Essays in Industrial Organization and Health Economics:

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ESSAYS IN INDUSTRIAL ORGANIZATION AND HEALTH ECONOMICS

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ESSAYS IN INDUSTRIAL ORGANIZATION AND HEALTH ECONOMICS

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Abstract

The unifying theme of this dissertation is the growing importance of pharmaceutical products in health care and in society more broadly. The first two chapters use structural and reduced-form models to study the effects of various policies on the choice and utilization of prescription drugs. The third chapter surveys the empirical literature on the competitive effects of a class of pricing arrangements used in the pharmaceutical and many other industries.

Chapter 1. One of the criticisms leveled against direct-to-consumer advertising of prescription drugs is that it overemphasizes the use of pharmaceuticals at the expense of other forms of treatment. In "Choice of Depression Treatment: Advertising Spillovers in a Model with Complementarity," I study how antidepressant TV ads affect demand for psychotherapy. Antidepressant advertising can increase demand for therapy if the products are complements or if advertising has spillover effects. To disentangle the different channels, I develop a discrete-choice demand model that

allows for complementarity between products, advertising spillovers, and flexible unobserved preference heterogeneity. Individual-level panel data on treatment choices and price variation allow me to separately identify complementarity and correlated preferences, whereas the average price of TV advertising, used as an instrument, identifies the causal effect of antidepressant ads on demand for each product. The results indicate that even though antidepressants and psychotherapy are substitutes, drug advertising increases demand for therapy through a spillover effect. Allowing for time-invariant and time-varying unobservables that can be correlated across products critically affects the estimated degree of complementarity and advertising elasticities.

Chapter 2. While prescription drugs have enabled the cost-effective treatment of a myriad of diseases, many pharmaceuticals come with potential for abuse. The growing use of opioid medications for chronic pain led to widespread misuse, addiction, and skyrocketing overdose death rates. In "Did Plain-Vanilla Prescription Drug Monitoring Programs Reduce Opioid Use? Evidence from Privately Insured Patients," I explore whether prescription drug monitoring programs (PDMPs) with no registration or use mandates were effective in reducing the utilization of opioid prescription drugs. Exploiting the staggered introduction of such programs between 2008 and 2010, I use difference-in-differences to estimate their causal effect on the number of prescriptions, days supply, and dosage per capita. Based on data from privately insured adults, the estimation results reveal that PDMPs successfully reduced opioid utilization, especially of high-dosage prescriptions. A battery of robustness checks suggests that the estimated effects are caused by the PDMPs and not by confounding factors such as broader trends in health care, attrition, out-of-state purchases, or

other anti-opioid policies.

Chapter 3. The assumption that buyers pay the same price for each unit of the good they purchase underlies many economic analyses. However, linear pricing is one of many pricing arrangements used in practice. In "Empirical Evidence on Conditional Pricing Practices: A Review," Julie Holland Mortimer and I review the existing empirical studies on the competitive impact of conditional pricing practices (CPPs), under which the price of a product may depend on a quantity, share, bundling, or other requirement. Examples of CPPs include all-units and loyalty discounts, full-line forcing contracts, and exclusivity arrangements. A common thread unifying the empirical literature is that CPPs often have both procompetitive and anticompetitive effects and that their net effect may depend on the details of the arrangements and the characteristics of the markets in which they are used.

Acknowledgements

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With an eye towards and hope for the future, I thank my future colleagues for keeping the excitement of being an economist alive.

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Chapter 1

Choice of Depression Treatment: Advertising Spillovers in a Model with Complementarity *

1.1 Introduction

Direct-to-consumer (DTC) advertising of prescription drugs on television has been controversial since its deregulation in 1997. One of the various criticisms leveled against this type of promotion is that it overemphasizes the use of pharmaceutical products at the expense of alternative treatment options.[1] In the context of depression, psychologists have expressed concern over the declining use of psychotherapy in

*Truven Health Analytics, through its Dissertation Support Program, generously made available for the purposes of this dissertation the Truven Health MarketScan® Commercial Claims and Encounters Database. Copyright © 2016 Truven. All Rights Reserved. Truven Health was not involved in preparing the results in this paper in any way. I am solely responsible for any errors.

[1]Ventola (2011) outlines the arguments for and against DTC advertising of prescription drugs.

depression treatment and pointed to antidepressant DTC ads as one of the reasons.[2]

I study how antidepressant TV advertising affects demand for psychotherapy. There are two possible channels: complementarity or substitutability between treatment options and spillover or business-stealing effects of advertising. If drugs and therapy are complements because they work better together, an increase in drug ads will increase demand for therapy. However, it is also possible that antidepressant advertising has a spillover effect by encouraging individuals to seek treatment, which can boost demand for therapy even if the products are substitutes. To disentangle the channels through which advertising operates, I develop a discrete-choice model that captures the key features of demand: complementarity, spillovers, and unobserved heterogeneity in preferences.

The model presents two identification challenges: separating complementarity from unobserved correlated preferences and identifying advertising spillovers. The first challenge arises from the fact that a relatively large share of patients may choose to take drugs and therapy either because they like both options for reasons unrelated to how well the two work together (unobserved correlated preferences) or because the products provide a greater benefit when taken in combination (complementarity). In addition, time-varying health shocks that boost demand for all treatment options can also make it seem that patients tend to choose drugs and therapy together relatively more often and falsely suggest that they are complements.

To separately identify complementarity from unobserved correlated preferences, I use two sources of identification. The first is individual-level panel data. The

[2]Nordbal (2010) discusses various possible causes of this trend.

intuition is that if drugs and therapy are complements, individuals will buy them in combination relatively more often than either one alone. I combine this with health plan level variation in prices. If demand for drugs and demand for therapy move in the same direction in response to an exogenous change in the price of either one, the products are complements; if they move in opposite directions, they are substitutes.

Identifying advertising spillovers typically requires separating own and cross effects when two products are advertising at the same time: if demand for both products increases, this can be rationalized by strong effects of own advertising and weak business-stealing of rival advertising or weak own effects and weak spillovers of rival advertising. This necessitates having two instruments to separately identify own and cross effects.[3] In the case of depression treatment, however, therapists are typically sole proprietorships that do little mass advertising. Thus, the only products that are advertised on TV are antidepressants, which significantly simplifies the problem. To alleviate concerns about the endogeneity of drug advertising, I use a cost-based instrumental variable.

I use two main sources of data. Individual-level panel data of treatment choices, prices, and patient demographics come from the Truven Health Marketscan® Commercial Claims and Encounters Database for the period 2008–2010. The Kantar Media Ad$pender database provides monthly advertising expenditures and counts of national and local TV ads for antidepressants.

[3]In general, one needs at least as many instruments as endogenous variables. I consider the effect of aggregate branded antidepressant advertising on demand for generic drugs, branded drugs, and therapy, and need a single instrumental variable. With more instruments, it is possible to study own and cross effects for each product. Shapiro (2018) and Sinkinson and Starc (2019) are two papers that successfully estimate business-stealing and spillover effects at the product level.

My model allows for complementarity as well as time-invariant and time-varying unobserved preferences that can be correlated across products. It extends Gentzkow (2007) by allowing for multiple markets and time periods and accommodating estimation of own and spillover advertising effects using linear instrumental variable methods. To estimate the model, I follow an approach that combines individual and market-level data similar to the one in Berry et al. (2004) (micro-BLP) and Goolsbee and Petrin (2004).[4] Estimation proceeds in two stages. The first estimates the parameters on individual-specific observables and the distributions of the unobservables using maximum simulated likelihood (MSL) while "concentrating out" bundle-market-time fixed effects.[5] I assume that the time-varying health shocks follow a Markov chain and use a technique from the literature on Hidden Markov Models (HMMs) to significantly simplify the calculation of the likelihood. I then use simulation to approximate integrals over the time-invariant unobserved preferences. Because advertising effects are not separately identified from the bundle-market-time fixed effects, the second stage of the estimation projects the recovered fixed effects on ads and market and time effects. To estimate the causal effect of advertising on demand for generic drugs, branded drugs, and psychotherapy, I use the average price of a 30-second TV ad spot, adjusted for ratings, as an instrument.

The results indicate that antidepressants and psychotherapy are substitutes on average, although there is substantial variation across time and space. The degree of substitutability increases slightly over time. In some markets, drugs and therapy are

[4]To avoid the estimation problem arising from the presence of observed zero market shares, I adopt a Bayesian procedure similar to the one in Li (2019).

[5]"Bundle" is defined as any single- or multiproduct depression treatment option.

complements.

The fact that drugs and therapy are substitutes would imply that, in the absence of spillovers, an increase in antidepressant advertising decreases demand for psychotherapy. However, the results show that there are strong advertising spillovers that make the net effect of drug ads on demand for therapy positive: on average, a 10 percent increase in advertising leads to a 0.093 percent increase in demand for therapy. The same change in advertising also leads to a 0.050 percent positive spillover on demand for generics and a 0.076 increase in demand for branded drugs.

The conclusions depend critically on allowing for advertising spillovers and flexible unobserved heterogeneity. Assuming that drug advertising only affects the utility of antidepressants implies that its effect on therapy is negative. Not allowing for correlated preferences leads to the erroneous conclusion that drugs and therapy are complements on average, but has a relatively small effect on the demand elasticity with respect to advertising. Assuming that the time-varying unobservables are uncorrelated across products also leads to estimates that suggest that the products are complements, and results in much larger demand elasticity with respect to advertising.

The paper contributes to two strands of economic literature: on the effects of prescription drug advertising and on discrete-choice models with complementarity. Using TV market borders as a source of exogenous variation in advertising exposure, Shapiro (2019) estimates that antidepressant ads increase depression treatment initiation and improve labor market outcomes by lowering absenteeism. He finds a positive but insignificant effect of drug ads on the use of therapy. Although seemingly

contradictory to my findings, this result may be due to the fact that most counties at TV market borders are rural and have few psychotherapy providers.[6]

A few papers find that DTC ads for one drug can have spillover effects on other drugs, but they do not consider the effects on non-drug treatments. Shapiro (2018) uses the TV market border strategy to establish that antidepressant TV ads have a spillover effect on demand for rival products and strong market expansion effect for the category of antidepressant drugs as a whole. Sinkinson and Starc (2019) use the variation in TV advertising of anticholesterol drugs induced by the U.S. election cycle and a regulatory action to find that branded drug ads steal share from other advertising branded drugs but have a spillover effects on non-advertised drugs and market expansion effect on the product category overall.

Other papers have documented that direct-to-consumer advertising can encourage an initial doctor visit or treatment initiation, which is a possible channel for the spillover effect that I find (Hosken and Wendling, 2013; Jayawardhana, 2013; Iizuka and Jin, 2005). Few papers have addressed the complementarity or substitutability of antidepressants and psychotherapy. Berndt et al. (1997) find that higher copayments for therapy are associated with higher use of antidepressants, suggesting that the two treatment options are substitutes. Butikofer et al. (2019) explore the effects of the 2007 FDA black box warnings on antidepressants and find that they led to a decline in use of antidepressants and psychotherapy, which is consistent with complementarity.

I also contribute to the literature on discrete-choice demand models with comple-

[6]Ellis et al. (2009) document that rural, low-income counties have the lowest number of mental health professionals per capita.

mentarity. In a study of the welfare effects of the entry of the online version of *The Washington Post*, Gentzkow (2007) estimates a model with complementarity and unobserved correlated preferences and establishes the conditions under which the model is identified. I extend his estimation procedure and build on his identification results by addressing the identification challenge that arises if time-varying shocks are correlated across products. Other papers that employ discrete-choice demand models that allow for complementarity are Ershov et al. (2018), Grzybowski and Verboven (2016), Song et al. (2017), and Wakamori (2015).

The rest of the paper is organized as follows. Section 1.2 provides background information about depression, its treatment options, and advertising. Section 1.3 describes the data used in the paper. Section 1.4 develops the demand model and discusses the identification challenges, while Section 1.5 explains in detail how I take the model to data. Section 1.6 interprets the results of the estimation, and Section 1.7 concludes.

1.2 Empirical Setting

Depression is a mental health disorder characterized by a variety of symptoms including feelings of deep sadness, loss of interest in activities previously enjoyed, suicidal thoughts, and bodily and cognitive changes that affect everyday functioning. Depending on the severity and duration of the symptoms, it can be diagnosed as major depressive disorder (MDD), persistent depressive disorder (dysthymia), or "other" de-

pressive disorder.[7] Depression affects roughly 10% of the adult U.S. population in any 12-month period.[8] In addition to personal suffering, it exacts a substantial economic toll. Greenberg et al. (2015) find that in 2010 the incremental economic cost of major depression was $210.5 billion, including $99 billion in direct medical costs, $9.5 billion in suicide costs, and $102 billion in workplace costs.

There are two main treatment options for depression: pharmacotherapy and psychotherapy. Pharmacotherapy involves the use of prescription drugs, called antidepressants, that target chemicals in the brain that control mood and stress. While antidepressants can improve the symptoms of depression, they often have undesirable side effects. Psychotherapy, or talk therapy, encompasses a variety treatment techniques that can be administered by a psychiatrist, psychologist, psychiatric nurse, or a social worker. The most widely used are cognitive-behavioral, interpersonal, and problem-solving therapies. The goal of these treatments is to help patients change negative thinking patterns, identify factors in their lives that contribute to their depression, and react better to stress.

The medical literature provides evidence that both antidepressants and psychotherapy can be effective in treating depression (Cuijpers et al., 2009; DeRubeis et al., 2008; Friedman et al., 2006). For mild to moderate depression, either treatment option can achieve the desired results (Croghan et al., 1998). As the severity of the disorder in-

[7]American Psychiatric Association (2013). The "other" depressive disorder category includes cases that meet some but not all criteria for any disorder in the depression category.

[8]Prevalence estimates vary based on data source and criteria used. Brody et al. (2018) report a 12-month prevalence of MDD of 8.1% for the period 2013-2016, which is in line with the prevalence from 2007 to 2012. National Institute of Mental Health (2017) reports 12-month prevalence of dysthymia of 1.5%. Prevalence rates of other depressive disorders are not available either because of data limitations or because it is often incorporated into a broader definition of depression.

creases, treatment guidelines encourage the use of both antidepressants and therapy (Silverman et al., 2015; Crismon et al., 1999). There is some evidence that drugs and therapy work better together. However, the evidence is not sufficient to conclude that they are complements in the health production function, in the sense that using one increases the incremental benefit of using the other.

While both antidepressants and psychotherapy are effective treatments for depression, only antidepressants are advertised on TV. Since 1997, when regulations regarding prescription drug advertising on television were relaxed, pharmaceutical companies have increased their TV ad expenditure significantly. Between 1998 and 2009 ad expenditure increased from \$1.2 to \$4.5 billion. This type of advertising directly to patients has attracted a lot of controversy. Arguments in favor of DTCA claim that it informs patients about existing treatment options, encourages them to contact a health care provider, reduces stigma associated with certain conditions, improves adherence to prescribed treatment, and encourages competition between pharmaceutical companies. The arguments against are that ads misinform or present a partial picture of the benefits and risks of the advertised drug, overemphasize drug treatment at the expense of alternative options, encourage inappropriate prescribing, and increase drug prices. As a result, policy makers have proposed a variety of actions toward prescription drug DTCA including an outright ban, removing its preferential treatment in corporate taxes, or stronger regulation.

In the context of depression treatment, psychologists have observed that the share of people diagnosed with depression who use psychotherapy has declined at the expense of antidepressants. Nordbal (2010) notes that between 1997 and 2008 30 percent

fewer patients received psychotherapy. While there could be multiple reasons for the decline, including introduction of antidepressants with fewer side effects, changing preferences and treatment styles, increased share of health maintenance organizations that put limits on the amount of therapy patients can receive, one of the alleged reasons for the decline is antidepressant DTC advertising. The purpose of this paper is to shed some light on these claims.

1.3 Data

1.3.1 TV Advertising Data

Advertising data come from Kantar Media's Ad$pender database. This data source keeps track of advertising expenditures for more than three million products through multiple marketing media, including television, radio, newspapers, magazines, outdoor, and online. For TV advertising, it provides monthly-level dollar expenditure and number of 30-second advertising segments (or slots). The data are available both at the national (network and cable TV) and local (spot TV) level. Local TV advertising is available for the 101 largest designated market areas (DMAs), the geographical definitions of TV markets.

The total ad expenditure on the class of antidepressant drugs for the 2008–2010 period is $655 million.[9] In stark contrast to antidepressants, psychotherapy is not

[9]The biggest spenders are Cymbalta, Pristiq, Effexor, Abilify, and Seroquel. Abilify and Seroquel are antipsychotics that were later approved for the treatment of depression as well. I include their ad expenditure in the analysis whenever it is categorized as antidepressant advertising in the Ad$pender database.

advertised on TV.[10]

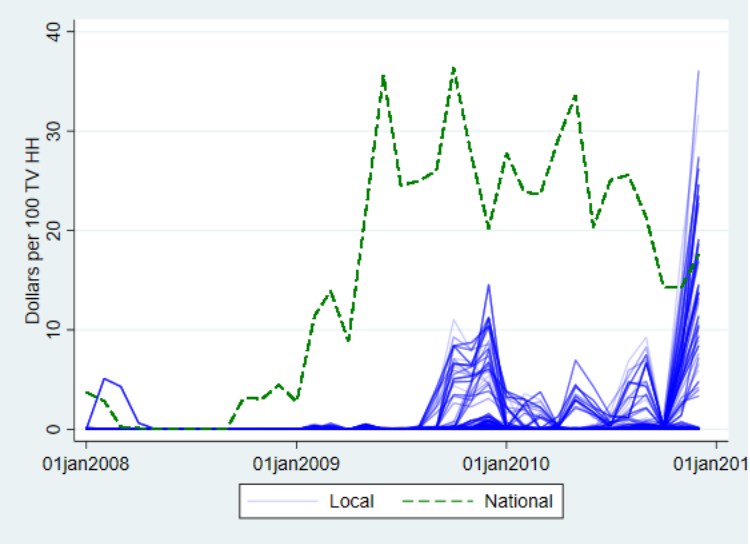


Figure 1.1: Antidepressant TV Advertising Intensity, National and Local

Notes: Advertising expenditures per 100 TV households for antidepressant prescription drugs at the national level (dashed line) and at the local level for each of the 270 MSAs (solid lines).

To combine national and local ads into a single measurement of advertising intensity, I calculate the ad expenditure per 100 households with a TV set.[11] Total ad intensity in a DMA is the sum of national ad expenditure divided by the number of TV households nationwide (in hundreds) and local ad expenditure divided by the number of TV households in the local market. This is the variable I use for advertising throughout this paper. Figure 1.1 shows national and local advertising for the period of interest. There is very little advertising activity in 2008, but it picks up in 2009 and 2010. Although national ad intensity is higher for most of the time period, there

[10]The closest category is "Mental Health & Chemical Dependency Clinics," which is predominantly composed of substance abuse clinics and rehabilitation centers. While some of them may provide treatment for depression, including psychotherapy, the focus of the ads is most likely on substance abuse. Furthermore, the total ad expenditure for this category is only 3% of the ad expenditure on antidepressants.

[11]Number of TV households is the unit that ratings company Nielsen often uses in its market size calculations. As of 2010, the United States had a population of 309 million and 115 million TV households for a 2.7 conversion factor from dollars per capita to dollars per TV household.

is substantial variation at the local level, which makes the estimation of advertising effects possible.

1.3.2 Medical and Prescription Drug Claims Data

Data on patient demographics, diagnoses, medical and prescription drug claims, and prices come from the Marketscan Commercial Claims and Encounters Database, which tracks individuals enrolled in employer-provided insurance plans from a convenience sample of large companies. The data cover 395 metropolitan statistical areas (MSAs) for the years 2008 through 2010. Because I am interested in the effect of advertising, I use only the 270 MSAs that can be matched to the 101 DMAs in the Ad$pender dataset.[12]

From the available data, I select individuals who were continuously enrolled for the full three-year period and had complete insurance plan type and demographic information. My sample includes only covered employees because employment information for spouses and dependents is unavailable.[13] I further subset the sample to individuals 18 years of age or older as of January 2008 who were not pregnant during the time period because depression treatment patterns for these subgroups are different.

Table 1.1 presents a summary of the demographic information for the 2,565,016 individuals that are in the final sample. There is a mixture of salaried and hourly,

[12]DMAs are typically centered at large MSA but may include nearby smaller MSAs. For example, the Dallas DMA includes the Dallas-Plano-Irving and Fort Worth-Arlington MSAs.

[13]Depression prevalence and treatment is different among the employed and unemployed. By limiting the analysis to employed individuals I avoid introducing bias from mixing in potentially unemployed individuals at the expense of the generalizability of the findings.

union and non-union employees, most of whom are full-time. 44.5% are female. The average age is 44.6 years. Preferred provider organization (PPO) is the most widely used type of health insurance plan, followed by health maintenance organization (HMO), point-of-service (POS), and consumer-driven and high deductible health plans (CDHP/HDHP). The sample is geographically representative at the region level.

Table 1.1: Demographics

	%		%
Female	44.5	Northeast	15.96
		Midwest	21.05
Age Group 18-24	3.03	South	37.63
Age Group 25-34	16.76	West	25.36
Age Group 35-44	26.27		
Age Group 45-54	32.74	Hourly	27.60
Age Group 55-64	21.20	Salaried	32.16
HMO	27.40	Non-Union	50.49
POS	15.51	Union	18.72
PPO	50.82		
CDHP/HDHP	6.28	Full-Time	87.01

Note: Based on 2,565,016 covered individuals. HMO = "Health Maintenance Organization", POS = "Point of service", PPO = "Preferred Provider Organization", CDHP = "Consumer Driven Health Plan", HDHP = "High Deductible Health Plan". Employees can be salaried, hourly, or unknown; union, non-union, or unknown; full-time, early retiree, part-time, or other.

Even though antidepressants and psychotherapy can treat a variety of conditions, the focus of this paper is on their use in treating depression. For this reason, I identify individuals diagnosed with three types of depression—major depressive disorder (MDD), dysthymia, and "other" depression—and analyze their choice of depression treatment.[14] Table 1.2 summarizes the share of the sample that diagnosed with each

[14]Based on the *International Classification of Diseases, Ninth Edition, Clinical Modifications* classification system, I use codes 296.2 and 296.3 for MDD, code 300.4 for dysthymia, codes 309.0, 309.1 (brief and prolonged depressive reaction), and 311 (depression not elsewhere classified) as other depression. I exclude individuals with comorbid schizophrenia, psychotic depression, or bipolar disorder because both the symptoms of and treatments for these conditions differ substantially from those for depression.

disorder from 2008 to 2010. Overall, between 4.5% and 5.0% were diagnosed with depression in any given year. This is not inconsistent with an overall depression prevalence of 10% because around one half of depression cases go undiagnosed.

Table 1.2: Depression Diagnosis Percentages by Year

	2008	2009	2010
Major Depressive Disorder	2.04	2.20	2.29
Dysthymia	0.73	0.77	0.79
Depression-Other	1.70	1.88	1.97
Total (any depression)	4.46	4.85	5.06

Note: Based on 2,565,016 covered individuals. All numbers are percentages. Depression-Other includes depression not elsewhere classified and brief and prolonged depressive reaction.

I assume that each individual makes treatment decisions monthly. For each month, I record whether the individual chose an antidepressant, therapy, both, or neither. Because I am examining the effect of advertising at the category level, I aggregate individual antidepressants into two broad categories: branded and generic.[15] If a 90-day supply of a drug was chosen, I assume that it was consumed from the month of purchase until the month the supply was exhausted. For psychoherapy, I combine individual, family, and group sessions into one good.

Figure 1.2 shows the share of all individuals that choose generics, branded drug, and therapy from January 2008 to December 2010. The use of generics increases steadily over the period; at a slower, pace the use of therapy does too. The share of branded drugs is relatively stable for most of the time period until it falls in the second half of 2010.[16] There seems to be a sharp increase in the use of both branded

[15]Only branded antidepressants advertise on TV. Once a generic version of a drug becomes available, the manufacturer of the branded version almost always stops advertising because its market share drops sharply. Ellison and Ellison (2011) find evidence that firms strategically decrease advertising before patent expiration to delay generic entry.

