suggest that a meal has an equivalent impact to a $118 decrease ($= \theta^m/\theta^p$) in out-of-pocket price. While this seems like a large effect, it is partially driven by the lack of price sensitivity. Perhaps more enlightening is the implied semi-elasticity $\frac{\partial s}{\partial m}\frac{1}{s}$, which measures the percent change in market share of the focal drug associated with a meal. The average of 68 percent suggests this payment effect is large indeed.

These large causal effects are interesting in an of themselves, but by themselves they cannot answer the policy question of the effect of payments on pharmaceutical markets. By construction, they measure the effect "holding all else equal", but both the focal firm and other firms in the market have other strategic variables that may adjust to any policy change, and with the oligopoly structure of the market, these strategic reactions will depend on one another in equilibrium.

6.2 Assessing the Welfare Effects of Industry Payments to Physicians

To better understand the effects of payments to physicians on market welfare, we consider three counterfactual scenarios, all banning meals/payments from pharmaceutical firms to physicians. The first scenario bans payments, but fixes prices at those observed in the data. The goal here is to isolate the direct effect of payments on the market. The second scenario bans payments and allows prices to adjust to a new equilibrium, allowing for the fact that payments may affect prices as well as quantities. The third and final scenario bans payments and fixes out-of-pocket prices for all statins to marginal cost—an efficient allocation benchmark. In each scenario, we compute equilibrium quantities and prices (except where prices are fixed as just mentioned), use these to calculate producer surplus, and then compute consumer surplus implied by the utility model of demand. Table 9 displays various components of welfare under the observed data and counterfactual regimes. Each is shown in 2011 and 2012 separately in order to show how the results depend on market structure.

Focusing first on quantities, the primary result is that while payments do increase prescribing, on average they do so in a way that offsets the underprovision of statins due to market power keeping prices above marginal cost. Whether quantity with payments under- or over-shoots the efficient allocation varies between 2011 and 2012, and relatedly, between Lipitor and Crestor. The model estimates that Lipitor is under-utilized in 2011 on the order of 1.36 million prescriptions with payments banned vs. 2.21 million at the efficient allocation, and the observed payments raise Lipitor to 1.52 million, only partially closing the gap. Even after the sharp price drop upon generic introduction in 2012, the model estimates that

Table 9: Welfare Estimates

	2011				2012			
	Obs	$m=0$ p^{Obs}_{oop} p^{Obs}_{pos}	$m=0$ p^{*}_{oop} p^{*}_{pos}	$m=0$ p^{mc}_{oop} p^{*}_{pos}	Obs	$m=0$ p^{Obs}_{oop} p^{Obs}_{pos}	$m=0$ p^{*}_{oop} p^{*}_{pos}	$m=0$ p^{mc}_{oop} p^{*}_{pos}
$Q_{statins}$ (millions)	6.66	6.38	6.39	6.95	7.45	7.06	7.06	7.35
$Q_{atorvastatin}$	1.52	1.36	1.36	2.21	2.35	2.14	2.14	2.43
$Q_{Crestor}$	1.08	0.73	0.73	0.82	1.18	0.73	0.73	0.88
$\bar{p}_{statins}$ ($, OOP)	29	26	26	0	16	14	14	0
$\bar{p}_{atorvastatin}$	94	94	93	0	29	29	29	0
$\bar{p}_{Crestor}$	37	37	37	0	37	37	36	0
PS_{retail} ($ millions)	191.6	163.8	163.2	0	121.2	98.7	98.2	0
PS_{bias}	-0.5				-0.6			
$PS_{atorvastatin}$	142.2	127.0	126.7	0	68.2	62.0	61.9	0
$PS_{Crestor}$	39.7	27.0	26.6	0	43.6	27.2	26.7	0
CS_{retail} ($ millions)	2,143.1	2,043.4	2,044.4	2,245.7	2,405.3	2,266.9	2,267.5	2,372.3
CS_{bias}	-135.8				-186.5			
TS ($ millions)	2,198.3	2,207.2	2,207.5	2,245.7	2,339.3	2,365.5	2,365.7	2,372.3
$PS_{transfers}$ ($ millions)	434.4	350.6	348.3	1,426.1	459.9	362.0	359.6	638.9

atorvastatin is still underprovided at 2.14 vs. 2.43 million. The spillovers from prior Lipitor payments raise prescribing to 2.35 million, which almost closes the gap.

By contrast, the model predicts much smaller shortfalls for Crestor of 0.73M in the no payments vs 0.82M in the efficient scenario. Combined with the fact that Crestor provides many payments, this results in Crestor quantity under the observed payments of 1.08M exceeding the efficient benchmark allocation. On net, all payments results in underprovision of statins in 2011, but overprovision in 2012 once the price of atorvastatin has declined.