[16]This is when generic Effexor XR enters the market.

and generic antidepressants in the first three months of the sample. However, this is likely because of 90-day drug supplies purchased in October through December 2007, prior to the beginning of the available data. To avoid understating demand for antidepressants from January to March 2008, I exclude these months from further analysis.

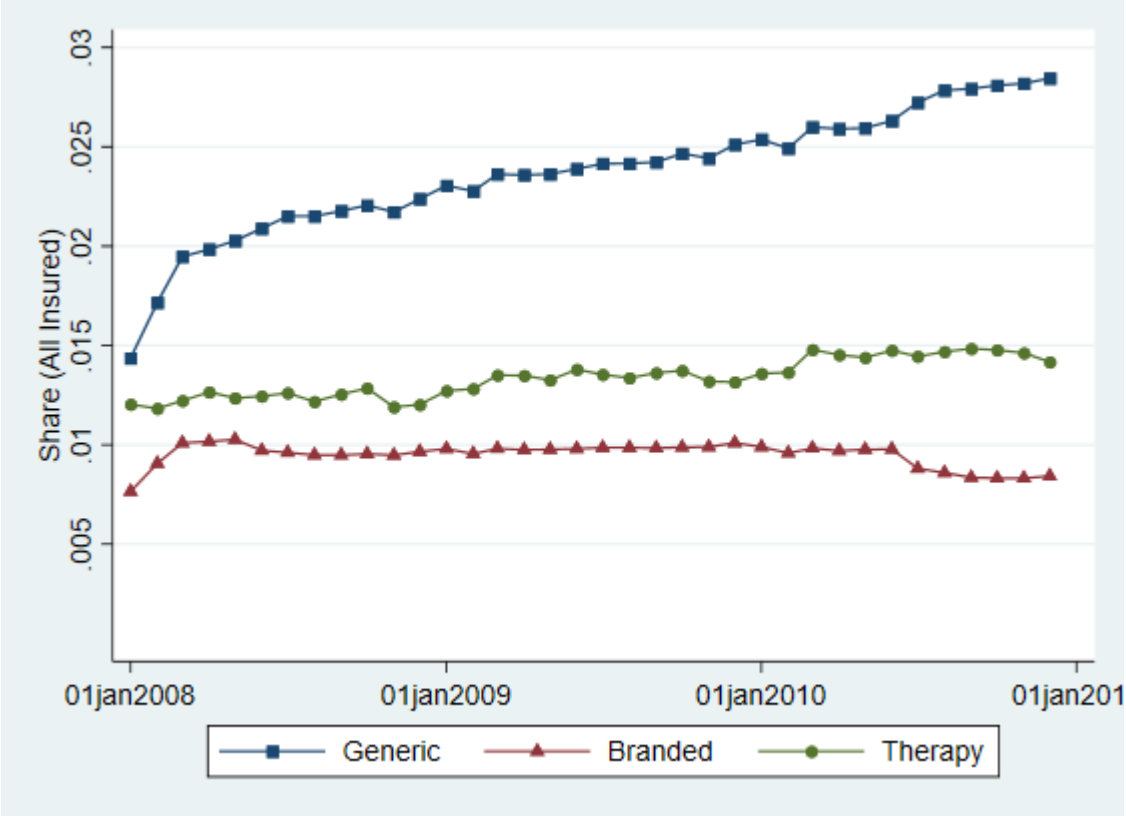


Figure 1.2: Depression Treatment Product Shares out of All Insured

Table 1.3: Average OOP Prices for Antidepressants and Psychotherapy by Insurance Type

	2008		2009		2010	
	Mean	SD	Mean	SD	Mean	SD
Health Maintenance Organization Plan						
Generic Antidepressants	6.98	2.72	6.61	2.91	6.09	2.50
Branded Antidepressants	26.81	9.76	28.19	9.12	30.13	12.68
Psychotherapy	19.70	10.26	19.88	12.13	22.49	11.88
Point of Service Plan						
Generic Antidepressants	6.85	2.05	6.98	2.51	6.61	2.20
Branded Antidepressants	24.60	7.84	24.82	8.47	25.16	12.16
Psychotherapy	31.56	18.75	34.01	22.14	33.76	17.62
Preferred Provider Organization Plan						
Generic Antidepressants	7.09	1.81	7.01	1.76	6.39	1.50
Branded Antidepressants	26.43	6.49	26.97	6.34	28.60	7.28
Psychotherapy	34.56	18.17	34.15	16.12	34.13	11.95
Consumer Driven/High Deductible Health Plan						
Generic Antidepressants	11.26	11.34	11.10	9.97	11.33	8.65
Branded Antidepressants	46.44	35.64	47.25	35.70	51.93	36.59
Psychotherapy	39.53	29.22	38.90	29.87	42.07	27.46

Note: Prices are in USD, per 30-day supply for drugs and per session for psychotherapy.

Table 1.3 summarizes the out-of-pocket (OOP) price variation for branded and generic antidepressants and psychotherapy. The OOP price is the dollar amount that the insured patient pays. It may be the full price, if a deductible applies and has not been reached, or just a copay. Because the focus of the paper is on the effect of advertising at the product category level, I calculate a price for each product category by averaging the prices of the component products at the month-state-insurance type level. Table 1.3 shows that the cheapest treatment option is generic drugs, at around \$6-7 per 30-day supply, while branded drugs and psychotherapy are considerably more expensive, at around \$25 per 30-day supply and \$30 per session, respectively. There is considerable variation around these averages based on insurance plan type, market, and time.

Table 1.4: Observed and Bayesian Posterior Mean Market Shares

	Mean	SD	Min	10^{th} Pctl	Median	90^{th} Pctl	Max	Share Zeros
# Individuals	9,589.9	16,922.4	130	482	3,201	27,082.5	154,258	0
Observed Market Shares								
Outside Option	0.9637	0.0121	0.9226	0.9481	0.9648	0.9780	1	0
Generic Only	0.0196	0.0087	0	0.0095	0.0185	0.0306	0.0622	0.003
Branded Only	0.0083	0.0036	0	0.0044	0.0079	0.0129	0.0308	0.014
Therapy Only	0.0054	0.0037	0	0.0014	0.0048	0.0102	0.0379	0.054
Generic & Therapy	0.0033	0.0026	0	0.0004	0.0028	0.0065	0.0197	0.097
Branded & Therapy	0.0015	0.0011	0	0	0.0014	0.0029	0.0090	0.179
Bayesian Posterior Mean Shares								
Outside Option	0.9585	0.0133	0.9066	0.9404	0.9605	0.9738	0.9921	0
Generic Only	0.0202	0.0086	0.002	0.0101	0.0191	0.0314	0.0621	0
Branded Only	0.0090	0.0038	0.0010	0.0049	0.0084	0.0140	0.0368	0
Therapy Only	0.0061	0.0036	0.0004	0.0024	0.0054	0.0108	0.0388	0
Generic & Therapy	0.0040	0.0025	0.0003	0.0017	0.0034	0.0071	0.0203	0
Branded & Therapy	0.0022	0.0013	0.0002	0.0009	0.0019	0.0037	0.0145	0

Notes: Summary statistics based on monthly data for April 2008–December 2010 for 270 MSAs. "# Individuals" is the number of people covered by the Marketscan data in an MSA. The Bayesian posterior mean shares are calculated using the Dirichlet-Multinomial model described in Section 1.5.6.

Table 1.4 provides a summary of the shares of each possible treatment choice at the MSA-month level. The positive shares of combination treatment with generic or branded drugs and therapy demonstrate that depressed patients can choose more than one treatment option in a given month. This cannot be the outcome of a typical discrete-choice model in which the consumer selects only the product that provides the highest utility. The model needs to be modified to accommodate choosing multiple alternatives. Furthermore, it is not possible to determine if the products are complements or substitutes just by looking at the market shares because of the confounding effect of unobserved correlated preferences. To determine the degree of complementarity between products and the effect of advertising on demand, I propose a discrete-choice model that allows for complementarity, spillover effects,

and unobserved preference heterogeneity.

1.4 Demand Model

Antidepressant advertising can have a positive effect on demand for therapy for two reasons. It may boost demand for drugs, which will in turn increase demand for therapy if the two are complements. Alternatively, it may have a spillover effect on therapy—by encouraging people to go to the doctor, for example—and raise demand for it even if the products are substitutes.

To determine the impact of advertising through each channel, I frame the patient's choice of depression treatment as a discrete-choice demand model. For simplicity, I assume that there are three treatment options: outside good (no treatment), drugs, and therapy, $j \in \{0, D, T\}$. Even though in reality the decision is made by a patient and a physician, I assume that their incentives are perfectly aligned and they act as a single decision maker.[17] The individual can choose no treatment, either drugs or therapy alone, or drugs and therapy in combination, $c \in \{0, D, T, DT\}$. The demand system is defined by the following indirect utility functions:

$$\begin{aligned} u_{i0t} &= \varepsilon_{i0t} \\ u_{iDt} &= \delta_D + \beta_D A_{Dt} + \nu_{iD} + \psi_{it} + \varepsilon_{iDt} \\ u_{iTt} &= \delta_T + \beta_T A_{Dt} + \nu_{iT} + \psi_{it} + \varepsilon_{iTt} \\ u_{iDTt} &= (\delta_D + \delta_T + \Gamma) + (\beta_D + \beta_T) A_{Dt} + (\nu_{iD} + \nu_{iT}) + 2\psi_{it} + \varepsilon_{iDTt} \end{aligned} \tag{1.1}$$

[17] I will call this decision maker an individual or patient for the rest of the paper.

All indirect utilities are normalized by the utility of the outside option. The mean utilities, δ_D and δ_T, capture the average desirability of drugs and therapy taken alone. The model allows for time-invariant preferences, ν_{iD} and ν_{iT}, time-varying health shocks, ψ_{it}, and idiosyncratic errors, ε_{ict}. Drug advertising, A_{Dt}, enters the utility of both drugs and therapy.[18] A positive coefficient on advertising in the utility of therapy indicates a spillover effect (in utility); a negative coefficient—business stealing. Spillovers can arise if drug ads encourage patients to see their doctor and they decide to take therapy, possibly in combination with drugs, to treat their depression. A business-stealing effect is possible if advertising convinces patients that antidepressants are all they need and discourages them from taking therapy.[19]

The complementarity parameter, Γ, captures the extent to which drugs and therapy work better in combination than on their own. More precisely, Γ is the amount by which the added utility of taking one treatment option changes when the other is taken as well, on average over the idiosyncratic errors ε_{ict}:

$$\Gamma = \mathbb{E}_{\varepsilon}[(u_{iDTt} - u_{iDt}) - (u_{iTt} - u_{i0t})] \tag{1.2}$$

Complementarity can arise if drugs and therapy treat depression better together than separately or if patients perceive them to do so. If this parameter tends to negative

[18]The available data do not allow me to distinguish between persuasive and informative effects of advertising, so I remain agnostic as to which one is present here. Ackerberg (2001) and Ackerberg (2003) provide a reduced-form and a structural approach to identifying informative and persuasive advertising.

[19]In a typical discrete-choice model with substitutes only, a business-stealing effect is present even if drug advertising does not affect the indirect utility of therapy. In a model with complementarity, however, if the products are complements, business-stealing is possible only if drug advertising enters the indirect utility of therapy.

infinity, the drug-therapy bundle is never chosen and the model becomes a traditional discrete-choice model with strict substitutes. Gentzkow (2007) proves that in this type of model if $\Gamma > 0$, the products are complements under the usual definition of complementarity, i.e. demand for one increases when the price of the other decreases (or its utility increases).[20]

Many of the factors involved in the choice of depression treatment are unobservable to the econometrician. Time-invariant unobserved preferences are captured by ν_{iD} and ν_{iT}. They may reflect patient attitudes towards the two treatment options that have nothing to do with how effective they are together. If individuals either dislike antidepressants but like psychotherapy (the "hippie" type) or like drugs but not therapy (the "pill-lover" type), these preferences will be negatively correlated in the population. If people either like both or neither, then the correlation in unobserved preferences will be positive.

Another unobservable variable is whether someone is depressed. Individuals go in and out of depression over time but do not always get treatment when they are depressed. While it is reasonable to infer that someone is depressed if they are diagnosed with depression and are taking antidepressants or therapy, it is unclear what their mental health state is if they are not diagnosed or getting treatment. This time-varying, potentially serially correlated, health shock unobservable is embodied by ψ_{it}. When it is in its "depressed" state, it lifts demand for all inside goods. Thus, it is correlated across products.

[20]Products are substitutes or independent if $\Gamma < 0$ or $\Gamma = 0$, respectively. See Samuelson (1974) for a comprehensive discussion of various definitions of complementarity.

1.4.1 Complementarity and Correlated Preferences

The fundamental identification problem in this model is that a large drug-therapy market share can be explained by complementarity between the products or by positively correlated unobserved preferences. Gentzkow (2007) proposes two solutions: an excluded variable that shifts the indirect utility of one product but not the other, and panel data.

The intuition behind the excluded variable approach is simple. If a variable that affects the utility of drugs but not therapy, such as the price of drugs, increases exogenously and demand for psychotherapy decreases, then the two products are complements. With Γ identified, the correlation in preferences is pinned down by the observed shares for each possible treatment choice in the market.

Identification through panel data exploits within-patient treatment shares. Assuming that the ε_{ict} errors are iid extreme value type-1 and integrating them out, it is straightforward to show that the probability of choosing the bundle relative to the product of the probabilities of choosing each product alone or in combination is:

$$\frac{s_{iDTt}(\nu_{\mathbf{i}}, \psi_{it})}{\Big[s_{iDt}(\nu_{\mathbf{i}}, \psi_{it}) + s_{iDTt}(\nu_{\mathbf{i}}, \psi_{it})\Big]\Big[s_{iTt}(\nu_{\mathbf{i}}, \psi_{it}) + s_{iDTt}(\nu_{\mathbf{i}}, \psi_{it})\Big]} \begin{cases} > 1 & \text{if } \Gamma > 0 \\ = 1 & \text{if } \Gamma = 0 \\ < 1 & \text{if } \Gamma < 0 \end{cases} \tag{1.3}$$

where $s_{ict}(\nu_{\mathbf{i}}, \psi_{it})$ is an individual's probability of choosing c conditional on the parameters of the model and the particular values of the unobservables $\nu_{\mathbf{i}} = (\nu_{iD}, \nu_{iT})$ and ψ_{it}. If products are complements, a patient consumes the drug-therapy bundle

relatively more often.[21] Given a long enough panel and time-varying errors that are not correlated across products, it is possible to identify complementarity even with no variation in an excluded variable.

1.4.2 Complementarity and Health Shocks

The crucial assumption in the panel data identification approach is that the time-varying shocks are independent across products. If they are not and that is ignored, the correlation in the shocks will be mistaken for complementarity. The situation is analogous to ignoring correlated unobserved preferences in a cross-sectional setting, in which case the correlation is also loaded onto complementarity.

Table 1.5 provides an example of the problem that unobserved health shocks can create. The health shock can take two values, "high" and "low," and the products are independent. If it were possible to observe the value of the health shocks and if the sequence of choices were long enough, I would be able to calculate the individual-specific ratio in (1.3) and conclude that the products are independent. However, the health shock is unobservable. If I calculate the ratio in (1.3) based on all observable data for an individual, I will incorrectly conclude that the products are complements.

[21] If D_{it} is the event that patient i chooses drugs alone or in combination with therapy at time t, and if T_{it} is defined similarly for therapy, I can rearrange equation (1.3) as $Cov(D_{it}, T_{it}|\nu_i, \psi_{it}) = \mathbb{E}_\varepsilon(D_{it} \cap T_{it}) - \mathbb{E}_\varepsilon(D_{it})\mathbb{E}_\varepsilon(T_{it}) = s_{iDT} - (s_{iD} + s_{iDT})(s_{iT} + s_{iDT}) > 0$ if $\Gamma > 0$. Thus, an equivalent way to express the result in (1.3) is that the within-individual covariance of the events of choosing drugs and therapy is positive.

Table 1.5: Identification Challenge with Unobserved Time-Varying Health Shocks

ψ_{it}	s_{iDT}	S_{iD}	S_{iT}	$s_{iDT}/(S_{iD}S_{iT})$
Low	0.04	0.20	0.20	1
High	0.16	0.40	0.40	1
Observed ($\frac{1}{2}$Low + $\frac{1}{2}$High)	0.10	0.30	0.30	$10/9 > 1$

Note: s_{iDT}, $S_{iD} = s_{iD} + s_{iDT}$, and $S_{iT} = s_{iT} + s_{iDT}$ are individual-specific shares of observed choices over time, either conditional on a value of the health shock (first two rows) or not (last row). The last row assumes the number of periods in which the health shock, ψ_{it}, is low and high are equal.

To avoid this pitfall, I allow for time-varying unobservable health shocks, ψ_{it}, that are correlated across products.[22] Getting the distributional assumptions for these shocks is important and puts a caveat to using panel data for identification of complementarity. This puts a greater burden on the excluded variables for identification.

1.4.3 Advertising Spillovers

The model is simplified substantially by the fact that antidepressants are advertised on TV but psychotherapy is not. This means that there is only one potentially endogenous advertising variable, and a single instrumental variable will be sufficient to address this problem. As long as complementarity and unobserved preferences are separately identified using excluded variables and panel data, it is straightforward to use an instrument that induces exogenous variation in advertising to determine the impact of drug ads on demand for drugs, therapy, and the drugs-therapy bundle.

[22]Gentzkow (2007) includes a news shock, τ_{it}, in the empirical specification of his model. He justifies it as a way to improve the fit of the model but doesn't discuss its implications about identifying complementarity.

1.5 Empirical Implementation

I generalize the model to handle more than two treatment options and more than one market, specify covariates, and parameterize the distributions of the error terms. I then use the fully specified model to derive its likelihood function. Following Goolsbee and Petrin (2004), estimation proceeds in two stages. The first stage uses maximum simulated likelihood (MSL) to estimate the coefficients on individual-specific variables and the parameters governing the distributions of the unobservables. Bundle-market-time fixed effects are "concentrated out" as in Berry et al. (2004) and Goolsbee and Petrin (2004), which eases the computational burden substantially. The second stage uses a two-stage least squares regression of the recovered fixed effects to estimate the causal effect of advertising.

1.5.1 Generalized Model

In month t, individual i, who lives in metropolitan statistical area (MSA) m, chooses a depression treatment option from among no treatment, generic antidepressants, branded antidepressants, and therapy, $j \in \{0, G, B, T\}$. The possible choices are all single- and multiproduct bundles except the ones combining branded and generic

drugs, $c \in \{0, G, B, T, GT, BT\}$.[23] The base utility from a single product j is:

$$\begin{aligned} \bar{u}_{ijmt} &= \underbrace{\delta_{jm} + \delta_{jt} + \beta_j A_{Bmt} + \xi_{jmt}}_{\delta_{jmt}} \underbrace{-\alpha P_{ijt} + \bar{\mathbf{X}}_{\mathbf{it}}\bar{\theta}_{\mathbf{j}}}_{\mathbf{X_{ijt}}\theta_{\mathbf{j}}} + \psi_{it} + \nu_{ij} \\ &= \delta_{jmt} + \mathbf{X_{ijt}}\theta_{\mathbf{j}} + \nu_{ij} + \psi_{it} \end{aligned} \tag{1.4}$$

The base utility consists of two parts: market- and individual-specific. Product mean utility may vary by market and time (δ_{jm}, δ_{jt}) to allow for different treatment styles and changing preferences. Branded antidepressants are the only product advertised on TV. Their advertising, A_{Bmt}, can have a direct effect on branded drugs (β_B) and a business-stealing or spillover effect on generics and therapy (β_G and β_T). Market-level demand shocks unobserved to the econometrician, ξ_{jmt}, may affect pharmaceutical companies' decision how much to advertise in a given market and time period, making advertising potentially endogenous.

Among the individual-specific components of base utility, OOP price, P_{ijt}, varies based on the type of insurance plan. Demographics (age, sex, insurance plan type, employment type, diagnosis; $\bar{\mathbf{X}}_{\mathbf{it}}$) can affect each product differently ($\bar{\theta}_{\mathbf{j}}$). For example, women may have different preferences for drugs and therapy than men.

Individual decisions are also affected by unobservable factors: correlated preferences (ν_{ij}) and health shocks (ψ_{it}).I assume that the time-invariant unobserved preferences are distributed multivariate normal with zero mean and unrestricted covariance matrix, $\nu_{\mathbf{i}} \sim MVN(\mathbf{0}, \mathbf{\Sigma})$, where $\nu_{\mathbf{i}} = (\nu_{iG}, \nu_{iB}, \nu_{iT})'$ and $\mathbf{\Sigma}$ is a 3-by-3 symmetric

[23] Taking more than one antidepressant at a time is strongly discouraged because of possible adverse interactions. Typically, prescribers require a two-week "wash-off" period when switching between antidepressants.

positive definite matrix.[24]

The time-varying health shock is a first-order Markov chain that can take two values:

$$\psi_{it} = \begin{cases} -\infty & \text{if healthy (H)} \\ 0 & \text{if depressed (U)} \end{cases}$$

Healthy individuals have no demand for depression treatment and never purchase any. Depressed individuals, on the other hand, choose their treatment based on the indirect utility of all possible choices. This way of modeling is convenient for three reasons: it is parsimonious; it does not require making arbitrary decisions on which individuals to include in the analysis and for what length of time; and it captures the idea that an individual that does not consume depression treatment can be either depressed or healthy.

The first-order assumption means that the distribution of ψ_{it} depends only on its value in the previous period.[25] Thus, the dynamics of ψ_{it} can be described by a 2-by-2

[24] All unobserved preferences, ν_{ij}, are relative to the preference for the outside option. All elements of Σ are normalized by variance and covariance terms of the outside option.

[25] This simplifying assumption makes the model a lot more tractable and allows me to focus on the estimation of complementarity and advertising effects while still acknowledging the transitory nature of depression. In reality, however, transition probabilities are likely endogenous.

row-stochastic matrix:[26]

$$\begin{array}{c} \\ H_{it-1} \\ U_{it-1} \end{array} \begin{array}{c} \begin{array}{cc} H_{it} & U_{it} \end{array} \\ \begin{bmatrix} \pi_{HH} & 1-\pi_{HH} \\ 1-\pi_{UU} & \pi_{UU} \end{bmatrix} \end{array}$$

The first row of the matrix gives the probability that the health shock is in state H (π_{HH}) or state U ($1-\pi_{HH}$), given that it was in state H in the previous period. The second row provides the analogous probabilities if the previous state was U.

To complete the specification of the health shock, I need to specify the probabilities that it is in each state in the initial period. For simplicity, I assume that the Markov chain is at its stationary (or long-run) distribution for each individual.[27] Thus, the initial-period distribution is $\pi^H = (1-\pi_{UU})/(2-\pi_{UU}-\pi_{HH})$ and $\pi^U = 1-\pi^H$.

With the distributions of the unobservables specified, the conditional indirect

[26] A matrix is row-stochastic if the elements of each row are between 0 and 1 and sum to 1.

[27] This assumption is one way to deal with the initial conditions problem, but is likely too strong. Fortunately, the simplification in estimation that the Markov chain health shock provides does not depend on it. In future versions of this paper, I plan to relax it by modeling the initial-state probabilities as a function of initial-period observables, as in Heckman (1981), or by using a subsample of patients who can reasonably be assumed to be healthy initially, as in Dickstein (2018).

utility function for each bundle is defined as:[28]

$$
\begin{aligned}
u_{i0mt} &= \varepsilon_{i0mt} \\
u_{icmt} &= \bar{u}_{ict} + \varepsilon_{icmt} && \text{for } c \in \{G, B, T\} \\
u_{icmt} &= \sum_{j \in c} \bar{u}_{ijt} + \Gamma_{cmt} + \varepsilon_{icmt} && \text{for } c \in \{GT, BT\} \qquad (1.5) \\
&= \underbrace{\sum_{j \in c} \delta_{jmt} + \Gamma_{cmt}}_{\delta_{cmt}} + \underbrace{\sum_{j \in c} \mathbf{X_{ijt}} \theta_{\mathbf{j}}}_{\mathbf{X_{ict}}\theta_{\mathbf{c}}} + \underbrace{\sum_{j \in c} \nu_{ij}}_{\nu_{ic}} + \underbrace{\sum_{j \in c} \psi_{it}}_{2\psi_{it}} + \varepsilon_{icmt} \\
&= \delta_{cmt} + \mathbf{X_{ict}}\theta_{\mathbf{c}} + \nu_{ic} + 2\psi_{it} + \varepsilon_{icmt}
\end{aligned}
$$

For each bundle, I include idiosyncratic extreme value type-1 error terms, ε_{icmt}, that are independent across patients, products, markets, and time periods.[29] They help rationalize the observed choices and provide closed-form choice probabilities.

I also allow the degree of complementarity to be different for generic drugs and therapy and branded drugs and therapy. Given the restrictions imposed, the complementarity for each market and time period, averaged over the ε_{icmt}'s, is:

$$\Gamma_{cmt} = \delta_{cmt} - \sum_{j \in c} \delta_{jmt} \qquad \text{for } c \in \{GT, BT\} \qquad (1.6)$$

It is necessary to allow complementarity to vary by market and month to fit the observed market shares. Forcing a single average complementarity is easily rejected

[28] With a slight abuse of notation, I use c to denote a particular choice (singleton or multi-product bundle) and the set of products that the choice contains.

[29] The scale parameter of the ε_{icmt} terms is set to 1, implying a variance of $\frac{\pi^2}{6} \approx 1.64$, to identify the model. The covariance matrix of the correlated preferences is scaled relative to the variance of ε_{ict}.

by the data.

This version of the model allows complementarity to vary by market and time but not by demographics and unobserved preferences. This restriction can be relaxed by allowing the parameters on the demographic variables (and unobservable preferences ν_{ic}) in the multi-product bundle utility functions to be estimated freely. In practice, relaxing it increases the likelihood at convergence modestly at the expense of a large increase in the number of parameters to be estimated.

1.5.2 Deriving the Likelihood Function

With the model fully specified, I can calculate individual-level choice probabilities. Healthy individuals always choose the outside option:[30]

$$s_{ict}^{H} = \begin{cases} 1 & \text{for } c = 0 \\ 0 & \text{for } c \neq 0 \end{cases} \tag{1.7}$$

For depressed patients, I analytically integrate out the idiosyncratic error term and derive the choice probabilities conditional on the patient's unobserved preferences:

$$s_{ict}^{U}(\nu_{\mathbf{i}}) = \frac{e^{\delta_{cmt} + \mathbf{X_{ict}}\theta_{\mathbf{c}} + \nu_{ic}}}{1 + \sum_k e^{\delta_{kmt} + \mathbf{X_{ikt}}\theta_{\mathbf{k}} + \nu_{ik}}} \tag{1.8}$$

Unfortunately, the likelihood of an individual's sequence of choices, even conditional on $\nu_{\mathbf{i}}$, is not simply the product of individual choice probabilities because the

[30] Superscripts H and U indicate the value of the patient's health shock, healthy ($\psi_{it} = -\infty$) or depressed ($\psi_{it} = 0$).

health shocks ψ_{it} are not independent over time. To overcome this problem, I need to integrate out the full sequence of ψ_{it}'s for each patient. In theory, this can be done analytically using the initial state probability and transition probabilities of the Markov chain. Let $\mathbf{c_i} = (c_{i1}, \ldots, c_{i\mathcal{T}})'$ be patient i's sequence of observed choices, where $\mathcal{T}$ is the last time period, and $\Theta = (\delta_{\mathbf{mt}}, \theta, \mathbf{\Sigma}, \pi_{HH}, \pi_{UU})$ be the parameters of the model, where $\delta_{\mathbf{mt}}$ is the vector of all δ_{cmt}'s. The likelihood of an individual's sequence of choices, conditional on $\nu_{\mathbf{i}}$, is:[31]

$$L_i(\Theta, \nu_i) = Pr(\mathbf{c_i}|\Theta, \mathbf{X}_i, \nu_i) = \sum_{\psi_i \in \Psi} \pi_{\psi_{i1}} s^{\psi_{i1}}_{ic_{i1}1} \pi_{\psi_{i1},\psi_{i2}} s^{\psi_{i2}}_{ic_{i2}2} \ldots \pi_{\psi_{i\mathcal{T}-1},\psi_{i\mathcal{T}}} s^{\psi_{i\mathcal{T}}}_{ic_{i\mathcal{T}}\mathcal{T}} \tag{1.9}$$

where $\pi_{\psi_{i1}}$ is the initial probability of being in state ψ_{i1} for patient i, $\pi_{\psi_{i1},\psi_{i2}}$ is the transition probability from state ψ_{i1} to ψ_{i2}, and $s^{\psi_{it}}_{ic_{it}t}$ is the probability of observed choice c_{it} in state ψ_{it}. The sum is over all possible health shock sequences $\psi_i = (\psi_{i1}, \ldots, \psi_{i\mathcal{T}})'$.

There are 33 months of data, which implies that there are 2^{33}, or about 8 billion, such sequences. Analytically integrating over all of them by brute force is not feasible. Simulation is an option, but given the length of the panel it will still be computationally expensive and introduce simulation error. Instead, I take advantage of a result from the statistical literature on Hidden Markov Models (HMMs).[32] Using a recursive relationship, the analytical expression for an individual's likelihood function becomes

[31] To simplify the notation, $s^{\psi_{it}}_{ic_{it}t}$ stands for $s^{\psi_{it}}_{ic_{it}t}(\nu_{\mathbf{i}}$.

[32] The name comes from the fact that the unobservable in this type of models is a Markov chain. The seminal paper in the literature on HMMs is Baum and Petrie (1966). A popular tutorial is Rabiner (1989). HMMs have been used heavily in speech recognition and genomic sequencing. Bartolucci et al. (2014) discuss uses of HMMs in economics.

much simpler.

Proposition. *Let f and g stand for any of the values that the first-order Markov chain ψ_{it} can take. Define the joint probability of the observed sequence up to time t and the Markov chain being in state f at time t, conditional on all observables $\mathbf{X_i}$, the unobservable $\nu_\mathbf{i}$, and the parameters of the model Θ:*

$$\phi_{it}(f) = Pr(c_{i1}, \ldots, c_{it}, \psi_{it} = f | \Theta, \mathbf{X}_i, \nu_i)$$

Claim: $\phi_{it}(f)$ can be computed recursively as:

$$\begin{aligned} \phi_{i1}(f) &= \pi_f s^f_{ic_{i1}1} && \text{for } f \in \{H, U\} \\ \phi_{it}(f) &= \Big[\sum_{g \in \{H,U\}} \phi_{it-1}(g)\pi_{gf} \Big] s^f_{ic_{it}t} && \text{for } f \in \{H, U\} \text{ and } t \in \{2, \ldots, \mathcal{T}\} \end{aligned}$$

Proof. See Appendix 1.8.1. □

Corollary. *The likelihood of an individual's sequence of choices can be calculated as:*

$$\begin{aligned} L_i(\Theta, \nu_i) &= Pr(c_{i1}, \ldots, c_{i\mathcal{T}} | \Theta, \mathbf{X}_i, \nu_i) \\ &= \phi_{i\mathcal{T}}(H) + \phi_{i\mathcal{T}}(U) \end{aligned} \tag{1.10}$$

Proof. The result follows directly from the Proposition and the definition of $\phi_{i\mathcal{T}}(f)$: sum the joint distribution over the different values of $\psi_{i\mathcal{T}}$ to get the marginal distribution, which is the joint distribution of the observed sequence of actions conditional on observables, $\nu_\mathbf{i}$, and parameters. □

Deriving the likelihood in this fashion is known as the *forward algorithm* or α-*pass*. The result is not new, although it is typically applied to models in which the probability of the observed outcome is fixed. In my application, the observed outcome is the result of a discrete-choice model. Conditioning on observables, the unobservable preference vector, and additional model parameters, the result still holds.

Given the likelihood conditional on ν_i, it is theoretically straightforward to derive the unconditional likelihood:

$$L_i(\Theta) = \int_{\nu_i} L_i(\Theta, \nu_i) dF(\nu_i)$$

There is no analytical solution of the integral over ν_i. I simulate it by drawing $R = 100$ 3-vectors $\nu_{\mathbf{i}}^{\mathbf{r}}$ distributed multivariate normal with zero mean and covariance matrix $\mathbf{\Sigma}$ for each patient, calculating the conditional likelihood, and taking the average:[33]

$$SL_i(\Theta) = \frac{1}{R} \sum_{r=1}^{R} L_i(\Theta, \nu_i^r) \tag{1.11}$$

With the individual likelihoods taken care of, it is straightforward to calculate the log simulated likelihood of the model:

$$LSL(\Theta) = \log\Big(\prod_i SL_i(\Theta) \Big) = \sum_i \log(SL_i(\Theta)) \tag{1.12}$$

[33]In practice, I take iid draws from a standard normal u_{ij}, for each j, and calculate $\nu_i = \Lambda \mathbf{u}_i$, where Λ is the lower-triangular Cholesky decomposition of Σ such that $\Lambda\Lambda' = \Sigma$. To draw the u_{ij}'s, I follow the modified Latin hypercube procedure proposed by Hess et al. (2006).

1.5.3 Estimation Stage 1: Maximum Simulated Likelihood (MSL)

I estimate the parameters of the model by maximum simulated likelihood.[34] Instead of maximizing over the entire parameter space Θ, however, I "concentrate out" δ_{cmt} and maximize over the rest of the parameters, $\tilde{\Theta} = (\theta, \mathbf{\Sigma}, \pi_{HH}, \pi_{UU})$ as in Goolsbee and Petrin (2004).[35]

For a given $\tilde{\Theta}$, I find δ_{cmt}'s such that predicted bundle shares, $s_{cmt}(\tilde{\Theta}, \delta_{\mathbf{mt}})$, match observed shares, s^{obs}_{cmt}, for each market and month. Berry et al. (1995) (BLP) prove that such δ_{cmt}'s exist and are unique. I use Goolsbee and Petrin's nonlinear least squares optimization approach to estimate the δ_{cmt}'s:[36]

$$\delta_{\mathbf{mt}}(\tilde{\Theta}) = \arg\min_{\delta_{\mathbf{mt}}} \sum_{c} (s_{cmt}(\delta_{\mathbf{mt}}, \tilde{\Theta}) - s^{obs}_{cmt})^2 \tag{1.13}$$

Given a vector of δ_{cmt}'s, I use a nonlinear optimization routine to find $\tilde{\Theta}$ that maximizes the log simulated likelihood in (1.12). The process of finding $\tilde{\Theta}$ and δ_{cmt}'s continues iteratively until convergence.

I calculate predicted market-time shares for each bundle by aggregating individual choice probabilities. Like calculating the individual-level likelihood, calculating individual-level choice probabilities involves evaluating a multidimensional integral, which I do by simulation, re-using the random draws already taken. To integrate over

[34] As explained in Greene (2012), Chapter 15, MSL estimation is not consistent for a fixed number of simulation draws. For this reason, I experimented with different numbers of draws and found no substantial difference in the estimates.

[35] The same approach, but in GMM estimation, is used in Berry et al. (2004).

[36] With 270 MSAs, 33 months, and 5 non-empty bundles, there are 44,550 δ_{cmt}'s. Goolsbee and Petrin's approach converges much faster than BLP's contraction mapping.

the unobserved health shock, I need the probabilities of being in one of two health states. They can be calculated using the initial-state and transition probabilities. The assumption that the Markov chain is in its steady state, however, implies that the probabilities of being in each state in each period are governed by the stationary distribution of the Markov chain: π^H and $\pi^U = 1 - \pi^H$. Let $\mathcal{I}_{mt}$ be the set of patients in MSA m and month t and N_{mt} be the number of these patients. The predicted share of bundle c is:

$$\begin{aligned} s_{cmt} &= \frac{1}{N_{mt}} \sum_{i \in \mathcal{I}_{mt}} s_{ict} \\ &= \frac{1}{N_{mt}} \sum_{i \in \mathcal{I}_{mt}} \frac{1}{R} \sum_{r=1}^{R} s_{ict}^{r} \\ &= \frac{1}{N_{mt}} \sum_{i \in \mathcal{I}_{mt}} \frac{1}{R} \sum_{r=1}^{R} (\pi^H s_{ict}^{H,r} + (1 - \pi^H) s_{ict}^{U,r}) \end{aligned} \tag{1.14}$$

Since the healthy type always chooses the outside option, the market shares can be rewritten as:

$$s_{0mt} = \pi^H + (1 - \pi^H) \frac{1}{RN_{mt}} \sum_{i \in \mathcal{I}_{mt}} \sum_{r=1}^{R} s_{i0t}^{U,r} \quad \text{, for } c = 0 \tag{1.15}$$

$$s_{cmt} = \quad (1 - \pi^H) \frac{1}{RN_{mt}} \sum_{i \in \mathcal{I}_{mt}} \sum_{r=1}^{R} s_{ict}^{U,r} \quad \text{, for } c \neq 0 \tag{1.16}$$

OOP price varies at the individual level and estimating its effect is part of the first stage. Typically, there is concern that price is endogenous because firms set it based partially on demand factors that are unobservable to the econometrician. This concern is attenuated here because the model includes bundle-MSA-month fixed

effects. Any product-level unobservables that vary by MSA-month, which are the typical source of endogeneity, are captured by the fixed effects. The variation that is left in the OOP is at the insurance plan type. If individuals choose their insurance plan based on their overall demand for health care and if demand for depression treatment is not correlated with that, OOP prices will be exogenous, conditional on the fixed effects.[37]

1.5.4 Estimation Stage 2: Two-Stage Least Squares

Since I observe advertising at the market-month level, its effect is not separately identified from the bundle-market-month fixed effects δ_{cmt}. However, following Berry et al. (2004), I project the estimated δ_{cmt}'s on advertising, time, and market fixed effects to estimate the own and any possible spillover or business-stealing effects of branded drug advertising. Using the definitions of δ_{cmt} from equations 1.4 and 1.5, I set up the regression:

$$\delta_{cmt} = \delta_{cm} + \delta_{ct} + \beta_c A_{Bmt} + \xi_{cmt} \qquad \text{for } c \in \{G, B, T, GT, BT\} \tag{1.17}$$

Like OOP prices, advertising is set strategically by firms and may be correlated with the error term of the model. To address concerns about endogeneity, I construct an instrument for advertising by calculating the average price for a 30-second TV

[37] If patients with unobservably high demand for depression treatment select insurance plans with lower OOP prices, the estimated coefficient on price will be biased in a negative direction. To address this issue, I plan to re-estimate the model using individuals on a particular plan type (HMO, PPO, etc.) and use variation in OOPs at the particular plan level (for patients for whom such information is available). If different plans of a given type are sufficiently similar, this approach will eliminate the selection bias.

ad slot in every DMA and month using all TV advertising data in the Ad$pender database.[38] The proposed instrument is relevant because firms have a downward sloping demand for ads.[39] It is also excluded as individual choice of depression treatment is unlikely to be influenced directly by the price of TV ads. Furthermore, given that antidepressant ads are a small portion of all TV advertising, depression treatment demand shocks are unlikely to affect the average price of an ad slot. It is possible, however, that overall viewership may affect the price of a 30-second ad slot and demand for depression treatment by making a single TV ad more effective by reaching a wider audience. To deal with this threat to identification, I adjust the average ad price by dividing it by the Nielsen Television Index (NTI) for national broadcast network television programs.[40] This eliminates the variation in prices due to the seasonality in ratings and leaves variation driven by competition for the limited number of ad slots and idiosyncratic factors unrelated to demand for depression treatment. Conditional on the MSA fixed effects and the year and trend time controls I include in the model, I claim that the adjusted ad price is exogenous to demand for depression treatment.[41]

[38] Such an instrument has been used in Murry (2017) in the study of vertical relationships between car manufacturers and dealers.

[39] Whether an increase in the price of an ad will increase or decrease a firm's advertising expenditure depends on the elasticity of advertising demand with respect to price of advertising. This effect on expenditure will be revealed in the first stage regression of advertising intensity on the price of a 30-second slot.

[40] The NTI index is provided by the Television Bureau of Advertising, Inc., at `www.tvb.org`. Because it varies monthly but not across markets, I have to assume that overall TV viewership moves similarly in different markets across the country. Ideally, I would use a market-specific ratings index.

[41] Shapiro (2018) and Sinkinson and Starc (2019) propose alternative identification strategies. I cannot use Shapiro's DMA border identification strategy because my demand data is at the MSA, rather than county, level. While using political advertising is possible, the fact that there is hardly any antidepressant advertising in 2008, when the bulk of the political advertising occurs, means that political ads will be a weak instrument at best.

1.5.5 Identification

As the model is quite complex, it is useful to discuss which moments of the data identify its parameters given the distributional and functional-form assumptions made.

The Markov transition probabilities are pinned down by the observed probabilities of switching from no treatment to consuming any depression treatment and vice versa. The fact that individuals spend long periods with no treatment (because they are healthy) and long periods under treatment (because depression tends to persist) implies that the probabilities of remaining in each state are high and the probabilities of switching are low.

The assumption that healthy individuals do not purchase any depression treatment provides a lot of identifying power because it implies that anyone taking treatment must be unhealthy. Thus, an individual's health state is uncertain only in periods with no depression treatment. For those periods, the Markov assumption helps put a probability on being depressed.[42]

Complementarity is identified by price variation and the within-individual share of the drug-therapy bundle. If demand for drugs moves in the same direction as demand for therapy in response to a price change, this implies that the two are complements. The same conclusion can be drawn if individuals purchase the bundle relatively more frequently than either product alone.

The coefficients on demographics and prices are identified by the co-variation of these variables and individual-level choices. If women are more likely to take

[42]Hidden Markov Models are particularly useful in speech recognition exactly because of the convenience with which the probability of the unobserved state can be calculated. This "decoding" step is not necessary in the estimation of my model but is useful in thinking about identification.

antidepressants but equally likely to go to psychotherapy, then the coefficient on the drugs-female interaction will be positive while the one on therapy-female interaction will be zero.

The parameters governing transitions, complementarity, and demographic and price effects imply certain predicted shares for each bundle in each MSA-month. The extent to which observed shares deviate from the predicted ones identifies the correlation in unobserved patient preferences. For example, if within-patient shares imply that the products are substitutes, but the observed share of the drug-therapy bundle is larger than what the model predicts, this suggests that preferences are positively correlated.

The variance of the unobserved preferences (relative to the normalized variance of the idiosyncratic error term), is identified by the dispersion of purchasing patterns across patients. The presence of patients who always choose drugs but not therapy and others who always choose therapy but not drugs suggests that variances are large. If everyone followed the same treatment plan, the variances would be close to zero.

Finally, the effects of branded antidepressant TV advertising are identified by the covariance between observed choices and the exogenous variation induced by the cost instrument. If advertising leads to greater probability of purchasing drugs, alone or in combination, but not of therapy alone, this implies that there is a positive effect of advertising on drugs but not on therapy.

Functional form and distributional assumption facilitate the estimation of the model, but are not crucial for identification. Berry et al. (2013) show that a nonparametric nonseparable demand system is invertible under the "connected substitutes"

condition. This condition requires that goods are weak gross substitutes and that there are no groups of goods that substitute only among themselves but not to the outside good. Because a discrete-choice demand model with complementarity can be framed as a regular discrete-choice model in which the goods are all possible combinations of the available products, the condition is satisfied. Given that demand is invertible, it is identified in the presence of good instruments. Berry and Haile (2014) provide conditions under which demand is identified with market-level data, while Berry and Haile (2010) focus on situations in which individual-level data are available.[43]

1.5.6 Zero Observed Market Shares

Table 1.4 shows that the number of people covered by the Marketscan data varies substantially, with as few as 130 in some MSAs. A prevalence of 10% implies that in such markets there are about 13 depressed individuals. This makes it likely that some of the treatment options will not be chosen in some months. Indeed, for the various bundles this happens in 0.3%–17.9% of all MSA-months. Overall, 21.8% of all MSA-months have at least one zero share.

Estimating the model requires that there are no bundles with zero observed share in any market and time period.[44] Rather than drop MSA-months with zero shares

[43]Berry and Haile (2016) provide an accessible overview of the identification arguments made in the three papers cited above.

[44]In this model, a zero share implies that for this bundle-MSA-month δ_{cmt} approaches negative infinity, which throws off the estimation procedure. In general, models that involve inverting market shares, such Berry (1994) and Berry et al. (1995), or estimating product-market(-time) fixed effects, such as Goolsbee and Petrin (2004) and Berry et al. (2004), require that there are no zero observed market shares.

and potentially introduce selection bias, I use a Bayesian procedure similar to the one proposed by Li (2019). Abstracting away from the complexity of the individual-level discrete-choice model, I assume that observed bundle purchase counts in each market and time period, K_{cmt}, are the outcome of a multinomial random variable parameterized by the number of individuals, N_{mt}, and the probabilities for each bundle, p_{cmt} such that $\sum_c p_{cmt} = 1$. Instead of using the observed market shares, which is equivalent to using maximum likelihood to estimate the true underlying probabilities, I use the posterior means from a Bayesian model. I put a weak and uninformative Dirichlet prior on $\mathbf{p_{mt}}$, which defines the Dirichlet-Multinomial model:[45]

$$K_{cmt} \sim Multinomial(N_{mt}, \mathbf{p_{mt}}) \tag{1.18}$$

$$\mathbf{p_{mt}} \sim Dirichlet(1, 1, 1, 1, 1, 1) \tag{1.19}$$

Such models are convenient because the Dirichlet distribution is a conjugate prior to the multinomial likelihood, which means that the posterior distribution is also Dirichlet with parameters $1 + K_{cmt}$ for each c. The posterior mean for each bundle is easy to derive:

$$\hat{p}_{cmt} = \frac{1 + K_{cmt}}{\sum_k (1 + K_{kmt})} = \frac{1 + K_{cmt}}{6 + N_{mt}} \tag{1.20}$$

[45]The Dirichlet distribution is a multivariate generalization of the beta distribution and is suitable as a prior for probability vectors because it is defined on the unit simplex. The prior is weak because the hyperparameters (the vector of 1's) are small in magnitude. It is uninformative because it implies that vector of multinomial probabilities is equally likely. Both of these characteristics of the prior allow the observed data to be the main determinant of the posterior distribution. A similar Dirichlet-Multinomial model has been used in Conlon and Mortimer (2019b) in the empirical study of diversion ratios.

The posterior mean is strictly positive, which solves the problem of zero shares and makes estimation possible without discarding any markets or time periods.

1.6 Results

Even with the use of the forward algorithm to avoid simulating the time-varying health shocks, the model is computationally challenging. To ease the burden, I estimate it using a panel spanning 33 months for a random subsample of 13,500 individuals from the 270 MSAs.

1.6.1 Price, Demographics, Distribution of Unobservables

Table 1.6 contains the results from the first stage of the model. These parameter estimates reflect the preferences of depressed patients. By definition, healthy individuals do not consume depression treatment.