These quantity effects highlight several of the issues motivated by the theory in Section 3 and Inderst and Ottaviani (2012). The extent to which payments distort efficient allocation depends upon their scale relative to that of the distortion due to market power maintaining high prices. In the market studied here, payments move closer to the efficient allocation, though they overshoot in 2012. However, translating these quantity effects into surplus measures requires further analysis, as it depend on the extent to which they better align consumption with true the true quality/cost tradeoffs of the various drugs in the market and vs. the outside option.

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 $\frac{1}{\theta^p}\sum_{jbd} q_{jbd} - \theta^m 1\{m_{jbd} > 0\}$, which is calculated as CS_{bias} in the Table. This can be offset to the extent that payments steer patients towards better treatments—in particular since two firms have patented drugs in 2011, payments may better align their market shares with their qualities—but the calculations of CS_{retail} show this is not the case here. Banning

payments results in an increase of $37.1M (1.8 percent) in consumer surplus.

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 will increase producer surplus in an efficient manner. However, because this is an oligopoly, some payments result in inefficient business stealing, which harms consumer surplus with no offsetting producer surplus gain. Here we see that this business stealing effect is sufficiently large that consumer surplus losses outweigh producer gains, resulting in payments being inefficient in terms of total surplus, in spite of moving closer to the aggregate efficient allocation on the extensive margin. Banning payments results in an increase of $8.9M (0.4 percent) in total surplus in the retail market for statins.

While the above effects in the retail market all hint towards a value in banning payments, they leave out at least two important features of these payments. The first is the valuable information they may provide, which has been assumed to be zero due to the late stage of the statin market, but could be large in other contexts. The second is their effect on 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 all insured (not just those taking statins at a point in time), and so it is not easily translatable into a per person effect on premiums. With that caveat, however, the calculations under $PS_{transfers}$ suggest that these drug cost effects are even larger in magnitude than the retail effects. Because payments steer patients toward more expensive drugs, they increase spending on statins by $86.1M (19.8 percent) relative to our counterfactual where payments are banned.

7 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. While recent theoretical work (Inderst and Ottaviani 2012) has shed new light on these tradeoffs, it has remained challenging to identify these relationships empirically. Specifically, it has been difficult to identify the causal impact of payments to experts on the advice they provide to end consumers. Payments are not random and likely correlated with characteristics of the expert. This gap in the literature is particularly important, given recent debates over conflicts of interest and disclosure in the U.S. health care and financial services industries.

We address this gap by proposing a theoretical model which indicates a potentially useful

instrumental variable to overcome the challenges of empirically estimating these effects in the health care industry. Using measures of the potential volume of patients for physicians and their local colleagues, we introduce plausibly exogenous variation in which physicians receive payments from pharmaceutical companies. We also exploit variation across physicians over time to payments using the expirations of patents on key molecules. Leveraging this approach with detailed data on prescriptions, prices, and payments we are able identify the impact of these interactions on physician behavior and overall welfare.

Overall, we generally find our IV estimates to be larger than our naŢve OLS estimates for both 2011 and 2012, though patent expirations influence between firm differences in the latter year. For example, our results indicate that physicians receiving a meal from PÞzer in 2011 had about a 20 percent larger share of their cardiovascular claims for Lipitor. But the IV estimate is almost 5 times larger. The results of our differences-in-differences analysis are similar. In sum, these results are consistent with firm-physician interactions being driven by negative selection, whereby payments target physicians who would otherwise have prescribed the focal drug with low probability.

These large average effects appear to be highly nonlinear within and across physicians. Larger or more frequent payments make little difference compared to the event of having any payment at all. And the marginal physicians who receive their first payments later show smaller response in prescribing patterns.

Despite these substantial effects on prescribing of the drugs that receive payments, our counterfactual welfare analysis of banning payments indicates that such a ban would have only a small, though positive, effect on consumer and total surplus. This is because high prices due to market power keep statin consumption–overall and off the powerful branded molecules– inefficiently low, and increased consumption due to payments partially offsets this, bringing the market closer to the efficient allocation, but at the cost of preserving higher prices. We estimate the net effect of these two forces on consumers to be small but in favor of a ban. The overall increase in producer surplus is not large enough to offset this due to the fact that some of the increase is business stealing from cheap generic alternatives, and that producer surplus in general is estimated to be a relatively small portion of welfare.

There are limitations in our approach. We focus on a narrow 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 the statin market in an earlier phase, 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, which is quite general, or by providing alternative approaches to identify causal effects and model market

responses.

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Appendix: Payment Data Construction

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,[16] categorization approach,[17] 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[18] collected these reports as a part of their initiative to analyze physician-firm interactions. 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 10 summarizes interactions levels for all of the firms, active physicians[19] and years of data we observe. In the focal analysis, we utilize only payments from Pfizer (who owns Lipitor) and Astrazeneca (who 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.[20] To identify those firm-years most 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

[16] For 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.)

[17] Some 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).

[18] See: www.kyruus.com.

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

[20] For anecdotes related to the inaccuracies of these early reports see: https://goo.gl/jDyHyS.

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 the result of misreporting and not any dramatic change in firm policies.

Table 10: 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 ever.