Table 1.6: First Stage (MSL) Estimates

	Est	SE	Est	SE	Est	SE
OOP	-0.206	(0.143)				
×MDD	0.721***	(0.083)				
×Depression-Other	0.039	(0.093)				
×Female	-0.041	(0.101)				
×Age	0.103**	(0.048)				
×Salaried	0.221	(0.140)				
×Union	0.283**	(0.136)				
	×Generic		×Branded		×Therapy	
Female	0.958***	(0.095)	1.106***	(0.142)	1.134***	(0.134)
Age	0.363***	(0.046)	0.132*	(0.08)	-0.202***	(0.068)
Age^2	-0.174***	(0.051)	-0.066	(0.058)	-0.144***	(0.041)
HMO	0.277	(0.181)	-0.254	(0.205)	1.025***	(0.233)
POS	-0.229	(0.198)	-0.151	(0.219)	1.456***	(0.223)
PPO	0.518***	(0.161)	1.232***	(0.189)	1.779***	(0.212)
Salaried	-0.574***	(0.133)	-0.114	(0.206)	-1.008***	(0.206)
Hourly	-1.099***	(0.126)	-0.571***	(0.141)	-0.706***	(0.116)
Union	1.148***	(0.144)	0.971***	(0.204)	0.784***	(0.178)
Non-Union	1.079***	(0.125)	1.29***	(0.130)	0.841***	(0.103)
Full-Time	-0.347***	(0.117)	0.066	(0.137)	-0.668***	(0.101)
MDD	0.487***	(0.066)	0.301**	(0.117)	0.335***	(0.102)
Depression-Other	0.662***	(0.078)	1.722***	(0.149)	-0.589***	(0.135)
Log-likelihood			-31,839.5			
Observations			445,500			

Notes: Bundle-MSA-month fixed effects included. Demographics are interacted with product dummies, OOP and OOP interactions are not. For the multiproduct bundles (not shown), the demographic effects equal the sum of the demographic effects on the component single-product bundles. OOP prices and age are measured in standard deviations ($24.52 and 10.62 years, respectively) and centered around their means ($26.70 and 44.85 years). Employees can be salaried, hourly, or unknown; unionized, non-unionized, or unknown; full-time, early retiree, part-time, or "other." Depression diagnosis can be major depressive disorder (MDD), dysthymia, or depression-other. The omitted health insurance type is CDHP/HDHP. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

Taking advantage of the individual-level data, I allow the price sensitivity to vary by demographic characteristics. The base group of individuals seems to dislike higher prices, although the coefficient is not statistically significant. While men and women are equally price sensitive, older individuals and those who are salaried or unionized employees seem less sensitive. This could partially be driven by the fact their younger, part-time or non-unionized counterparts are likely to have lower income, which I do

not observe. Strangely, patients diagnosed with major depressive disorder seem to derive greater utility from higher prices. This result should be taken with a grain of salt. It is likely driven by the fact that depression severity varies unobservably even within major depression and more severely depressed patients choose more expensive treatments.[46]

In addition to the interaction with price, the type of diagnosis affects choice directly. Patients with MDD have higher demand for all types of treatments, while those with other forms of depression are more heavily reliant on antidepressants and less on psychotherapy compared to patients with dysthymia.[47]

Women use depression treatment more heavily, which has been documented in the medical literature. Generic and branded antidepressant use increases at a decreasing rate until around age 56, whereas therapy use is heaviest among 37-year-olds and decreases away from this age. Relative to hourly workers, salaried ones use more antidepressants and slightly less therapy, although both groups use less depression treatment than workers with unknown status. Unionized and non-unionized workers use approximately the same amount of depression treatments, which is more than workers with unknown unionization status. Full-time employees fill fewer generic antidepressant prescriptions and take less psychotherapy compared to (mostly) early retirees.

Finally, PPO plans are associated with the highest level of depression treatment usage, although the results cannot tell if this is due to selection or because PPO

[46]To address this problem, I plan to use more granular diagnosis information, which I am currently aggregating over.

[47]The current version of the model assumes that the degree of complementarity does not depend on the type of diagnosis. This can be relaxed in future versions.

plans have more generous coverage. HMO, POS, and CDHP/HDHP plans have approximately the same utilization of generic and branded antidepressants. In terms of psychotherapy use, PPO is the highest, followed by POS, HMO, and CDHP/HDHP plans. This can be explained partially by the ease with which patients can access therapists under each plan—PPO plans do not require a referral from a primary care physician, whereas POS and HMO plans do.

Table 1.7: Covariance/Correlation Matrix of Unobserved Preferences

	Generic	Branded	Therapy
Generic	8.64	0.34	0.25
Branded	4.06	16.25	0.44
Therapy	2.34	5.51	9.84

Notes: Variance and covariance terms on the main diagonal and below; correlation coefficients—above. All parameter estimates are significant at the 1% significance level.

The estimated covariance matrix of the unobserved preferences for each product, ν_{ij}, is in Table 1.7. The variance terms (8.6, 16.3, 9.8 for generics, branded, and therapy, respectively) are much larger than the normalized variance of the extreme value type-1 error of 1.64, which suggests that unobservable factors play a much larger role in the choice of depression treatment than observable characteristics. It also means that observably similar patients choose radically different treatment plans: from exclusively pharmacologic to exclusively psychotherapeutic treatment and anywhere in-between. Neither of these results is surprising given how idiosyncratic the manifestation of depression and the effectiveness of different treatments are.

Table 1.8: Markov Chain Health Shock Transition Probabilities and Stationary Distribution

Panel A: Transition probabilities		
	Healthy_{t+1}	Depressed_{t+1}
Healthy_t	0.9982	0.0018
	(0.0000)	(0.0000)
Depressed_t	0.0175	0.9825
	(0.0000)	(0.0000)
Panel B: Stationary distribution		
	Healthy	Depressed
Long-run share	0.9066	0.0934

Note: Standard errors in parentheses.

The positive covariance terms imply that patients tend to either like any two treatment options or neither. They also suggest that patients see all three treatment options as somewhat "similar" in the sense that a change in the price of one would lead to greater substitution to inside bundles than to the outside option compared to what an IIA logit model would predict.[48]

The estimated health state transition probabilities, shown in Table 1.8, indicate that both the healthy and unhealthy states are highly persistent. The probability of falling into depression, having been healthy the previous month, is 0.18% whereas the probability of recovery once in a depression is 1.75%. The implied stationary distribution of healthy and unhealthy people is 90.66% healthy and 9.34% depressed. The share of depressed is higher than what is actually observed, as reported in Table 1.2, but there is no inconsistency because the former includes both diagnosed and undiagnosed cases whereas the observed share includes only diagnosed cases. Given that roughly half of depression cases go undiagnosed, the results are in line with the

[48]Greater substitution to inside bundles, however, does not necessarily mean that the products comprising the bundles are substitutes. That is determined by the degree of complementarity.

medical literature (Williams et al., 2017).

1.6.2 Complementarity

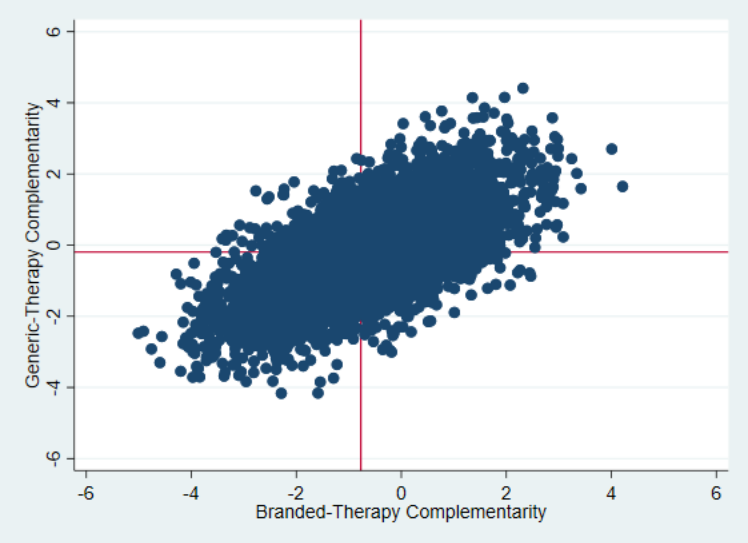


Figure 1.3: Generic-Therapy and Branded-Therapy Complementarity by MSA and Month

Note: Each point represents a $(\Gamma_{GTmt}, \Gamma_{BTmt})$ pair for a particular MSA-month. The vertical line is at the mean branded-therapy complementarity (-0.77), the horizontal—at the mean generic-therapy complementarity (-0.19).

Equation 1.6 defines the market- and time-specific complementarity parameters for the generic-therapy and branded-therapy combination treatments. Using the estimated bundle-MSA-month fixed effects, I calculate the implied complementarities and plot them in Figure 1.3. If there were a single complementarity parameter for each multiproduct bundle, the plot would consist of a single point. The wide dispersion in the plot suggests that the degree of complementarity varies over markets and time. The reason for that could be that patients have different preferences, physicians follow different treatment guidelines, or there are idiosyncratic factors that shift complementarity.

While both generic and branded antidepressants are substitutes to psychotherapy on average, there's substantial variation over product categories, markets, and time. As indicated by the average complementarity parameters, branded drugs are more substitutable with therapy ($\Gamma_{BT} = -0.77$) than generic drugs ($\Gamma_{GT} = -0.19$). Furthermore, Figure 1.4 reveals the distribution of average (over time periods) complementarity at the MSA level: in 113 out of the 270 MSAs (42%) generic drugs and therapy are complements; for branded drugs, there are 42 such MSAs (16%).

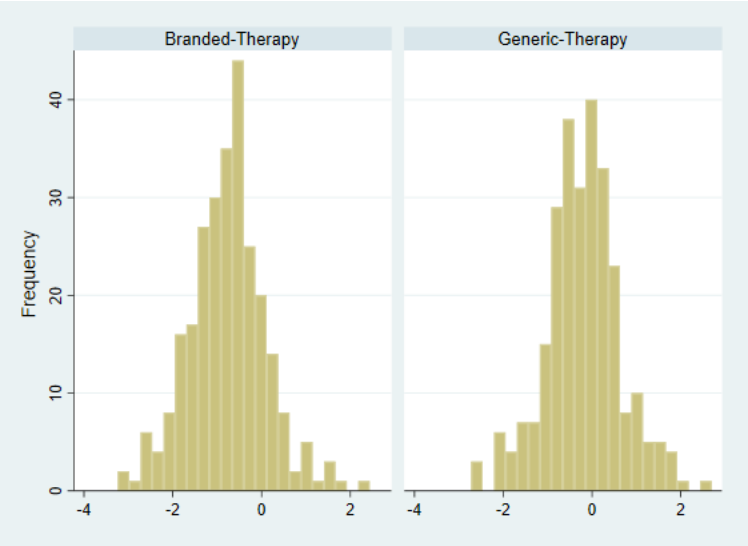


Figure 1.4: Distribution of Average MSA Complementarity

Figure 1.5 shows that the average complementarity across MSAs decreased over the 2008–2010 period, which suggests that patients became less likely to take combination treatment over time. If this trend had been going on for a while, it might partially explain the fall of psychotherapy's share over time.

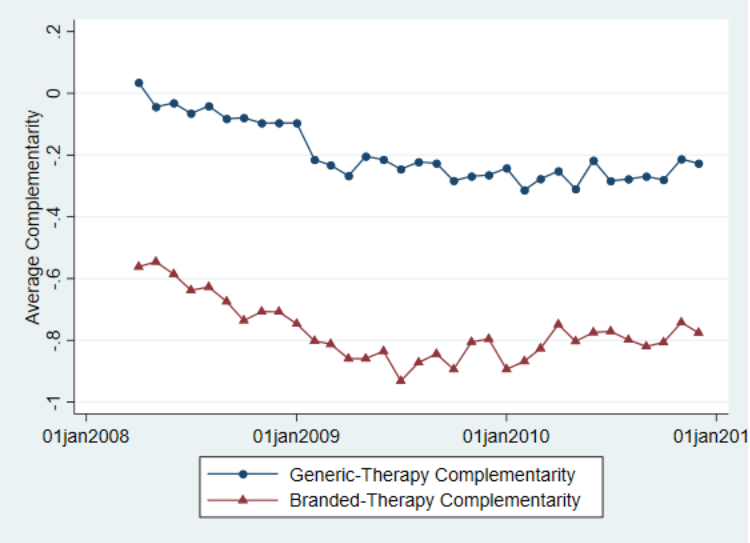


Figure 1.5: Average Generic-Therapy and Branded-Therapy Complementarity Over Time

1.6.3 Advertising Effects

The first stage of the estimation recovers the bundle-MSA-month fixed effects, which reveals the average degree of complementarity and its distribution. Advertising effects, however, are not separately identified from the fixed effects. To estimate the effects of advertising, I project the estimated δ_{cmt}'s on advertising intensity, MSA and year fixed effects, and a time trend.

Ordinary least squares (OLS) results are presented in Table 1.9. An increase in advertising intensity is associated with higher utility for branded drugs, lower utility for generics, and no effect for therapy. Because branded drugs are substitutes with both generics and therapy, this implies that branded drug TV ads increase demand for branded drugs overall and decrease it for generics and therapy.

Table 1.9: Second Stage, OLS Estimates

	Generic	Branded	Therapy	G-T Bundle	B-T Bundle
δ_c	-2.8545***	-5.8453***	-5.1612***	-8.2096***	-11.7781***
	(0.0673)	(0.0789)	(0.072)	(0.0813)	(0.0889)
Ads ($/100 TV HH)	-0.0026***	0.0059***	0.0009	-0.0017**	0.0068***
	(0.0006)	(0.0007)	(0.0006)	(0.0007)	(0.0008)
Time effects	Year-Trend	Year-Trend	Year-Trend	Year-Trend	Year-Trend
Market FEs	MSA	MSA	MSA	MSA	MSA
Obs	8,910	8,910	8,910	8,910	8,910

Notes: Standard errors in parentheses. δ_c is the mean utility for a bundle for the base time period and MSA. For the multiproduct bundles, the advertising effects equal the sum of the effects on the component single-product bundles. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

To address potential endogeneity concerns, I use the average price of a 30-second TV advertising segment, adjusted for ratings, as an instrument. Panel B of Table 1.10 shows the outcome of the first stage of the two-stage least squares (2SLS) procedure. The relationship between advertising and the instrument is negative, as expected. The partial F-statistic of 505 is much larger than conventionally used cutoff points, indicating that the instrument is strong.

Table 1.10: Second Stage, 2SLS Estimates

Panel A: regression of bundle-MSA-month fixed effects on advertising					
	Generic	Branded	Therapy	G-T Bundle	B-T Bundle
δ_c	-2.8148***	-5.8388***	-5.1303***	-8.139***	-11.7407***
	(0.0689)	(0.0803)	(0.0735)	(0.0833)	(0.0906)
Ads ($/100 TV HH)	0.0053***	0.0072***	0.0071***	0.0124***	0.0143***
	(0.0025)	(0.003)	(0.0027)	(0.0031)	(0.0033)
Time effects	Year-Trend	Year-Trend	Year-Trend	Year-Trend	Year-Trend
Market FEs	MSA	MSA	MSA	MSA	MSA
Obs	8,910	8,910	8,910	8,910	8,910
Panel B: first stage of instrumental variables regression					
Adj Avg Price per Ad			-0.0057		
Partial F-stat			504.8		

Notes: Standard errors in parentheses. δ_c is the mean utility for a bundle for the base time period and MSA. For the multiproduct bundles, the advertising effects equal the sum of the effects on the component single-product bundles. The average price per ad is adjusted for TV ratings. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

The second-stage 2SLS results are in Panel A of Table 1.10.[49] The estimates suggest that advertising makes branded drugs more desirable, as in the OLS version. In the instrumented version of the model, however, there is evidence of advertising spillovers in utility: advertising significantly increases the utility from generic antidepressants and therapy as well. The difference compared to the OLS results suggests that firms may be advertising more heavily in markets with lower demand, especially for generics and therapy.

The temptation to interpret the positive advertising coefficients as an indication that demand for all three products (therapy, generic and branded drugs) increases in response to higher advertising should be resisted. The overall effect of advertising depends both on these coefficients and on the degree of complementarity between the products. Given that generic and branded drugs are substitutes to therapy, if there were no advertising spillovers in utility but only a positive effect on branded drugs, an increase in advertising intensity would increase demand for the branded-only and branded-therapy bundles and decrease demand for the therapy-only, generics-only, and generics-therapy bundles. Despite the positive effect on the branded-therapy bundle, the overall effect on demand for therapy would be negative. The presence of advertising spillovers in utility, however, dampens the substitution away from therapy and, if the spillover is strong enough, may boost demand for therapy overall.

[49] I have also estimated the model using three-stage least squares (3SLS), which uses the covariance in the errors of the equations for each bundle to enhance estimation efficiency. The results are similar, with slightly smaller standard errors. The downside of 3SLS is that misspecification in one equation is transferred to the entire system of equations. Because of this, I chose the less efficient but more robust estimation method.

Table 1.11: Demand Elasticities with Respect to Advertising

Bundles	Elasticity	Products	Elasticity
Outside Option	-0.0053	Outside Option	-0.0053
Generic-Only	0.0033	Generic	0.0050
Branded-Only	0.0063	Branded	0.0076
Therapy-Only	0.0061	Therapy	0.0093
Generic-Therapy	0.0147		
Branded-Therapy	0.0177		

Note: Demand elasticities with respect to branded drug advertising are calculated by averaging individual-level elasticities over patients and time periods.

Table 1.11 presents the estimated average (over patients and time periods) elasticities for each bundle and each product. They indicate that advertising lifts all individual inside bundles and, as a result, each product overall. Thus, the advertising spillovers in utility translate into spillovers in demand as well: even though branded and generic antidepressants are substitutable with psychotherapy, the overall effect of advertising is to increase demand for all three of them.

The effect of advertising is positive but modest in magnitude. The estimates imply that a 10% increase in advertising increases demand for generic drugs, branded drugs, and therapy by 0.050%, 0.076%, and 0.093%, respectively.[50] The share of the outside option shrinks by 0.053% in response to the same increase in advertising. The numbers are slightly lower, although largely in line, with other estimates from the literature.[51]

[50]These are contemporaneous effects. Additional specifications suggest that advertising has no significant lagged effect on branded and generic antidepressants. The advertising effect on psychotherapy, however, persists for an additional month or two.

[51]Sinkinson and Starc (2019) find that the category of statin drugs expands by 0.13% in response to a 10% increase in advertising. Shapiro (2018) finds that the outside share in the market for antidepressants decreases by between 0.08% and 0.23% in response to the same change in advertising. Caution should be used for these last comparisons since the outside option is defined differently in Shapiro's model and mine.

1.6.4 Importance of Assumptions on the Unobservables

There are three features of the model that are crucial for estimating the correct degree of complementarity and advertising elasticities: allowing for time-invariant correlated preferences, time-varying health shocks, and advertising spillovers. I evaluate their importance by eliminating them from the model one at a time and re-estimating it.

Because advertising is subsumed by the bundle-MSA-month fixed effects, eliminating spillovers does not change the results from the first stage of the estimation. Thus, the effects of price and demographics on utility, the covariance matrix of the unobserved preferences, and the Markov transition probabilities remain the same as in the main specification. It also implies that the average level of complementarity and the market-time deviations from it are the same as in the main model. The differences appear in the effect of advertising on utility and especially in the elasticities as shown in Tables 1.16 and 1.17, column (2), in Appendix 1.8.2. The effect of advertising on branded drugs is somewhat higher than in the main model, although within a standard deviation from it. The most significant difference, however, is in the implied elasticities. While the main results suggest that advertising "lifts" all antidepressants and therapy, the model with no spillovers indicates that branded drugs benefit at the expense of generics, therapy, and the outside option. The results are driven by the fact that both generics and therapy are substitutes for branded drugs and even though the branded-therapy bundle benefits from advertising, this effect is not strong enough to offset the decline in therapy-only and generic-therapy. The fact that the ad effects on generics and therapy flip in sign underscores the importance of

allowing for spillovers.

Column (3) in Tables 1.12–1.17 shows the results for a model that allows for random coefficients on generics, branded drugs, and therapy but restricts them to be uncorrelated, while maintaining the other features of the main model. The results from the MSL estimation are qualitatively similar to those from the main specification. The difference in the log-likelihood at convergence is relatively small, which suggests that allowing for correlated preferences affects the overall fit relatively little. Most significantly, every single Γ_{cmt} is larger than in the baseline model. As a result, the average level of complementarity increases substantially and implies that therapy and both types of antidepressants are complements on average. This, however, translates into relatively modest changes in the estimated average elasticities. In response to a 10% increase in advertising, demand for generics, branded drugs, and therapy increases by 0.051%, 0.089%, and 0.097%, which is respectively 2%, 17%, and 4% higher than the main model.

Finally, column (4) in Tables 1.12–1.17 shows the results from the model in which there are no health shocks, so that the only time-varying unobservables are the extreme value type-1 error terms, but spillovers and correlated preferences are allowed. The first stage of the estimation indicates that the correlation in preferences is much larger than in the main specification. This is expected—the ignored positive correlation in the health shocks is loaded (partially) on the correlation of the unobserved preferences. The log-likelihood at convergence indicates a significantly worse fit than the main model. Like ignoring correlation in the time-invariant unobservable preferences, ignoring time-varying health shocks increases each individual Γ_{cmt}, which once

again leads to the conclusion that drugs and therapy are complements on average. In this specification, however, there is also a significant increase in the average elasticities—from 6 to 13 times. The reason for this result is that this version of the model assumes that all individuals in the sample are in the market for depression treatment whereas the main model estimated a sizeable portion of healthy individuals that have no demand for depression treatment. Even though not all individuals are advertising-marginal, the share that are is much larger. This underscores the importance of time-varying product-correlated shocks in discrete-choice models with complementarity that use panel data.

1.7 Conclusion

I study the effect of antidepressant advertising on demand for depression treatment using a discrete-choice model that allows for complementarity and advertising spillovers. The model allows for flexible unobserved heterogeneity: time-invariant preferences and time-varying health shocks, both of which can introduce correlation in utility across products. To separately identify complementarity from unobserved correlated preferences, I use panel data on choices and variation in an excluded variable, price. I estimate the causal effect of advertising by using a cost-based instrument. The model advances existing discrete-choice models with complementarity by allowing for advertising spillovers, multiple markets, and endogenous variables; discussing the threats to identification arising from time-varying, product-correlated unobservables; and modeling such unobservables in a computationally feasible way.

The results indicate that branded drug TV advertising increases demand for psychotherapy. This is the outcome of two forces working in opposite directions. First, drugs and therapy are substitutes, which implies that advertising, which boosts demand for drugs, should decrease demand for therapy. However, advertising has a spillover effect on the utility of therapy. This spillover effect dominates the substitution effect for a net positive impact on psychotherapy.

This result has important policy implications. First, providers of psychotherapy, who feel that antidepressant ads are stealing their patients, need not worry—drug advertising actually helps them. Second, policymakers that propose banning or curtailing prescription drug ads need to be aware of the unintended consequences of such actions. Shapiro (2019) shows that antidepressant advertising improves labor market outcomes and that the benefits far outweigh the costs. This paper sheds additional light on one of the channels through which the effect occurs.

A direction for future work is to add a supply-side model and study the effects of a counterfactual ban of prescription drug TV ads. This will require the demand analysis to proceed at the product level, which is possible but will be more computationally cumbersome. It will also require data on firms detailing, or direct-to-physician advertising, which may be a substitute or complement to direct-to-consumer advertising for firms.

Another potentially fruitful application of this type of demand model is the study of the welfare effects of tying and bundling. The effects of these pricing practices depend both on the degree of complementarity and the correlation and preferences, which can be estimated with the model I propose.

1.8 Appendix

1.8.1 Proof of the Proposition on Hidden Markov Models

Proposition. *Let f and g stand for any of the values that the first-order Markov chain ψ_{it} can take. Define the joint probability of the observed sequence up to time t and the Markov chain being in state f at time t, conditional on all observables $\mathbf{X_i}$, the unobservable $\nu_\mathbf{i}$, and the parameters of the model Θ:*

$$\phi_{it}(f) = Pr(c_{i1}, \ldots, c_{it}, \psi_{it} = f | \Theta, \mathbf{X}_i, \nu_i)$$

Claim: $\phi_{it}(f)$ can be computed recursively as:

$$\begin{aligned}
\phi_{i1}(f) &= \pi_f s^f_{ic_{i1}1} && \text{for } f \in \{H, U\} \\
\phi_{it}(f) &= \Big[\sum_{g \in \{H,U\}} \phi_{it-1}(g)\pi_{gf} \Big] s^f_{ic_{it}t} && \text{for } f \in \{H, U\} \text{ and } t \in \{2, \ldots, \mathcal{T}\}
\end{aligned}$$

Proof. By definition

$$\begin{aligned}
\phi_{i1}(f) &= \pi_f s^f_{ic_{i1}1} \\
&= Pr(\psi_{i1} = f) Pr(c_{i1} | \psi_{i1} = f, \Theta, \mathbf{X}_i, \nu_i) \\
&= Pr(c_{i1}, \psi_{i1} = f | \Theta, \mathbf{X}_i, \nu_i)
\end{aligned}$$

Thus, $\phi_{it}(f)$ is the joint probability of choosing the observed choice c_{i1} and being in health state f at $t = 1$.

For any $t > 1$:

$$
\begin{aligned}
\phi_{it}(f) &= \Big[\sum_{g \in \{H,U\}} \phi_{it-1}(g)\pi_{gf} \Big] s^f_{ic_{it}t} \\
&= \Big[\sum_{g \in \{H,U\}} Pr(c_{i1}, \ldots, c_{it-1}, \psi_{it-1} = g | \Theta, \mathbf{X}_i, \nu_i) Pr(\psi_{it} = f | \psi_{it-1} = g) \Big] s^f_{ic_{it}t} \\
&= \Big[\sum_{g \in \{H,U\}} Pr(c_{i1}, \ldots, c_{it-1}, \psi_{it-1} = g | \Theta, \mathbf{X}_i, \nu_i) \times \\
&\qquad\qquad \times Pr(\psi_{it} = f | \psi_{it-1} = g, c_{i1}, \ldots, c_{it-1}, \Theta, \mathbf{X}_i, \nu_i) \Big] s^f_{ic_{it}t} \\
&= \Big[\sum_{g \in \{H,U\}} Pr(c_{i1}, \ldots, c_{it-1}, \psi_{it-1} = g, \psi_{it} = f | \Theta, \mathbf{X}_i, \nu_i) \Big] s^f_{ic_{it}t} \\
&= Pr(c_{i1}, \ldots, c_{it-1}, \psi_{it} = f | \Theta, \mathbf{X}_i, \nu_i) Pr(c_{it} | \psi_{it} = f, \Theta, \mathbf{X}_i, \nu_i) \\
&= Pr(c_{i1}, \ldots, c_{it-1}, \psi_{it} = f | \Theta, \mathbf{X}_i, \nu_i) Pr(c_{it} | \psi_{it} = f, c_{i1}, \ldots, c_{it-1}, \Theta, \mathbf{X}_i, \nu_i) \\
&= Pr(c_{i1}, \ldots, c_{it-1}, c_{it}, \psi_{it} = f | \Theta, \mathbf{X}_i, \nu_i)
\end{aligned}
$$

Moving from line 2 to 3 is possible because of the first-order Markov assumption. Moving from line 4 to 5 requires integrating out ψ_{it-1}. Moving from line 5 to 6 stems from the assumption that there is no structural dependence in choices—this assumption, however, is not required for the proof of the proposition and can be relaxed. The crucial assumption is the first-order Markov assumption. □

1.8.2 Alternative Assumptions on the Unobservables

Table 1.12: First Stage (MSL) Estimates - Sensitivities

	(1)/(2) Main/No Spillovers		(3) Uncorrelated Preferences		(4) No Health Shocks	
	Est	SE	Est	SE	Est	SE
OOP	-0.206	(0.143)	0.077	(0.141)	-0.284***	(0.102)
×MDD	0.721***	(0.083)	1.139***	(0.083)	0.709***	(0.068)
×Depression-Other	0.039	(0.093)	-0.034	(0.093)	0.358***	(0.082)
×Female	-0.041	(0.101)	-0.353***	(0.106)	-0.251***	(0.077)
×Age	0.103**	(0.048)	0.164***	(0.052)	0.079**	(0.038)
×Salaried	0.221	(0.14)	0.002	(0.135)	0.001	(0.091)
×Union	0.283**	(0.136)	0.684***	(0.113)	-0.22*	(0.124)
Demographics×Generic						
Female	0.958***	(0.095)	1.169***	(0.139)	1.504***	(0.06)
Age	0.363***	(0.046)	0.502***	(0.063)	0.345***	(0.031)
Age^2	-0.174***	(0.051)	-0.047	(0.065)	0.222***	(0.032)
HMO	0.277	(0.181)	0.077	(0.19)	-0.627***	(0.121)
POS	-0.229	(0.198)	-0.5**	(0.247)	-0.86***	(0.13)
PPO	0.518***	(0.161)	0.019	(0.165)	-0.057	(0.105)
Salaried	-0.574***	(0.133)	-0.626***	(0.146)	-0.876***	(0.085)
Hourly	-1.099***	(0.126)	-0.482***	(0.152)	-1.424***	(0.089)
Union	1.148***	(0.144)	0.929***	(0.148)	1.132***	(0.104)
Non-Union	1.079***	(0.125)	0.282**	(0.119)	0.599***	(0.082)
Full-Time	-0.347***	(0.117)	-0.512**	(0.23)	-0.467***	(0.076)
MDD	0.487***	(0.066)	1.022***	(0.065)	2.678***	(0.047)
Depression-Other	0.662***	(0.078)	1.095***	(0.073)	3.489***	(0.066)
Demographics×Branded						
Female	1.106***	(0.142)	0.76***	(0.136)	2.942***	(0.108)
Age	0.132*	(0.08)	-0.355***	(0.076)	0.228***	(0.051)
Age^2	-0.066	(0.058)	-0.993***	(0.058)	-0.057	(0.037)
HMO	-0.254	(0.205)	0.062	(0.213)	-1.142***	(0.149)
POS	-0.151	(0.219)	0.461**	(0.229)	-0.639***	(0.158)
PPO	1.232***	(0.189)	0.58***	(0.191)	0.931***	(0.131)
Salaried	-0.114	(0.206)	-1.173***	(0.199)	-0.559***	(0.148)
Hourly	-0.571***	(0.141)	0.371***	(0.138)	-1.046***	(0.101)
Union	0.971***	(0.204)	1.139***	(0.205)	2.419***	(0.161)
Non-Union	1.29***	(0.13)	0.336***	(0.123)	0.821***	(0.092)
Full-Time	0.066	(0.137)	-0.262**	(0.133)	-0.827***	(0.096)
MDD	0.301**	(0.117)	1.32***	(0.118)	2.685***	(0.096)
Depression-Other	1.722***	(0.149)	3.04***	(0.156)	4.281***	(0.118)
Demographics×Therapy						
Female	1.134***	(0.134)	0.722***	(0.138)	1.616***	(0.098)
Age	-0.202***	(0.068)	-0.298***	(0.074)	-0.483***	(0.049)
Age^2	-0.144***	(0.041)	-0.36***	(0.044)	-0.266***	(0.029)
HMO	1.025***	(0.233)	0.331*	(0.193)	0.058	(0.146)
POS	1.456***	(0.223)	1.09***	(0.203)	0.791***	(0.147)
PPO	1.779***	(0.212)	0.893***	(0.182)	0.855***	(0.133)
Salaried	-1.008***	(0.206)	-0.596***	(0.197)	-0.098	(0.129)
Hourly	-0.706***	(0.116)	0.268**	(0.107)	-0.296***	(0.078)
Union	0.784***	(0.178)	0.159	(0.169)	0.044	(0.156)
Non-Union	0.841***	(0.103)	-0.366***	(0.102)	-0.241***	(0.073)
Full-Time	-0.668***	(0.101)	-0.465***	(0.109)	-1.206***	(0.085)
MDD	0.335***	(0.102)	0.267***	(0.1)	2.042***	(0.086)
Depression-Other	-0.589***	(0.135)	-0.353***	(0.128)	0.695***	(0.107)
Log-likelihood	-31,839.5		-31,927.2		-37,952.4	
Observations	445,500		445,500		445,500	

Notes: Bundle-MSA-month fixed effects included. Demographics are interacted with product dummies, OOP and OOP interactions are not. For the multiproduct bundles (not shown), the demographic effects equal the sum of the demographic effects on the component single-product bundles. OOP prices and age are measured in standard deviations ($24.52 and 10.62 years, respectively) and centered around their means ($26.70 and 44.85 years). Employees can be salaried, hourly, or unknown; unionized, non-unionized, or unknown; full-time, early retiree, part-time, or "other." Depression diagnosis can be major depressive disorder (MDD), dysthymia, or depression-other. The omitted health insurance type is CDHP/HDHP. ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$.

Table 1.13: Covariance/Correlation Matrix of Unobserved Preferences - Sensitivities

(1) Main / (2) No Spillovers			
	Generic	Branded	Therapy
Generic	8.64	0.34	0.25
Branded	4.06	16.25	0.44
Therapy	2.34	5.51	9.84

(3) Uncorrelated Preferences			
	Generic	Branded	Therapy
Generic	7.37	0	0
Branded	0	19.85	0
Therapy	0	0	7.98

(4) No Health Shocks			
	Generic	Branded	Therapy
Generic	22.03	0.89	0.73
Branded	23.84	32.21	0.76
Therapy	13.59	17.19	15.84

Notes: The first stage of the estimation is the same for the models with and without advertising spillover effects. Variance and covariance terms on the main diagonal and below; correlation coefficients—above. All parameter estimates are significant at the 1% significance level.

Table 1.14: Markov Chain Health Shock Transition Probabilities and Stationary Distribution - Sensitivities

(1) Main / (2) No Spillovers Panel A: Transition Probabilities		
	Healthy_{t+1}	Depressed_{t+1}
Healthy_t	0.9982	0.0018
	(0.0000)	(0.0000)
Depressed_t	0.0175	0.9825
	(0.0000)	(0.0000)
Panel B: Stationary Distribution		
	Healthy	Depressed
Long-run share	0.9066	0.0934

(3) Uncorrelated Preferences Panel A: Transition Probabilities		
	Healthy_{t+1}	Depressed_{t+1}
Healthy_t	0.9984	0.0016
	(0.0000)	(0.0000)
Depressed_t	0.0159	0.9841
	(0.0000)	(0.0000)
Panel B: Stationary Distribution		
	Healthy	Depressed
Long-run share	0.9066	0.0934

Note: Standard errors in parentheses. The first stage of the estimation is the same for the models with and without advertising spillover effects. The model with no health shocks has no transition probabilities and does not appear in the table.

Table 1.15: Second Stage, OLS Estimates - Sensitivities

	(1) Main	(2) No Spillovers	(3) Uncorr. Pref.	(4) No Health Shocks
Generic: Ads	-0.0026***		-0.0027***	-0.0012***
	(0.0006)		(0.0006)	(0.0004)
Branded: Ads	0.0059***	0.0062***	0.0073***	0.005***
	(0.0007)	(0.0007)	(0.0007)	(0.0005)
Therapy: Ads	0.0009		0.0011	0.001**
	(0.0006)		(0.0007)	(0.0005)
Time Effects	Year-Trend	Year-Trend	Year-Trend	Year-Trend
Market FEs	MSA	MSA	MSA	MSA

Notes: Standard errors in parentheses. For the multiproduct bundles, the advertising effects equal the sum of the effects on the component single-product bundles. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

Table 1.16: Second Stage, 2SLS Estimates - Sensitivities

	(1) Main	(2) No Spillovers	(3) Uncorr. Pref.	(4) No Health Shocks
Panel A: average complementarity				
Generic-Therapy	-0.1939***	-0.1939***	0.5424***	0.6883***
	(0.0113)	(0.0113)	(0.0113)	(0.0084)
Branded-Therapy	-0.7716***	-0.7716***	0.7139***	0.2379***
	(0.0124)	(0.0124)	(0.0128)	(0.0094)
Panel B: advertising effects on each bundle				
Generic: Ads	0.0053***		0.0053***	0.0039***
	(0.0025)		(0.0024)	(0.0016)
Branded: Ads	0.0072***	0.0096***	0.0076***	0.0048***
	(0.003)	(0.0028)	(0.0031)	(0.0021)
Therapy: Ads	0.0071***		0.0068***	0.0036***
	(0.0027)		(0.0028)	(0.002)
Time Effects	Year-Trend	Year-Trend	Year-Trend	Year-Trend
Market FEs	MSA	MSA	MSA	MSA
Panel C: first stage of instrumental variables regression				
Adj Avg Price per Ad			-0.0057	
Partial F-stat			504.8	

Notes: Standard errors in parentheses. For the multiproduct bundles, the advertising effects equal the sum of the effects on the component single-product bundles. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

Table 1.17: Demand Elasticities with Respect to Advertising, Sensitivity

	(1) Main	(2) No Spillovers	(3) Uncorr. Pref.	(4) No Health Shocks
		Bundles		
Outside Option	-0.0053	-0.0016	-0.0053	-0.0034
Generic-Only	0.0033	-0.0016	0.0033	0.0642
Branded-Only	0.0063	0.0138	0.0070	0.0797
Therapy-Only	0.0061	-0.0016	0.0057	0.0583
Generic-Therapy	0.0147	-0.0016	0.0142	0.1259
Branded-Therapy	0.0177	0.0138	0.018	0.1414
		Products		
Outside Option	-0.0053	-0.0016	-0.0053	-0.0034
Generic	0.0050	-0.0016	0.0051	0.0654
Branded	0.0076	0.0138	0.0089	0.0805
Therapy	0.0093	-0.0004	0.0097	0.0613

Note: Demand elasticities with respect to branded drug advertising are calculated by averaging individual-level elasticities over patients and time periods.

Chapter 2

Did Plain-Vanilla Prescription Drug Monitoring Programs Reduce Opioid Use? Evidence from Privately Insured Patients *

2.1 Introduction

This paper estimates the effect of prescription drug monitoring programs without registration or use requirements, or "plain-vanilla" PDMPs, on the use of opioid pain medications.[1] Multiple such state-run programs were established in the 2000s in

*Truven Health Analytics, through its Dissertation Support Program, generously made available for the purposes of this dissertation the Truven Health MarketScan® Commercial Claims and Encounters Database. Copyright © 2016 Truven. All Rights Reserved. Truven Health was not involved in preparing the results in this paper in any way. I am solely responsible for any errors.

[1]I use the adjective "plain-vanilla" to reflect the fact that this type of PDMPs did not have registration or access mandates, unlike programs that were introduced or modified after 2010. In

response to the growing opioid epidemic. They collected controlled-substance prescription drug information and provided it to physicians and pharmacists who could review it before prescribing or dispensing drugs. Because registration into and use of these programs was optional, many were skeptical of their ability to lower opioid use and misuse. I fill an important gap in the understanding of PDMPs by carefully evaluating their effect on opioid utilization.

To measure opioid use, I use the Truven Health Marketscan® Commercial Claims and Encounters Database for the period 2008–2010. The database contains all medical and prescription drug claims for individuals with employer-provided health insurance for a convenience sample of companies. This allows me to study opioid utilization by working age adults, the population that was hit the hardest by the opioid crisis.[2]

I take advantage of the staggered introduction of PDMPs in some states but not others and use the difference-in-differences technique to estimate the causal effect of these programs on opioid utilization. To minimize the chance that the "treated" and "control" states have different pre-trends, I select only those states that passed a law authorizing the creation of a PDMP after 2010. The reason for this choice is that states that establish PDMPs before and after 2010 are otherwise similar, but the timing of their PDMPs differs for idiosyncratic reasons. This provides greater credibility of the parallel trends assumption.

The results of my preferred specification indicate that, despite their use being

addition to mandates, later PDMPs were augmented with the ability to send unsolicited reports to health care providers, more frequent data reporting, improved user interface, and integration with electronic health records, among other features (Pew Charitable Trusts, 2016).

[2]Jones (2013) notes that the opioid overdose death rate in 2010 was the highest among 45-54 year-olds, closely followed by 35-44 and 25-34 year-olds. The overdose death rate among those younger than 18 or older than 65 was much lower.

optional, plain-vanilla PDMPs were successful in reducing opioid utilization. On average, these programs lowered the number of prescriptions per capita by 2.8%, days supply per capita by 3.8%, and morphine milligram equivalent (MME) per capita by 8.3%. The numbers suggest that although PDMPs brought about a relatively modest decrease in the number of prescriptions, they were more successful in lowering the intended length of treatment and the intensity of treatment. These findings are important from a public-health point of view because pills from prescriptions that provide a longer-term supply are often diverted, fuelling drug misuse, and because high-dosage prescriptions are more likely to lead to overdose (Dowell et al., 2016a).

I perform a variety of robustness checks to make sure that the results are not caused by a confounding factor. First, parallel trends tests do not reveal differences in the trends of treated and control states prior to the introduction of PDMPs. Second, falsification tests are consistent with PDMPs having no effect on outcomes they are not supposed to affect, namely, non-controlled substance prescription drugs and medical procedures unrelated to alcohol or drug abuse. These findings suggests that the results were not caused by broader health care trends that were different in treated and control states. Third, the fact that the Marketscan data track all prescription drug claims of an employee, regardless of the state in which they were filled, alleviates concerns that out-of-state purchases may bias the results. Fourth, I show that various other anti-opioid policies, such as laws governing pain clinics, access to naloxone, and the use of marijuana for medical purposes, are not confounding the difference-in-differences analysis.

Fifth, I carefully study the effect that attrition has on my estimates. Attrition oc-

curs naturally in the Marketscan dataset because it tracks individuals only for as long as they work for one of the companies contributing data. If attrition is correlated with unobservable determinants of opioid use and if these unobserved determinants change differently in treated and control states, this may bias the difference-in-differences results. To address such concerns, I include demographics as covariates in my main specification to control for the changing composition of the sample. I also run a sensitivity test using only individuals who do not attrit for the entire three-year period. The estimated PDMP effects are similar to those of the main specification. However, to further analyze whether attrition is biasing the estimation, I estimate a Cox proportional hazards model that shows that even though heavy users of opioids attrit more, they do not attrit more in states and time periods with active PDMPs. Based on the results of all attrition-related sensitivities, I conclude that attrition is unlikely to bias my main results.

The following subsection surveys the findings and shortcomings of the existing literature on PDMPs. Section 2.2 provides a brief history of the opioid epidemic and PDMPs. Those familiar with this history can skip to Section 2.3, which describes the data and the main patterns in it. Section 2.4 describes the identification strategy and results, while Section 2.5 performs a variety of robustness checks. Section 2.6 concludes.

2.1.1 Literature Review

This paper contributes to the literature on the impact of PDMPs on opioid utilization and health outcomes by providing credible causal estimates of the effect of "plain-vanilla" PDMPs. Many of the prior studies of these programs are correlational in nature and their findings vary significantly. Meara et al. (2016) and Brady et al. (2014) find no association between PDMPs and opioid utilization, whereas Reifler et al. (2012), Moyo et al. (2017), Rutkow et al. (2015), and Bao et al. (2016) find a negative association between PDMPs and prescription rates and, in some cases, dosage per prescription. With respect to health outcomes, findings are even more dispersed: Paulozzi et al. (2011) find no correlation between PDMPs and opioid-related overdose deaths, Li et al. (2014) find a positive correlation, and Patrick et al. (2016) find a negative one.[3] Mallatt (2017) and Yarbrough (2018) provide two studies that use difference-in-differences to estimate the causal effect of PDMPs among Medicaid and Medicare beneficiaries, respectively. Both studies find negative effects on opioid prescribing rates but do not distinguish between plain-vanilla and augmented PDMPs, which leaves the question of the effectiveness of the earlier PDMPs open.

There is stronger evidence on "augmented" PDMPs. PDMP Center of Excellence (2014) and Pew Charitable Trusts (2016) summarize the evidence on the effectiveness of PDMPs overall as well as that of aspects of PDMPs such as prescriber registration and use mandates, delegation, data timeliness, and sending unsolicited reports to stakeholders. Mandatory registration and use requirements have attracted the

[3]Haegerich et al. (2014) and Finley et al. (2017) provide surveys of the existing literature on PDMPs and note that most of the studies are correlational and fail to credibly identify a causal effect.

most attention. Using difference-in-differences analyses, Dowell et al. (2016b), Buchmueller and Carey (2018), Haffajee et al. (2018), Ayres and Jalal (2018), Wen et al. (2017), and Wen et al. (2019) show that "must-access" PDMP significantly reduce opioid prescription rates.[4] In addition, these studies find causal evidence that mandatory access requirements lead to reductions in doctor and pharmacy shopping, opioid misuse, inpatient and emergency room visits, and overdose deaths for a variety of populations, including those covered by Medicare, Medicaid, and private insurance.[5] Focusing specifically on health outcomes, Grecu et al. (2019), Pardo (2017), and Guy and Zhang (2020) find that PDMPs with mandatory use clauses lead to significantly lower opioid abuse, inpatient stays, emergency department visits, and opioid-related deaths.

Bao et al. (2018) provide further evidence that augmenting PDMPs with policies requiring mandatory use, delegation, and participation in interstate data exchanges strengthens the negative effect of basic PDMPs on risky prescribing and doctor shopping. Young et al. (2018) and Sacarny et al. (2016) show, however, that other PDMP features, such as unsolicited notifications to prescribers either about a patient's doctor shopping behavior or about the physician's own prescribing patterns, are not effective at decreasing opioid utilization.

[4]Contrary to most other analyses, Sun et al. (2018) find no statistically significant effect on either the opioid prescription rate or the dosage per prescription. Their study, however, differs in that it takes advantage of the staggered introduction of automatic PDMP queries in emergency departments in Washington state, rather than a typical multi-state difference-in-differences analysis, to evaluate the impact of this program.

[5]Strickler et al. (2019) use the comparative interrupted time series approach to show that mandatory registration and use policies increase the use of PDMPs by prescribers and decrease the rate of opioid prescribing and doctor shopping.

2.2 Background

On October 26, 2017, the President of the United States declared the opioid crisis a public health emergency. The pronouncement came after nearly two decades of increasing numbers of drug overdoses and nearly half a million deaths, 60% of which involved an opioid (Centers for Medicare and Medicaid Services, 2020). The economic cost of opioid abuse, including health care, mortality, productivity, and criminal justice costs, were estimated to be $55.7 billion in 2007 (Birnbaum et al., 2011) and $170.9 billion in 2017 (Davenport et al., 2019).[6] This section explains what opioids are, how attitudes toward pain management changed over time, how the opioid crisis unfolded, and how prescription drug monitoring programs were used to address the crisis.

2.2.1 Opioids, Pain Management, and the Opioid Epidemic

Opiates are drugs derived directly from opium, the dried latex of the opium poppy (Rosenblum et al., 2008). They act by binding to opioid receptors and inducing an analgesic, or pain-numbing, effect. Morphine, codeine, and thebaine are naturally occurring opiates which can be extracted from the poppy plant. Semi-synthetic opiates, such as heroin, oxycodone, hydrocodone, oxymorphone, and hydromorphone, are synthesized from naturally occurring opiates using simple chemical manipulations. "Opioid" is a more general term that designates any substance that binds to

[6]The two numbers are not directly comparable because Davenport et al. (2019) include child and family assistance and education costs as well. However, Davenport et al. (2019) estimate that even between 2015 and 2018 the economic cost of opioid abuse increased substantially–from $124.3 to $179.4 billion.

opioid receptors. It encompasses naturally occurring and semi-synthetic opiates as well as synthetic opioids. The latter include compounds that have chemical structure unrelated to that of morphine but nevertheless bind to opioid receptors and have opiate-like properties. Methadone, meperidine, tramadol, and fentanyl are the most widely known synthetic opioids, although there are many others as well.[7] Some of these substances are better known under their names as branded prescription drugs: Vicodin (hydrocodone with acetaminophen), OxyContin (oxycodone), and Percocet (oxycodone with acetaminophen).

Opioids have had a long and controversial history in the United States.[8] Morphine was used for pain management during the Civil War. Continued use and abuse by veterans led to restrictions on its use in the late 19th and early 20th century. These restrictions were soon followed by the rise of heroin and, once its addictive and deleterious properties became widely known, by more legislation curbing its use, the Harrison Act of 1914 and the Heroin Act of 1924. Over the following years, attitudes of physicians and patients shifted against the use of opiates for pain management. Even cancer patients were encouraged to postpone opioid treatment as long as possible, whereas other pain sufferers were discouraged from using opioids altogether.

This "opiophobia" continued well into the second half of the 20th century. Atti-

[7]In addition to how closely related they are to naturally occurring opiates, opioids are also categorized based on their effect on opioid receptors. Agonists induce a full response after binding to a receptor, partial agonists induce a partial response, while antagonists produce no response and block agonists from binding with the receptors (Pathan and Williams, 2012). Opioid agonists are used for pain management (and illegally for recreational purposes), partial agonists (buprenorphine) is used in medication-assisted treatment of opioid use disorder, while antagonists (naloxone) are used in reversing opioid overdoses.

[8]Bernard et al. (2018), Baker (2017), Jones et al. (2018), Alam and Juurlink (2016), Wilkerson et al. (2016), and Rummans et al. (2018) provide a good overview of the changing attitudes toward pain management and the opioid crisis in the United States. Schiff Jr (2002) reviews the history of opium since ancient times.

tudes started to change again when physicians began to point out the under-treatment of pain in the 1970s and 1980s. Backed by two small (and, subsequently, highly contentious) studies that claimed that addiction rarely arises from therapeutic use of opiate analgesics, the consensus shifted and opioids began to be used for the treatment of cancer and acute pain much more freely in the 1990s (Jones et al., 2018). Given the success of opioids in treating cancer pain, proponents started advancing their use for chronic pain and eventually succeeded (Bernard et al., 2018). The American Pain Society declared pain "the fifth vital sign" to highlight the need for better pain management. This concept was soon after adopted by the Veterans Health Administration and the Joint Commission on Accreditation of Healthcare Organizations.[9] As a result, pain management became an important component of patient satisfaction scores, a proxy for patient experience in hospitals, creating incentives for overly-aggressive pain treatment (Rummans et al., 2018). This shift was further facilitated by a lower level of scrutiny on opioid prescribers by the DEA and local authorities and by aggressive marketing on the part of pharmaceutical companies (Jones et al., 2018).[10]

The pendulum had swung from under- to over-treatment of pain. The end of the 1990s marked the beginning of the opioid crisis, which progressed in three waves. The first wave lasted from 1999 to 2010. As illustrated in Figure 2.1, it was characterized by a 3.5-fold increase in the natural and semisynthetic opioid overdose death rate

[9]The Joint Commission certifies hospitals for Medicare reimbursement.

[10]Purdue Pharma got FDA approval for OxyContin, controlled-release oxycodone, in 1995 and marketed the drug aggressively over the years. Alpert et al. (2019) provide evidence that Purdue's strategic promotional efforts led to higher growth rates in opioid overdose deaths.

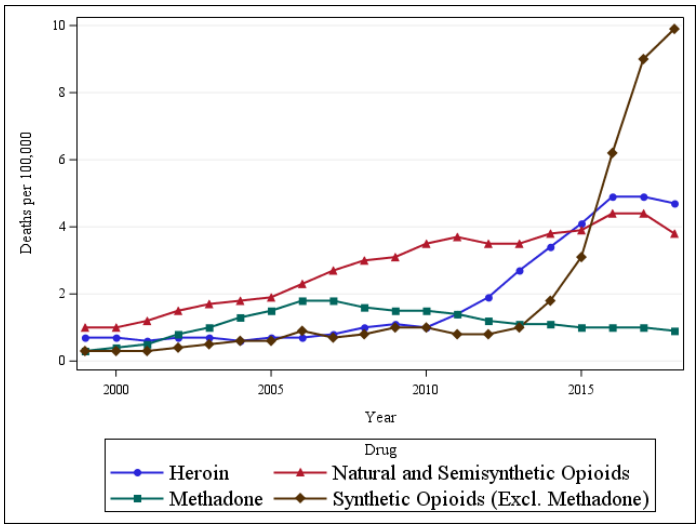


Figure 2.1: Opioid Overdose Death Rates, 1999-2018

Notes: Natural and semisynthetic opioids include drugs such as morphine, oxycodone, and hydrocodone. Synthetic opioids other than methadone include drugs such as fentanyl, fentanyl analogs and tramadol. *Source*: Centers for Disease Control and Prevention (2020).

– from 1 to 3.5 overdose deaths per 100,000 – after which it levelled off.[11] These deaths were caused by both prescription and non-prescription use of legal opioid pain relievers such as morphine, oxycodone, and hydrocodone (Rummans et al., 2018).

The second waive was marked by a nearly 5-fold increase in the heroin overdose death rate between 2010 and 2016. As various policies were introduced to curb the spread of opioid prescription drugs, addicted individuals substituted to heroin, an illegal opiate twice as potent as morphine. Alpert et al. (2017) and Evans et al. (2019) provide evidence that one of these policies, the introduction of abuse-deterrent OxyContin, led to large increases in heroin use and related overdose deaths.

Illegally manufactured fentanyl was the drug that defined the third, still ongoing, wave of the opioid epidemic. Between 2013 and 2018 the fentanyl-related overdose death rate shot up from 1 to 9.9 per 100,000.[12] Fentanyl is 50-100 times more potent

[11] This equates to an increase from 2,749 to 10,943 in the number of overdose deaths per year.

[12] Fentanyl is a DEA Schedule II drug and has legitimate medical uses such as inducing anesthesia

than morphine. It can also be synthesized in a lab, without the need to grow poppies and harvest opium as is the case with heroin. These two characteristics make it cheap and easier to smuggle, which is why fentanyl is increasingly popular with drug traffickers (Drug Enforcement Administration, 2019). It is pressed into counterfeit oxycodone, hydrocodone, and alprazolam (Xanax) pills and mixed with heroin, cocaine, and methamphetamines, often unbeknownst to the users, which contributes to its lethality. Powell and Pacula (2020) provide evidence that the introduction of abuse-deterrent OxyContin led to long-term increases in overdose deaths related not only to heroin but to fentanyl and cocaine as well.

2.2.2 History and Evolution of Prescription Drug Monitoring Programs

In response to the opioid crisis, states and the federal government took various steps aimed at prevention and treatment. One of these measures was the implementation of prescription drug monitoring programs. A PDMP is a state-run database that collects controlled-substance prescription information from pharmacies and dispensing providers and distributes it to physicians, pharmacists, state licensing boards, and, in certain situations, law enforcement.[13]

and treating cancer and other acute pain. As reported by Centers for Disease Control and Prevention (2017), he number of prescriptions for fentanyl, typically in the form of transdermal patches and lozenges, were stable or declining between 2013 and 2015, which suggests that illegally manufactured fentanyl is behind the rise in overdose deaths.

[13]The Controlled Substances Act of 1970 provides the legal framework for government oversight of certain substances. Schedule I drugs, such heroin, LSD, and marijuana, have no medical use and high potential for abuse. Controlled substances with medical use are in Schedule II (high potential for abuse; e.g., oxycodone, fentanyl, morphine) through Schedule V (low potential for abuse; e.g., cough syrups with a low dose of codeine).

Although these programs became widely known and adopted during the opioid epidemic, some of them had been in operation for a long time. The first PDMP in the United States was established in New York in 1918 in response to growing public health problems associated with the unregulated prescribing of opiates (PDMP-TTAC, 2018). Although it existed for only three years, it served as a blueprint for the ones that followed it: California in 1939, which is the oldest continuously operating PDMP, and eight more over the next 50 years.[14] These programs used duplicate (one copy for the pharmacy, one for the PDMP) or triplicate (one copy for the prescriber as well) prescription forms to track the distribution and use of Schedule II substances. The purpose of these early PDMPs was to assist law enforcement investigations and deter drug diversion by creating an audit trail (GAO, 2002). They typically operated by responding to requests for information rather than by proactively referring suspicious patient, prescriber, or dispenser behavior to law enforcement or licensing boards.

The arrival of electronic transmission of data facilitated more timely reporting as well as the wider adoption of PDMPs by obviating the need to manufacture, sell, distribute, fill out, and mail paper duplicate and triplicate forms. Most of the programs established in the 1990s and later years mandated electronic transfer of prescription data.[15] With the help of electronic reporting and the Internet, the focus of PDMPs shifted towards providing physicians and pharmacists with direct access

[14]Hawaii introduced a PDMP in the 1940s, Illinois and Idaho–in the 1960s, Pennsylvania, New York, and Rhode Island–in the 1970s, and Texas and Michigan–in the 1980s.

[15]Six of the seven PDMPs established in the 1990s and seventeen of the eighteen PDMP initiated in the 2000s required electronic reporting. By mid-2017, all states and the District of Columbia had functioning programs with electronic reporting and online access.

to patients' prescription drug histories. Online access, introduced for the first time in the late 1990s, allowed prescribers and dispensers to get instantaneous access to patients' records, removing the need to put in requests with the PDMP and wait several hours or days for the record to arrive by fax, further improving detection of opioid misuse and doctor shopping behavior (GAO, 2002).

Table 2.1 presents the timing of PDMP implementation, as well as the introduction of electronic reporting, user access, and online access. Under the pressure of having to counteract the opioid crisis, these programs continued evolving and acquiring new features: mandatory registration and use by prescribers and pharmacists, cross-border information sharing, high frequency reporting, proactive notifications, and integration into electronic health records (Pew Charitable Trusts, 2016). Most of these improvements, however, came after 2010, which allows me to evaluate the impact of "plain-vanilla" PDMPs using data for 2008-2010 described in the next section.[16]

[16]The timing of some of these additional PDMP features is discussed in Section 2.5.4.

Table 2.1: PDMP Introduction and Modification Dates

State	Law Passed	Operational	e-Reporting	User Access	Online Access
PDMPs Operational Before 2008					
California	01/1939	01/1939	01/2007		09/2009
Hawaii	01/1943	01/1943	01/1992		01/1997
Idaho	01/1967	01/1967	01/2004	06/1999	06/1999
Illinois	01/1961	01/1968	01/2000	01/1984	01/2008
Pennsylvania	01/1972	01/1973		01/2016	01/2016
New York	01/1972	04/1973	01/1999	02/2010	
Rhode Island	01/1978	01/1979	01/2006		07/2012
Texas	09/1981	01/1982	01/2001	01/1982	06/2012
Michigan	01/1988	01/1989	01/2003		01/2003
Oklahoma	05/1990	01/1991	01/1991		07/2006
Massachusetts	01/1992	01/1994	01/1994		01/2011
West Virginia	07/1995	07/1995	09/2002		12/2004
Utah	01/1995	01/1996	01/1996	01/1997	01/1997
Nevada	06/1995	01/1997	01/1997	07/1997	07/1997
Indiana	01/1997	01/1998	01/1998		12/2004
Kentucky	07/1998	01/1999	01/1999	07/1999	03/2005
Virginia	04/2002	09/2003	09/2003	06/2006	06/2006
Maine	06/2003	07/2004	07/2004	01/2005	01/2005
Wyoming	03/2003	07/2004	07/2004	10/2004	10/2004
Mississippi	01/2005	01/2005	01/2008	12/2005	12/2005
New Mexico	07/2004	01/2005	01/2005	08/2005	08/2005
Alabama	05/2005	01/2006	01/2006	06/2007	06/2007
Ohio	05/2005	07/2006	07/2006	10/2006	10/2006
Tennessee	01/2003	12/2006	12/2006	01/2007	01/2007
Colorado	06/2005	07/2007	07/2007	02/2008	02/2008
North Carolina	08/2005	07/2007	07/2007	10/2007	10/2007
North Dakota	12/2005	09/2007	09/2007	09/2007	09/2007
PDMPs Operational Between 2008 and 2010					
South Carolina	06/2006	02/2008	02/2008	09/2008	09/2008
Connecticut	06/2006	07/2008	07/2008		
Arizona	09/2007	10/2008	10/2008	12/2008	12/2008
Louisiana	07/2006	11/2008	11/2008	01/2009	01/2009
Iowa	05/2006	01/2009	01/2009	03/2009	03/2009
Vermont	05/2006	01/2009	01/2009	04/2009	04/2009
Minnesota	07/2007	01/2010	01/2010	04/2010	04/2010
PDMPs Operational After 2010					
Kansas	07/2008	02/2011	02/2011	04/2011	04/2011
Nebraska	04/2011	04/2011	04/2011	04/2011	04/2011
Oregon	07/2009	06/2011	06/2011	09/2011	09/2011
Alaska	09/2008	08/2011	08/2011	01/2012	01/2012
Florida	06/2009	09/2011	09/2011	10/2011	10/2011
New Jersey	01/2008	09/2011	09/2011	01/2012	01/2012
Washington	07/2007	10/2011	10/2011	01/2012	01/2012
South Dakota	03/2010	12/2011	12/2011	03/2012	03/2012
Delaware	07/2010	03/2012	03/2012	08/2012	08/2012
Montana	07/2011	03/2012	03/2012	11/2012	12/2012
Arkansas	03/2011	03/2013	03/2013	05/2013	05/2013
Wisconsin	05/2010	04/2013	04/2013	06/2013	06/2013
Georgia	05/2011	07/2013	07/2013	07/2013	07/2013
Maryland	05/2011	08/2013	08/2013	12/2013	12/2013
New Hampshire	06/2012	09/2014	09/2014	10/2014	10/2014
District of Columbia	02/2014	08/2016	08/2016	10/2016	10/2016
Missouri	03/2016	04/2017	04/2017	04/2017	04/2017

Note: The table is sorted by the year-month in which a PDMP became operational. States in bold are the ones used in the preferred specification of analysis in Section 2.4. *Source*: PDMP-TTAC (2019).

2.3 Data

The primary source of data on opioid utilization is the Marketscan Commercial Claims and Encounters Database. It contains all medical and prescription drug claims, as well as demographic information, for individuals covered by private health insurance plans provided by over 100 large employers across the United States over the 2008-2010 period. Data are available for the 48 contiguous states, Alaska, Hawaii, and the District of Columbia. Because the number of covered individuals in Hawaii is very low, I drop it from the analysis.[17]

The number of individuals included in the Marketscan dataset changes over time for two reasons. First, employees come and go as part of the natural staff turnover at included companies. Second, entire firms start or stop contributing data to the Marketscan database, typically in January of each year. To limit the noise that these changes introduce, I use the cohort of enrollees that are in the data as of January 2008 and follow them until December 2010.

I focus on three main outcomes of interest: the number of opioid prescriptions, the days supply of medication, and the morphine milligram equivalent (MME), all per 1,000 adults aged 18-64.[18] These measures provide a perspective not only on the rate of prescribing but also on the "size" of the prescriptions and the intensity of treatment.[19] These dimensions of opioid use are important because large-supply

[17]On average across months, there are 983 covered individuals in Hawaii, compared to 4,100 in the second least-covered territory (Washington D.C.) and over 155,000 on average across all states.

[18]Health outcomes such as heroin or other opioid poisonings and opioid dependence and abuse hospital admissions are also of interest. However, the data on these measures are not suitable for difference-in-differences analysis, and it is therefore difficult to draw conclusions about the causal impact of PDMPs on them. I discuss these outcomes at greater length in Appendix 2.7.1.

[19]MME captures the potency of various opioids relative to morphine. For example, 1 mg of

prescriptions facilitate diversion, while high-dosage treatment increases the risk of overdose (Dowell et al., 2016a).

Each prescription drug claim in the Marketscan data identifies the therapeutic class of the prescribed drug and its strength, as well as how many prescriptions, days supply, and physical units ("pills"–typically tablets or capsules for opioids) were dispensed. Using the identity of the active ingredient, the strength, the number of pills, and the appropriate MME conversion factor, I calculate the MME for every opioid claim.[20] I then sum up the number of prescriptions, days supply, and MME to the state-month level and calculate outcome variables in per 1,000 adults aged 18-64 terms. This yields a dataset with 50 states and 36 months of data for each state.[21] To that, I add state-month demographics based on the demographic data for each enrollee.

oxycodone is equivalent to 1.5 mg of morphine, whereas 1 mg of codeine is equivalent to 0.15 mg of morphine. Sullivan et al. (2008) provide MME conversion factors for a wide variety of opioids.

[20]By number of prescriptions filled, the most popular opioid is hydrocodone (58.0% of all prescriptions), followed by oxycodone (19.8%), propoxyphene (8.1%), codeine (5.1%), morphine, fentanyl, methadone, and others.

[21]For ease of exposition, I call Washington D.C. a state.

Table 2.2: Descriptive Statistics, Opioid Use Measures and Demographics

	Mean	Std Dev	Min	25th Pctl	50th Pctl	75th Pctl	Max
Opioid Use Measures							
Prescriptions per 1k	52.9	12.3	18.8	46.7	51.8	58.1	107.2
Days Supply per 1k	750.8	222.3	197.8	600.7	753.0	880.8	1,719.6
MME per 1k	46,015.8	14,062.9	6,255.1	35,816.9	45,791.0	54,493.2	113,717.6
Demographics							
Enrollees	155,158	204,431	2,908	28,481	81,299	196,018	1,343,211
Female	0.533	0.025	0.466	0.520	0.532	0.537	0.632
Average Age	44.92	1.46	38.78	43.84	44.95	45.98	48.50
Age 18-34	0.227	0.038	0.128	0.196	0.223	0.254	0.424
Age 35-44	0.220	0.019	0.174	0.212	0.217	0.234	0.273
Age 45-54	0.280	0.018	0.187	0.269	0.280	0.291	0.355
Age 55-64	0.273	0.042	0.147	0.244	0.269	0.298	0.389
HMO Plan	0.210	0.179	0.004	0.079	0.162	0.277	0.604
POS Plan	0.139	0.115	0.003	0.054	0.128	0.173	0.703
PPO Plan	0.520	0.138	0.243	0.454	0.515	0.611	0.837
Full-time Employees	0.808	0.057	0.646	0.783	0.824	0.845	0.915
Salaried Employees	0.312	0.150	0.074	0.154	0.327	0.427	0.586
Hourly Employees	0.304	0.134	0.072	0.155	0.349	0.387	0.613
Mining & Manuf.	0.340	0.170	0.098	0.186	0.367	0.444	0.792
Transp., Comm., Util.	0.184	0.095	0.052	0.094	0.174	0.250	0.461
Retail and Services	0.156	0.106	0.011	0.077	0.133	0.181	0.610
Fin., Ins., Real Estate	0.103	0.073	0.009	0.047	0.093	0.142	0.463
Cancer per 1k	8.192	2.466	2.842	6.248	7.535	9.790	17.165
Pain per 1k	0.681	0.259	0.000	0.503	0.671	0.829	3.247
Fracture per 1k	1.791	0.471	0.000	1.447	1.651	2.009	5.408
Heart Disease per 1k	18.373	7.005	6.681	12.526	16.867	22.110	41.346
Diabetes per 1k	7.176	3.377	0.598	4.780	6.087	8.615	22.283
Mental Illness per 1k	1.274	0.589	0.000	0.951	1.171	1.460	4.868

Notes: Statistics based on all 1,800 state-months, weighted by number of enrollees. Opioid use measures and diagnoses are per 1,000 enrollees aged 18-64. Demographics except average age, number of enrollees, and diagnoses are shares. "MME" stands for morphine milligram equivalent. Other insurance plan types include comprehensive, consumer-driven, and high-deductible health plans. Employees can be full-time, part-time, or early retirees; salaried, hourly, or with unknown status. Other industries include agriculture, construction, wholesale, and unknown. Mental illnesses include major depressive and bipolar disorders, schizophrenia, and other nonorganic psychoses.

Table 2.2 summarizes the distribution of opioid utilization and demographic characteristics over all months, from January 2008 to December 2010, and geographic locations, Washington D.C. and all U.S. states except Hawaii. On average, there are 155,158 enrollees in a state-month, with more than three quarters of state-months

having more than 28,000, which provides a good basis for accurately measuring opioid utilization rates. 53% of the enrollees covered in the median state-month are female. The average age is 44.9, with each group of 18-34, 35-44, 45-54, and 55-64 year-olds accounting for approximately a quarter of the sample, although there is a fair amount of variation across states and time periods. The most widely used health insurance plan type is the preferred provider organization (PPO), which covers at least 24% of all enrollees in any state-month and 52% on average. It is followed by health maintenance organization (HMO) and point of service (POS), covering 21% and 14% on average, respectively. The rest of the enrollees are covered by a combination of high-deductible, consumer-driven, exclusive provider, and comprehensive health plans. 81% of enrollees are full-time employees, the rest being primarily part-time and early-retirees. Salaried and hourly employees each make up roughly 30% of the sample, with the payment arrangement for the rest being unknown. In the average state-month, 34% of enrollees are employed in the mining and manufacturing industries, which is substantially higher than the national average of 10.5% and reflects the convenience sample nature of the Marketscan data.

The data reveal substantial variation in the health status of the covered individuals across time and space. The average numbers of enrollees with heart disease, diabetes, and mental illness are 18.4, 7.2, and 1.3 per 1,000, respectively, with sizeable standard deviations.[22] The number of people who are likely to need pain treatment also varies

[22]For every state-month pair, I count the number of enrollees who have an inpatient or outpatient visit with a certain diagnosis. I use *International Classification of Diseases, 9^{th} Revision, Clinical Modifications* (ICD-9-CM) codes beginning with 393-398 and 40-43 to identify heart disease and beginning with 250 to identify diabetes. Mental illnesses include major depression, bipolar disorder, schizophrenia, and other nonorganic psychoses, which correspond to ICD-9-CM codes beginning with 295, 2962-2968, and 297.

significantly. On average, out of every 1,000 adults, between 2.8 and 17.2 receive diagnosis and treatment for cancer; between 0 and 3.2 are diagnosed with pain; and between 0 and 5.4 experience fractures.[23]

Overall health and pain-related diagnoses are some of the factors affecting opioid usage. Between January 2008 and December 2010, every 1,000 adult enrollees filled an average of 52.9 prescriptions for a total of 751 days' supply and 46,016 MME of opioid medication, which amounts to approximately 14 days' supply per prescription and 61 MME per day's supply. There is substantial variation in the opioid utilization measures, both across states and over time. Figure 2.2 shows average opioid utilization measures for three groups of states: those that instituted a PDMP between 2008 and 2010, those that did that after 2010 (and had no PDMP during 2008-2010), and those that already had a PDMP in place prior to 2008. All three graphs show a significant increase in the per-capita use of opioids, consistent with national trends for the period. The graphs also show variation across the groups based on PDMP timing.

Figure 2.2 shows that in the first six months of data, when there were no newly-introduced PDMPs, the rate of increase in monthly opioid use is approximately the same in the three groups of states. Over the next 18 months, six new PDMPs become operational: Connecticut (July 2008), Arizona (October 2008), Louisiana (November 2008), Iowa and Vermont (January 2009), and Minnesota (January 2010). Over this time period, utilization measures between the treatment group (the six states with

[23]Cancer incorporates all ICD-9-CM codes beginning with 14-20 and 230-234, pain – beginning with 338, and fractures – beginning with 80-82.

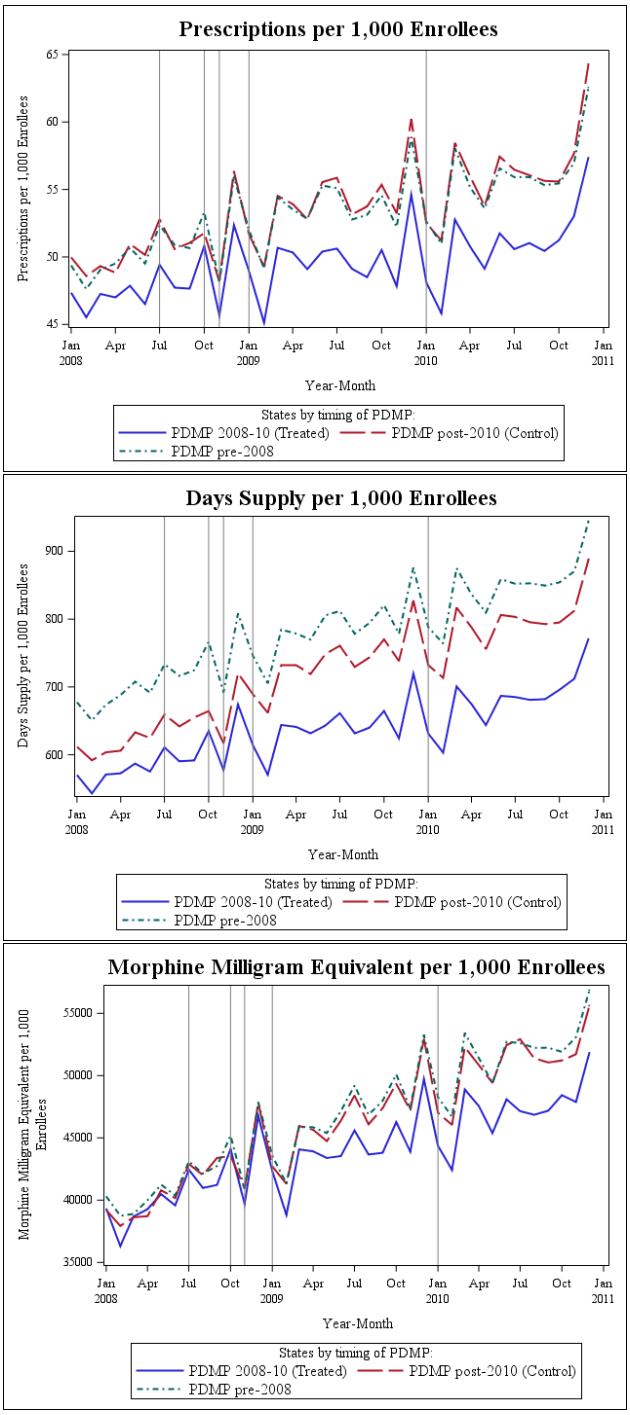


Figure 2.2: Opioid Utilization over Time

Notes: The statistics for each group of states are the averages over all states within a group, weighted by number of enrollees. The vertical lines indicate the months in which PDMPs became operational.

newly introduced PDMPs) and the other two groups of states start to diverge.

The divergence is captured in Table 2.3, which shows average utilization measures and demographics for the three groups of states for the periods January-June 2008 and January-June 2010.[24] The average number of prescriptions per 1,000 adult enrollees increased by 2.8 in treated states and by 5.3 in states with no PDMP in 2008-2010. The difference between the two differences, -2.5 (5.3% of the base value), is a rough estimate of the effect of introducing a PDMP on the number of opioid prescriptions per capita. Similar calculations suggest that PDMPs reduce days' supply and MME per capita by 12.3% and 8.4%, respectively.

Table 2.3 also shows demographic summary statistics for each group of states and for the "before" and "after" periods. At baseline, all three groups look fairly similar. The treated group has a lower share of enrollees on HMO plans and higher shares of salaried employees and employees in the mining and manufacturing industries than the other two groups. In a difference-in-differences analysis, these differences will affect the estimates only if the demographics change differently in treated and control states *and* if they are correlated with the outcome of interest. At first sight, it seems that the demographic changes are similar across groups: the share of female enrollees stays roughly constant, average age increases similarly across groups due to significant declines in enrollees aged 18-34. Cancer cases increase slightly more in treated than control states, but pain and fracture diagnoses increase slightly less. Visually examining the rest of the changes does not reveal significant differential

[24] I focus on January-June rather than January-December 2010 to partial out any seasonal effects. However, the patterns that emerge using January-December 2010 are very similar.

changes.

Table 2.3: Descriptive Statistics, By PDMP Timing Group, Before and After

	PDMP '08-'10		PDMP Post-'10		PDMP Pre-'08	
	1-6/08	1-6/10	1-6/08	1-6/10	1-6/08	1-6/10
Opioid Use Measures						
Prescriptions per 1k	46.9	49.7	49.6	54.9	49.3	54.5
Days Supply per 1k	569.5	656.4	611.6	768.3	681.3	821.7
MME per 1k	38,923.0	46,089.4	39,220.7	49,647.9	39,914.9	50,293.1
Demographics						
Enrollees	91,400	66,551	136,474	103,094	237,157	179,805
Female	0.506	0.507	0.540	0.543	0.531	0.534
Average Age	42.52	45.45	43.22	45.95	43.36	46.09
Age 18-34	0.296	0.212	0.272	0.194	0.273	0.194
Age 35-44	0.220	0.216	0.228	0.223	0.219	0.218
Age 45-54	0.265	0.297	0.263	0.293	0.262	0.288
Age 55-64	0.219	0.274	0.236	0.290	0.246	0.300
HMO Plan	0.132	0.103	0.223	0.148	0.233	0.215
POS Plan	0.183	0.124	0.139	0.112	0.154	0.131
PPO Plan	0.519	0.566	0.534	0.541	0.496	0.503
Full-time Employees	0.851	0.838	0.829	0.809	0.816	0.793
Salaried Employees	0.389	0.407	0.305	0.303	0.306	0.304
Hourly Employees	0.318	0.323	0.300	0.300	0.308	0.300
Mining & Manuf.	0.468	0.505	0.321	0.332	0.335	0.335
Transp., Comm., Util.	0.151	0.148	0.218	0.215	0.176	0.167
Retail and Services	0.129	0.165	0.105	0.083	0.183	0.179
Fin., Ins., Real Estate	0.149	0.148	0.120	0.112	0.101	0.093
Cancer per 1k	6.871	8.433	7.013	8.310	7.606	8.891
Pain per 1k	0.340	0.495	0.494	0.818	0.507	0.799
Fracture per 1k	1.753	1.557	1.626	1.562	1.863	1.790
Heart Disease per 1k	14.737	17.920	15.101	17.707	17.256	20.713
Diabetes per 1k	5.900	6.973	5.453	6.601	6.765	8.278
Mental Illness per 1k	1.210	1.235	1.065	1.150	1.336	1.395

Notes: All statistics except number of enrollees are weighted averages based on the first six months of 2008 and 2010. Opioid use measures and diagnoses are per 1,000 enrollees aged 18-64. Demographics except average age, number of enrollees, and diagnoses are shares. "MME" stands for morphine milligram equivalent. Other insurance plan types include comprehensive, consumer-driven, and high-deductible health plans. Employees can be full-time, part-time, or early retirees; salaried, hourly, or with unknown status. Other industries include agriculture, construction, wholesale, and unknown. Mental illnesses include major depressive and bipolar disorders, schizophrenia, and other nonorganic psychoses.

The largest changes occur in the number of enrollees. Approximately a quarter of enrollees drop out between the first half of 2008 and the first half of 2010, but

the share of drop-outs is similar for all three groups of states. While attrition does not necessarily affect the results of my analyses, changing demographics and the possibility that they are correlated with opioid use remain a concern and are explored in greater detail in the next section.

2.4 Empirical Strategy and Results

2.4.1 Difference-in-Differences Strategy and Choice of Control States

I take advantage of the staggered implementation of PDMPs and use a difference-in-differences identification strategy to estimate their effect on opioid use. Because there are multiple states that introduce PDMPs in the 2008-2010 period, I employ the generalized version of this empirical approach:

$$y_{it} = \delta_i + \delta_t + \beta D_{it} + \varepsilon_{it} \tag{2.1}$$

where y_{it} is the natural logarithm of the outcome variable, δ_i and δ_t are state and year-month fixed effects, respectively, D_{it} is an indicator variable that equals 1 if state i has an operational PDMP in month t, and ε_{it} is the error term. In this setup, the coefficient β captures the average treatment effect of a PDMP in percentage terms.

To control for attrition-induced changes in demographics, including pain-related diagnoses such as cancer, which might coincide with the introduction of the PDMPs,

I include demographic variables in my preferred specification:

$$y_{it} = \delta_i + \delta_t + \beta D_{it} + \mathbf{X}_{it}\gamma + \varepsilon_{it} \tag{2.2}$$

where $\mathbf{X}_{it}$ includes age and the shares of female enrollees, HMO, POS, PPO plans, full-time, salaried, hourly employees, and employees in each industry aggregation.

Introduction of PDMPs in some states but not others is not sufficient to ensure unbiased estimation. Treatment and control states need to be sufficiently similar. In particular, the trends in the outcome variables must be the same in the pre-intervention period. Since PDMPs were typically established to counteract the growing opioid epidemic, this may not be the case. If state lawmakers pass PDMP legislation based on the rate of increase in an outcome variable, then the parallel trends assumption will not hold and difference-in-differences estimation yield biased results.[25]

While visual inspection of Figure 2.2 suggests that outcome variable trends are the same in the pre-intervention period for the three groups of states, I also use a restricted set of control states to alleviate concerns about potentially non-parallel trends. Specifically, I use only those states that enacted PDMPs after 2010. The reasoning is that outcome trends for states that adopted right after 2010 are very similar to the trends of the 2008-2010 adopters, but idiosyncracies in the legislative and implementation processes could have led to different PDMP timing. Such idiosyncracies could be the amount of time health officials need to notice a problem with opioid abuse in their state and bring it up with lawmakers, the amount of time

[25]However, if state lawmakers introduce PDMPs based on the *level* of an outcome variable, then estimation is still unbiased.

legislators need to pass a law, and the amount of time it takes to set up the infrastructure for a PDMP. Further, to avoid bias from anticipation effects, I also drop states in which a PDMP law had been passed but the program had not become operational in 2008-2010.[26]

PDMPs that became operational prior to 2008 can also serve as a control group. However, I do not use them because of the possibility of dynamic effects. Bao et al. (2018) find that PDMP effects strengthen over time. If that is the case with pre-2008 PDMPs as well, these dynamic effects can bias estimation by making 2008-2010 PDMP seem less effective since the control group would be experiencing the effect of the older PDMPs. Given that 16 out of the 27 pre-2008 PDMPs either became operational or got modified (by introducing electronic reporting, initial user access, or internet access) in the three years before 2008, it is plausible that such dynamic effects are present.[27]

2.4.2 Results

Panel A of Table 2.4 shows the results from the difference-in-differences regressions using all states that did not experience a change in PDMP status between 2008

[26]Anticipation effects may arise if physicians reduce the number of opioid prescriptions they write after the PDMP law is passed but before the PDMP becomes operational. If this happens in control states, it will lead to an underestimation of the growth in opioid use without a PDMP, which will underestimate the magnitude of the PDMP effect. If it happens in treated states, it will underestimate the magnitude of the total (i.e. anticipation plus post-implementation) PDMP effect. By restricting the set of control states, I eliminate bias from anticipation in control states, although it is possible that the results still underestimate the total PDMP effect if there is anticipation in treated states.

[27]I explore a variety of alternative treatment and control groups in Appendix 2.7.2. These sensitivity analyses show that the conclusions drawn from the main specification used in the paper are robust and that using an extended set of control states may introduce bias due to anticipation or dynamic effects.

and 2010 as a control group. The estimates suggest that establishing an operational PDMP leads to a 2.0% decline in the number of opioid prescriptions per capita (albeit not statistically significant at conventional levels), a 2.3% decline in the days supply per capita, and a more sizeable 4.5% decline in the MME per capita. Thus, in addition to fewer opioid prescriptions being filled, they are also for a shorter duration and lower dosage, which is consistent with PDMPs reducing the highest-risk opioid prescribing. These results are in line with the rough estimates based on Figure 2.2 and Table 2.3, although of lower magnitude.

Table 2.4: Difference-in-differences Results

	$\hat{\beta}$	p-val	State FEs	Yr-Mth FEs	Demo-graphics	R^2	Obs
Panel A: All States, No Demographics							
Log(Presc. per 1k)	-0.0197	0.164	Yes	Yes	No	0.9850	1800
Log(Days Supp. per 1k)	-0.0231*	0.070	Yes	Yes	No	0.9880	1800
Log(MME per 1k)	-0.0446**	0.024	Yes	Yes	No	0.9716	1800
Panel B: States with Post-2010 PDMP Law as Control Group, No Demographics							
Log(Presc. per 1k)	-0.0362*	0.071	Yes	Yes	No	0.9774	504
Log(Days Supp. per 1k)	-0.0576**	0.031	Yes	Yes	No	0.9830	504
Log(MME per 1k)	-0.1053***	0.005	Yes	Yes	No	0.9678	504
Panel C: States with Post-2010 PDMP Law as Control Group, Demographics Included							
Log(Presc. per 1k)	-0.0281**	0.036	Yes	Yes	Yes	0.9820	504
Log(Days Supp. per 1k)	-0.0379*	0.056	Yes	Yes	Yes	0.9862	504
Log(MME per 1k)	-0.0834**	0.039	Yes	Yes	Yes	0.9777	504

Notes: Each *row* is a separate regression. An observation is a state-month pair in the period of 2008-2010. Dependent variables are natural log of opioid utilization measures per 1,000 adults aged 18-64. Included demographic variables are cancer, pain, and fracture diagnoses per 1,000 enrollees, as well as age, age squared, share female, HMO, POS, PPO plan type, full-time, salaried, hourly employee, employed in mining or manufacturing, transportation, communications, or utilities, retail trade or services, and finance, insurance, or real estate. Panel A shows p-values based on robust standard errors clustered at the state level. Panels B and C show p-values based on the wild-bootstrap procedure in Cameron et al. (2008). $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

The existence of multiple pre- and post-treatment periods in the data allows me to perform an event study. An event study compares the difference in outcomes between treated and control states in a focal period and a base period. The focal period can

be any lead or lag of the time period in which a PDMP was introduced. Event studies are useful as they allow the researcher to assess whether the parallel trends assumption holds and whether the policy's effect becomes stronger or weaker over time. I implement the event study by including leads and lags of ΔD_{it}, an indicator for the time period in which a state introduced a PDMP:

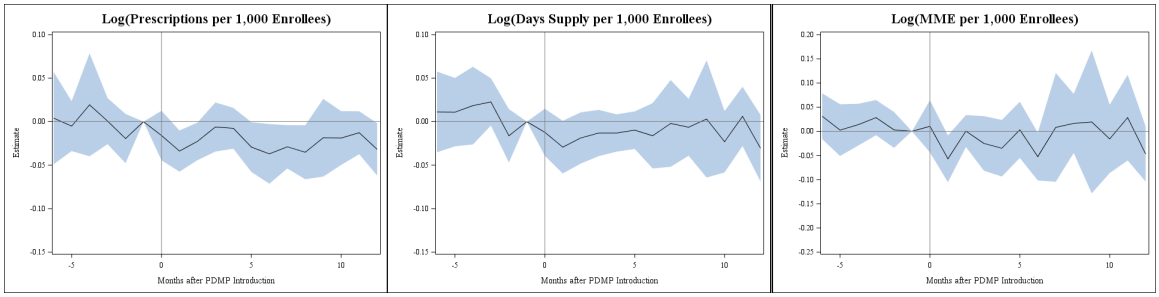
$$y_{it} = \delta_i + \delta_t + \sum_{s=-12}^{7} \beta_s \Delta D_{it+s} + \varepsilon_{it} \tag{2.3}$$

Because the first PDMP becomes operational in July 2008, I include six leads to keep the set of states on which the estimates of these six leads are based the same.[28] The seventh lead aggregates all leads of order seven and higher.[29] Based on analogous reasoning, since the last PDMP was introduced in January 2010, I include 11 lags and a twelfth aggregate lag. I drop the first lead to allow the period just before the introduction of a PDMP to be the base period.

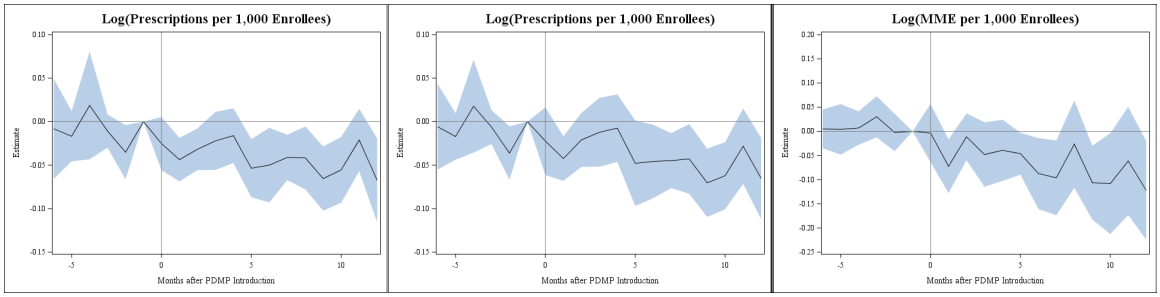
The idea of the parallel trends test is that if the assumption holds, the leads will be statistically indistinguishable from zero. Panel A of Figure 2.3 plots the estimated β_s coefficients of the event study for each outcome measure and their 95% confidence intervals, with leads to the left of the vertical reference line, which indicates the period of PDMP implementation. The leads are all statistically insignificant, which gives credibility to the parallel trends assumption. It is difficult to discern a clear upward or downward trend. To the extent that there might be a slight downward

[28] If I were to include a seventh lead, its estimate would be based on states that introduced a PDMP in August 2008 or later.

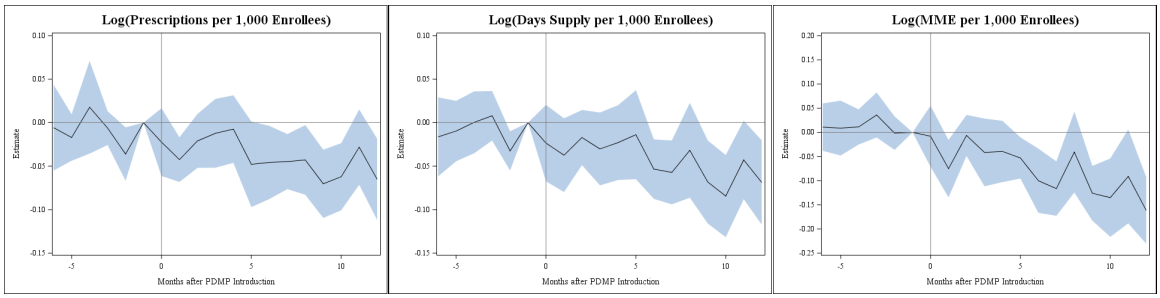
[29] The state composition of this seventh lead is not constant.



(a) Panel A: All States, No Demographics



(b) Panel B: States with Post-2010 PDMP Law as Control Group, No Demographics



(c) Panel C: States with Post-2010 PDMP Law as Control Group, Demographics Included

Figure 2.3: Pre-trends Tests

Notes: The month before a PDMP becomes operational is the base period. The band is a 95% confidence interval around each point estimate.

pre-trend in the plots for days supply and MME, this might reflect faster growing opioid use in control states. If that were the case, it would make PDMPs seem more effective at reducing opioid utilization than they are, i.e. there would be a negative bias in the difference-in-differences estimates in Panel A of Table 2.4.

To alleviate concerns about non-parallel pre-trends, I restrict the set of control states to only those that passed PDMP legislation after 2010. There are eight such states: Nebraska, Montana, Arkansas, Georgia, Maryland, New Hampshire, Wash-

ington DC, and Missouri. The estimates reported in Panel B of Table 2.4 indicate larger PDMP effects on all three outcome measures. To account for the small number of states (14) used in these analyses, I use p-values based on the wild-bootstrap procedure described in Cameron et al. (2008).[30] Even based on the wild-bootstrap p-values, all estimates are statistically significant at least at the 10% significance level. The estimated coefficients of the event studies, plotted in Panel B of Figure 2.3, reveal no clear pre-trend.[31]

The attrition-induced changing demographic composition of the sample is another issue that raises concerns about the unbiasedness of the estimates. If, for example, young people from treated states attrit more than their peers from control states and if young people consume more opioids per capita, then this form of differential attrition will lead to downward (negative) bias in the difference-in-differences estimate of the effectiveness of PDMPs, i.e. it will make PDMPs seem better at reducing opioid usage.[32] However, if attrition rates among the young are the same in treated and control states or if age is uncorrelated with opioid consumption, then attrition will not affect the results.

To account for the changes in the demographics of the sample, I include the number of cancer, pain, and fracture diagnoses per 1,000 adults, as well as age, age squared,

[30]The usual cluster-robust standard errors are an asymptotic result. Cameron et al. (2008) show that they tend to over-reject when the number of clusters is less than 30 and provide alternative ways to calculate standard errors that have better small-sample (in terms of number of clusters) properties. Unlike a typical bootstrap procedure, the wild bootstrap keeps the values of the regressors the same and creates new values of the dependent variable by resampling the residuals of the model and assigning them a weight of -1 or 1 with equal probability.

[31]Even though the coefficient on the second lead is statistically different from zero, a joint test for significance cannot reject the null hypothesis that all leads are jointly zero.

[32]Bias with the opposite sign will arise if the number of enrollees with cancer increase more in treated than in control states and if cancer patients use more pain medication. This will make it seem as if PDMPs are increasing opioid utilization.

shares of female enrollees, HMO, POS, PPO plan types, full-time, salaried, hourly employees, and shares of enrollees employed in mining or manufacturing, transportation, communications, or utilities, retail trade or services, and finance, insurance, or real estate as control variables. The results appear in Panel C of Table 2.4. The estimates are somewhat smaller in magnitude than those in Panel B, which suggests that differentially changing demographics might have introduced some bias in the estimates in Panel B. However, the results are qualitatively the same. In the period after a PDMP becomes operational opioid prescriptions per capita decline by 2.8%, days supply - by 3.8%, and MME - by 8.3%, consistent with PDMPs being particularly effective at reducing the highest-risk prescribing. Like Panel B, Panel C of Figure 2.3 shows no clear differential trends between treatment and control states prior to the introduction of PDMPs.

2.5 Robustness Checks and Sensitivities

To examine the robustness of the results of the previous section, I conduct multiple checks. As a starting point, I use the model with included demographics with states that passed a PDMP law after 2010 as a control group, i.e. the results in Panel C of Table 2.4.

2.5.1 Falsification Tests

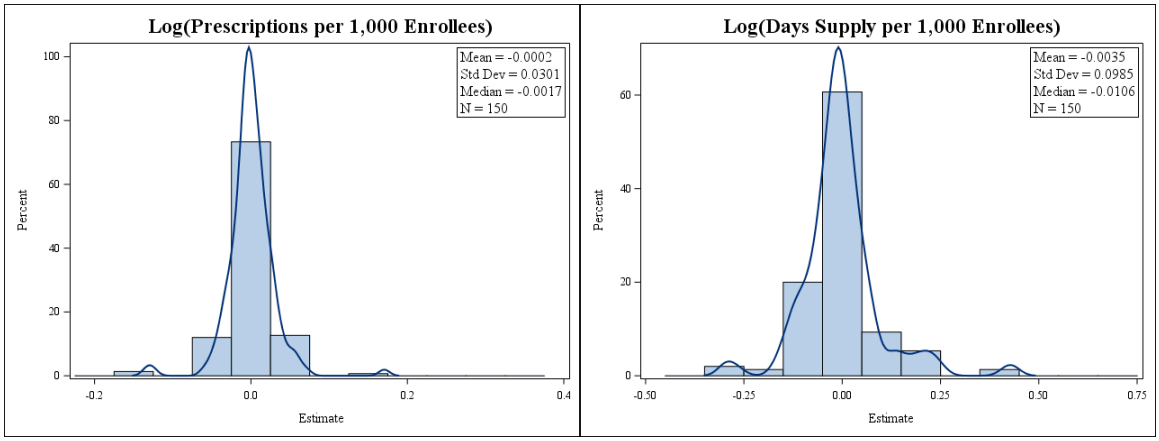
While the results of the previous section suggest that PDMPs had a causal effect on opioid utilization, an alternative explanation could be that these changes, lower

utilization of opioids after PDMP implementation in treated states relative to control states, reflect broader trends in the health care field in these states. Perhaps overall prescription drug or medical procedure utilization increased more slowly in treated states, and opioid use simply tracked these larger trends. If this were the case, a difference-in-differences analysis would find a similar negative effect of PDMPs on the utilization of non-controlled-substance drugs and medical procedures.

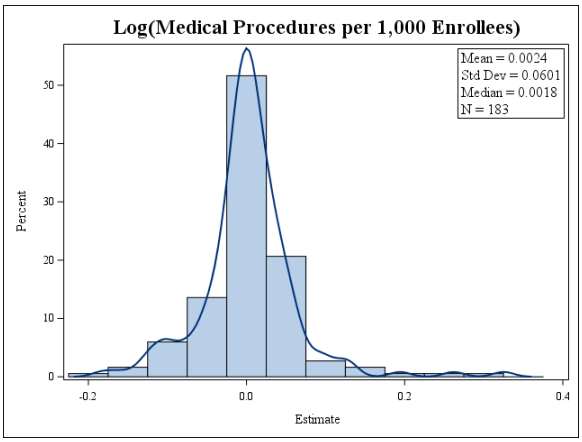
The richness of the Marketscan data allows me to test this hypothesis. Instead of focusing on any particular prescription drug, I take all non-controlled substances except for other pain medications (to avoid substitution effects), for the sample of enrollees used in the main analyses, aggregate the number of prescriptions and days supply up to the therapeutic class level, and perform a difference-in-differences using the preferred specification from the previous section.[33] I follow the same approach for medical procedures, excluding those related to alcohol or drug use and poisonings, except I calculate the number of procedures per capita instead of the number of prescriptions per capita.

There are 150 prescription drug therapeutic classes and 183 medical procedures groups that I perform difference-in-differences analysis on. The histograms of the estimates in Figure 2.4 show that they are centered fairly close to zero. The mean of the estimates using prescriptions per capita is -0.0002. 10 of these 150 estimates, or 6.7%, are statistically significant at the 10% significance level using wild-bootstrap inference as in Cameron et al. (2008). The mean estimate and share significant at 10% for prescription drugs days supply are -0.0035 and 10%, whereas the same for

[33]For non-controlled substances, I do not calculate MME because they are not opioids.



(a) (b)



(c)

Figure 2.4: Falsification Tests

Notes: Each panel shows a histogram of the estimated PDMP effects for each prescription drug therapeutic class (a, b) or medical procedure group (c).

medical procedures per capita are 0.0024 and 8.7%.

Note that if PDMPs had no effect on non-controlled substances, roughly 10% of the estimates would be statistically significant at the 10% significance level by chance– these would be the type I errors or false positives. Therefore, finding estimates that are centered at zero and that are significant roughly 10% of the time provides strong support to the notion that PDMPs did not coincide with general trends in health care that affected treated and control states differently. This conclusion is supported by a closer examination of the regression results for each therapeutic class and medical procedure group, available in Tables 2.11 and 2.12 in Appendix 2.7.3, reveals no pattern in the positive and negative results.[34]

2.5.2 Attrition

Table 2.3 reveals that a substantial number of enrollees drop out of the sample over time. To make sure that attrition is not driving the results, I perform two robustness checks. First, I re-estimate the model in Panel C of Table 2.4 using data from "stayers," i.e. enrollees that were present for the full length of the sample. This minimizes concerns about changes in observable and unobservable enrollee characteristics by focusing on the same individuals in each period. Second, I use individual-level data to estimate a Cox proportional hazards model to determine whether heavy use of opioids is associated with higher attrition in treated states.

[34]Interestingly, PDMPs seem to have an effect on the utilization of various treatments for constipation, which can be induced by opioid drugs. The use of stimulant and saline laxatives increases, while the use of enemas decreases. However, the net effect on all laxatives, aggregated into one category, is insignificant (zero for number of prescriptions, negative for days supply).

Table 2.5: Difference-in-differences Results, "Stayers" Only

	$\hat{\beta}$	p-val	State FEs	Yr-Mth FEs	Demo-graphics	R^2	Obs
Log(Presc. per 1k)	-0.0207	0.144	Yes	Yes	Yes	0.9709	504
Log(Days Supp. per 1k)	-0.0218	0.315	Yes	Yes	Yes	0.9829	504
Log(MME per 1k)	-0.0765	0.335	Yes	Yes	Yes	0.9606	504

Notes: Each *row* is a separate regression. An observation is a state-month pair in the period of 2008-2010. Dependent variables are natural log of opioid utilization measures per 1,000 adults aged 18-64 who were present for the full length of the sample period. Included demographic variables are cancer, pain, and fracture diagnoses per 1,000 enrollees, as well as age, age squared, share female, HMO, POS, PPO plan type, full-time, salaried, hourly employee, employed in mining or manufacturing, transportation, communications, or utilities, retail trade or services, and finance, insurance, or real estate. p-values are based on the wild-bootstrap procedure in Cameron et al. (2008). $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

The results of the difference-in-differences analysis using only enrollees that stay for the entire period are shown in Table 2.5.[35] The estimated effects of PDMPs on prescriptions, days supply, and MME per capita are somewhat smaller than those of the main specification. However, the qualitative interpretation of the results remains unchanged - PDMPs reduce the overall number of opioid prescriptions per capita but especially high-dosage prescriptions. The estimates are much less precise possibly because they are based on a lower number of enrollees.[36]

Although the results are qualitatively very similar, the differences in magnitude may raise some concerns. To investigate whether heavy users of opioids are more likely to drop out in treated states, I estimate a Cox proportional hazards model using individual-level data.[37] It models the hazard rate, i.e. the probability that an

[35]This analysis uses demographics as controls. However, given that the enrollees that make up the sample remain the same and that I include state and year-month fixed effects, there is no or very little residual variation in some of the demographics. Thus, observable demographics play a much smaller role in this specification.

[36]On average across state-months, there are 79,608 enrollees in the sample based on stayers versus 102,858 enrollees in the full 2008 cohort sample.

[37]To ease the computational burden associated with estimating this nonlinear model, I use a 10% random sample of the adult enrollees in the 14 states of interest. This yields 176,609 individuals

individual drops out in the current period conditional on having survived up to the current period, as function of covariates.[38] The covariates I use are the three-month trailing average of opioid use (measured in MME per month), the PDMP indicator variable D_{it}, an interaction between opioid use and D_{it}, as well as individual-level demographics and state and time fixed effects.[39]

The estimated coefficients of the Cox model are reported in Table 2.6. The coefficient on the PDMP indicator is positive, which suggests that the probability of dropping out of the sample increases after a PDMP becomes operational, but it is insignificant. Furthermore, even if higher attrition was associated with PDMP implementation, this would not introduce bias in the difference-in-differences analysis unless attrition was also correlated with higher or lower use of opioids. While there is some evidence that heavy opioid use is associated with higher attrition (regardless of whether the state the enrollee is in has a PDMP or not), the insignificant coefficient on the interaction between the PDMP indicator and opioid usage implies that attrition is not different for heavy users in states and time periods with PDMP. Therefore, the attrition present in the data is unlikely to bias the estimates of the primary difference-in-difference analysis.

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and 4,751,890 enrollee-months.

[38] Proportional hazards models are a type of survival models in which covariates are multiplicatively related to the hazard function, the conditional probability of "failing" in a period given survival up to that period. Cox proportional hazards models are a semi-parametric variety in which the baseline hazard function is left unspecified (Greene, 2012).

[39] In addition to the three-month trailing average of MME per month, I also explored other measures of opioid use. Two and three-month trailing averages of MME, days supply, and number of opioid prescriptions yield qualitatively the same results.

Table 2.6: Cox Proportional Hazards Model

	Estimate	p-value
PDMP	0.133147	0.294
MME 3-month tr. avg.	0.000012	0.106
PDMP × MME 3-month tr. avg.	0.000003	0.800
State FEs	Yes	
Year-Month FEs	Yes	
Demographics	Yes	
Enrollee-Months	4,751,890	
Enrollees	176,609	

Notes: An observation is an enrollee-month. The dependent variable is an indicator whether an enrollee drops out after a given period, having survived up to that period. Included demographic variables are current or prior cancer diagnosis, pain or fracture diagnosis in the current or past two months, as well as age, age squared, share female, HMO, POS, PPO plan type, full-time, salaried, hourly employee, employed in mining or manufacturing, transportation, communications, or utilities, retail trade or services, and finance, insurance, or real estate. p-values are based on the robust standard errors clustered at the state level. $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

2.5.3 Cross-border Opioid Purchases

Potential out-of-state opioid purchases are a common concern when the analysis of PDMP effects on opioid prescribing is done at the state level. The reason is that a user (or an abuser) who finds it difficult to obtain opioids after the implementation of a PDMP may travel to a neighboring state without a PDMP and obtain the drugs there. If sales were tracked at the state level, this type of behavior would decrease per-capita opioid consumption in the PDMP state and increase it in the neighboring state, which will bias the results towards finding a negative effect of PDMP on opioid use.

This problem is significantly attenuated in my analysis because the Marketscan database tracks prescription drug purchases at the individual level. As long as enrollees pay for prescriptions through their insurance, the prescription drug will be associated with a particular enrollee and her state of residence. Therefore, even if a

person travels out of state to get a drug, this purchase will be counted toward the PDMP state's count and it will not bias the estimation.

A problem arises if an individual travels out of state *and* pays for the opioids out-of-pocket (or obtains them on the black market in the PDMP state). This will lower the observed opioid sales in the PDMP state and make the PDMP seem more effective at curtailing opioid use. Roberts et al. (2016) provides evidence that when patients are forced to obtain opioid prescriptions from a single physician, they are much more likely to avoid using their insurance. Whether the combined cost of having to travel out-of-state and pay out-of-pocket deters most opioid users from doing so is an open question. Unfortunately, I am not able to address it with the available data and, therefore, leave it for future research.

2.5.4 Other Policy Changes as Confounding Factors

States responded to the opioid crisis by adopting a multitude of policies, one of which was the implementation of PDMPs. If other policies were driving differential changes in the treated and control states between 2008 and 2010, a difference-in-differences analysis that does not take them into account would yield biased estimates of the effectiveness of PDMPs. In this subsection, I review the timing of some of these other policies and argue that they are unlikely to affect my results.

Mandates requiring physicians and pharmacists to register and consult the state's PDMP prior to prescribing or dispensing controlled substances are among the most commonly studied opioid-related policies. Mallatt (2017) provides the timing of these

mandates. The first state to introduce them was Nevada in 2007, while the second was Ohio in 2011. Given that Nevada is not part of my restricted control group and that there were no changes between 2008 and 2010, registration and use mandates are unlikely to be introducing bias into my estimation.

Pain clinics, pejoratively called "pill mills," are facilities primarily focused on pain treatment for patients with various ailments. Laws governing their operation are another potential confounding factor. However, between 2008 and 2010 there were only two such laws: the Louisiana one, introduced in July 2005 and unchanged during the period of interest, and the Texas one, introduced in June 2009.[40] Once again, my preferred specification with a restricted set of control states should be unaffected.

For similar reasons, naloxone access laws, Good Samaritan laws, state policies limiting high morphine equivalent daily dose (MEDD) prescribing, and state Medicaid expansions are unlikely to affect my results. There were no changes in naloxone laws during 2008-2010 and only one new Good Samaritan law passed in Washington state in June 2010 (Doleac and Mukherjee, 2018). Similarly, Heins et al. (2020) document that the earliest MEDD policy was enacted in Washington state in 2007 was not changed until 2012, while other states implemented such policies in 2011 and later. Medicaid expansions under the Affordable Care Act did not start until 2014 Maclean and Saloner (2019).

Evans et al. (2019) and Alpert et al. (2017) study the effects of the introduction of abuse-deterrent OxyContin in August 2010 and find a substantial increase in the use of heroin. To the extent that reformulated OxyContin differentially affected treated

[40]Timing provided in Mallatt (2017).

and control states in my sample, it may bias my estimation of PDMP effects. As a sensitivity, I reestimate my main model on data only prior to August 2010. The results are qualitatively the same and quantitatively very similar to my main results.[41]

The medical use of marijuana, treating severe and chronic pain among other ailments and a potential substitute for opioid medications, started to get legalized in the late 1990s. During the 2008-2010 period, medical marijuana laws came into effect in Arizona (November 2010), Michigan (December 2008), New Jersey (July 2010), and Washington DC (July 2010), and were amended in Colorado (June 2010), Maine (November 2009), Rhode Island (June 2009), and Washington (November 2008).[42] Out of these states, only Arizona and Washington DC are in my preferred specification. Dropping Arizona and DC entirely or just the periods after these laws were introduced does not significantly affect the estimates of my preferred specification.

Given all of these findings, I conclude that it is unlikely that other policies are confounding my results.

2.6 Conclusion

The contribution of this paper is to estimate the effect of plain-vanilla PDMPs for the population of working-age adults that bore the brunt of the opioid crisis. PDMP with no registration or use mandates were the predominant type of monitoring programs until 2010. Many considered them to be ineffective. However, the results of the

[41] This sensitivity also takes care of the withdrawal of propoxyphene from the market on November 19, 2010 (Haffajee et al., 2018) .

[42] Description and timing of these marijuana laws are provided by ProCon.org (2019).

current paper suggests that, to the contrary, these programs were effective at reducing opioid use, especially prescriptions for longer-term supply and high dosage. Therefore, even though modifications such as use mandates strengthened their effects, these early PDMPs were a step in the right direction in the fight against the opioid crisis.

2.7 Appendix

2.7.1 PDMP Effect on Health Outcomes

In this section, I analyze the impact of PDMPs on health outcomes such as heroin poisonings, poisonings with any opioids, and opioid dependence and abuse inpatient and outpatient admissions.[43] Given that Alpert et al. (2017) and Evans et al. (2019) provide evidence that opioids and heroin are substitutes, it is possible that heroin poisonings increase if PDMPs are successful in reducing opioid utilization and especially if they prevent those addicted to opioids from obtaining them. The effect on the other two health outcomes is a priori ambiguous: any-opioid poisonings may increase or decrease depending on whether the decline in opioid usage is larger or smaller than the increase in possible substitutes like heroin and methadone. Similarly, treatment for opioid dependence and abuse may increase or decrease depending on the changes in utilization in prescription and other types of opioids.

Figure 2.5 shows the evolution of these health outcomes over time. Poisoning events are fairly rare and panels (a) and (b) show very irregular patterns over time. While the number of dependence and abuse admissions per capita in panel (c) is higher, it is quite variable over time, especially in the treated states. The same applies for panel (d), which combines the poisoning counts in panel (b) and the dependence and abuse counts from panel (c). Compared to Figure 2.2, it is much more difficult to claim that the parallel trends assumption is likely to hold for health outcomes.

[43] I identify these health outcomes using the following ICD-9-CM codes: 965.01 for heroin poisoining, all 965.0 codes for poisoning with any opioid, and all 304.0, 304.7, and 305.5 codes for opioid dependence and abuse.

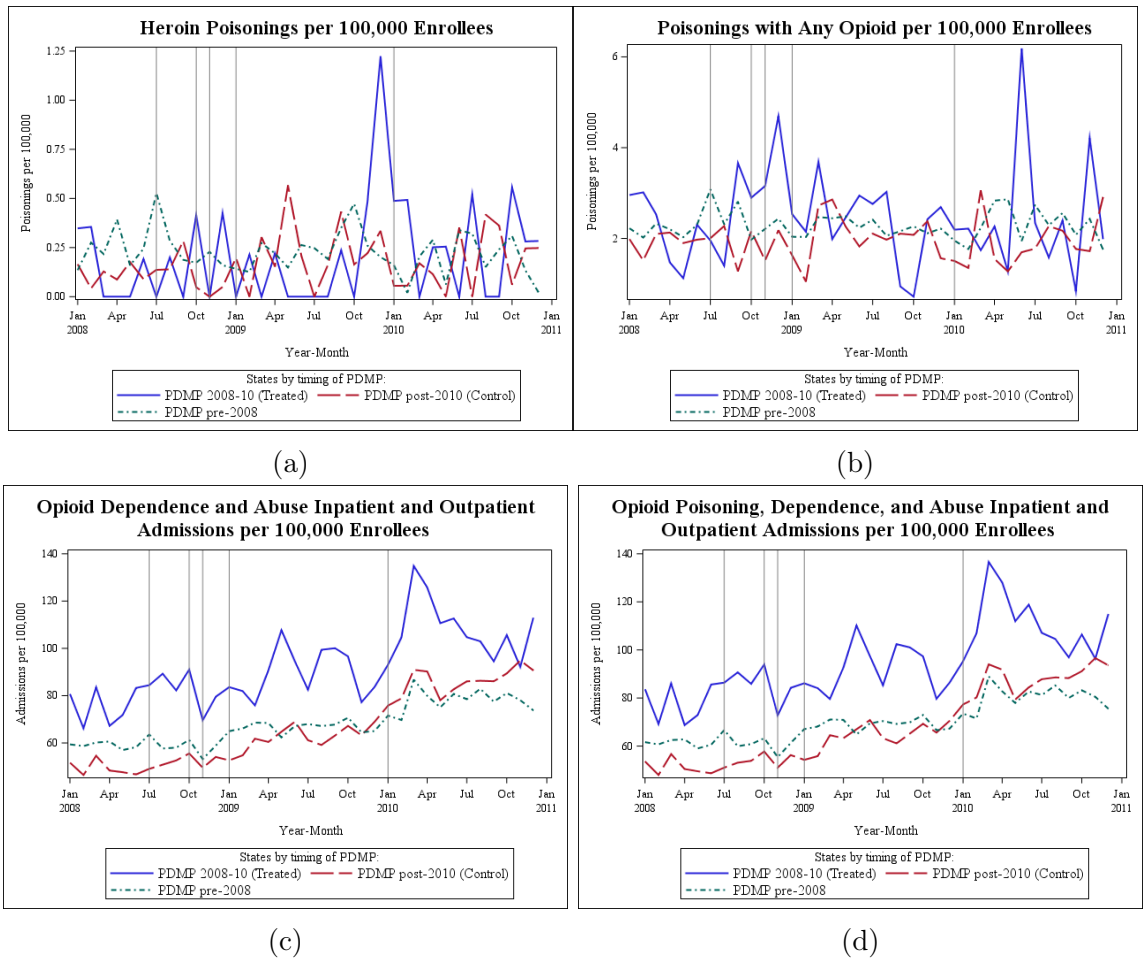

Figure 2.5: Health Outcomes over Time

Notes: The statistics for each group of states are the averages over all states within a group, weighted by number of enrollees. The vertical lines indicate the months in which PDMPs became operational.

Therefore, any results from a difference-in-differences analysis of these data should be treated with caution.

Table 2.7: Difference-in-Differences Analysis, Health Outcomes

	$\hat{\beta}$	p-val	R^2	Obs
Panel A: All States				
Log(Heroin Pois. per 100k)	0.0642**	0.025	0.200	1,800
Log(Any-Opioid Pois. per 100k)	0.2321**	0.050	0.196	1,800
Log(Opioid Dep. & Ab. Adm. per 100k)	0.0071	0.967	0.697	1,800
Log(Opioid Pois., Dep. & Ab. Adm. per 100k)	0.0579	0.723	0.680	1,800
Panel B: States with Post-2010 PDMP Law as Control Group				
Log(Heroin Pois. per 100k)	-0.0035	0.946	0.245	504
Log(Any-Opioid Pois. per 100k)	0.2521	0.192	0.261	504
Log(Opioid Dep. & Ab. Adm. per 100k)	-0.0667	0.890	0.694	504
Log(Opioid Pois., Dep. & Ab. Adm. per 100k)	-0.0372	0.935	0.684	504

Notes: Each *row* is a separate regression. An observation is a state-month pair in the period of 2008-2010. Dependent variables are natural log of health outcome measures per 100,000 adults aged 18-64. Demographic variables, year-month and state fixed effects are included in each regression. p-values are based on the wild-bootstrap procedure in Cameron et al. (2008). $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

Table 2.7 summarizes the difference-in-differences analyses of the health outcomes. Even though the PDMP effect on any-opioid poisonings seems to be large and positive, the patterns in Figure 2.5 make these estimates highly suspect. The estimated effect on combined poisoning, dependence, and abuse admissions may be somewhat more believable based on Figure 2.5, but they are highly insignificant, positive in one case and negative in the other, based on the full and restricted set of states. Therefore, I cannot make any strong conclusions about the effect of PDMPs on health outcomes.

2.7.2 Alternative Control and Treatment Groups

In the main specification of the difference-in-differences model, I use states that have not passed PDMP legislation as of the end of 2010 as a control group. I do that to eliminate possible bias from dynamic effects of PDMPs passed in years prior to 2008 and from anticipation effects of PDMPs that were approved prior to 2010 but did not become operational only after 2010. In this section of the Appendix, I explore the sensitivity of the results with respect to different control and treatment groups.

Table 2.8 summarizes the first set of robustness checks. I proceed by including South Carolina, which I had excluded in the main specification because it has only one non-treated month, as part of the treatment group and then successively adding the PDMPs that became operational in 2007, 2006, 2005, and in all years prior to 2005. Adding South Carolina decreases the magnitude of the PDMP effect on prescriptions but increases it for days supply and MME per capita, leaving the overall results very similar to the baseline. Including the PDMPs of 2007 (Colorado, North Carolina, and North Dakota) leads to much smaller coefficients. However, closer examination of the data for North Dakota, shown in Figure 2.6, reveals that it is an outlier both in terms of the magnitude and trends of opioid utilization. Adding only Colorado and North Carolina, which are much closer to the typical treatment and control states, produces results that are much more similar to the main results. The magnitudes are slightly smaller, which suggests that the strengthening effects of these PDMPs over time may be biasing the results by making the increase in opioid consumption in control states seem smaller. Similar results arise from including PDMPs that became operational

in 2006 and 2005. Finally, adding all PDMPs that were operational prior to 2005 significantly decreases the estimated effects possibly because these states saw smaller increases in opioid utilization thanks to the well-established monitoring programs.

Table 2.8: Alternative Control Groups, Add Pre-2008 PDMPs

	$\hat{\beta}$	p-val	R^2	Obs
Panel A: Baseline (Panel C in Table 2.4)				
Log(Presc. per 1k)	-0.0281**	0.036	0.982	504
Log(Days Supp. per 1k)	-0.0379*	0.056	0.986	504
Log(MME per 1k)	-0.0834**	0.039	0.978	504
Panel B.1: Baseline + SC				
Log(Presc. per 1k)	-0.0258**	0.017	0.983	540
Log(Days Supp. per 1k)	-0.0395**	0.011	0.987	540
Log(MME per 1k)	-0.0868**	0.011	0.978	540
Panel B.2: Baseline + SC + PDMP-07				
Log(Presc. per 1k)	-0.0152*	0.080	0.98	648
Log(Days Supp. per 1k)	-0.0105	0.521	0.984	648
Log(MME per 1k)	-0.0136	0.742	0.969	648
Panel B.3: Baseline + SC + PDMP-07 (excl. ND)				
Log(Presc. per 1k)	-0.0244**	0.025	0.983	612
Log(Days Supp. per 1k)	-0.0331**	0.036	0.987	612
Log(MME per 1k)	-0.0706**	0.018	0.978	612
Panel B.4: Baseline + SC + PDMP-06-07 (excl. ND)				
Log(Presc. per 1k)	-0.0262***	0.005	0.986	720
Log(Days Supp. per 1k)	-0.0349***	0.007	0.990	720
Log(MME per 1k)	-0.0768***	0.009	0.980	720
Panel B.5: Baseline + SC + PDMP-05-07 (excl. ND)				
Log(Presc. per 1k)	-0.0269***	0.005	0.985	792
Log(Days Supp. per 1k)	-0.0374***	0.005	0.989	792
Log(MME per 1k)	-0.0820***	0.003	0.978	792
Panel B.6: Baseline + SC + PDMP-05-07 (excl. ND) + PDMP-Pre05				
Log(Presc. per 1k)	-0.0171	0.216	0.989	1440
Log(Days Supp. per 1k)	-0.0177	0.189	0.992	1440
Log(MME per 1k)	-0.0604***	0.004	0.981	1440

Notes: Each *row* is a separate regression. An observation is a state-month pair in the period of 2008-2010. Dependent variables are natural log of opioid utilization measures per 1,000 adults aged 18-64. "MME" stands for morphine milligram equivalent. Demographic variables, year-month and state fixed effects are included in each regression. p-values are based on the wild-bootstrap procedure in Cameron et al. (2008). $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$. PDMP-07 = CO, NC, ND; PDMP-06 = AL, OH, TN; PDMP-05 = MS, NM; PDMP-Pre05 = CA, ID, IL, PA, NY, RI, TX, MI, OK, MA, WV, UT, NV, IN, KY, VA, ME, WY.

The results in Table 2.9 analyze the sensitivity of the main results by adding

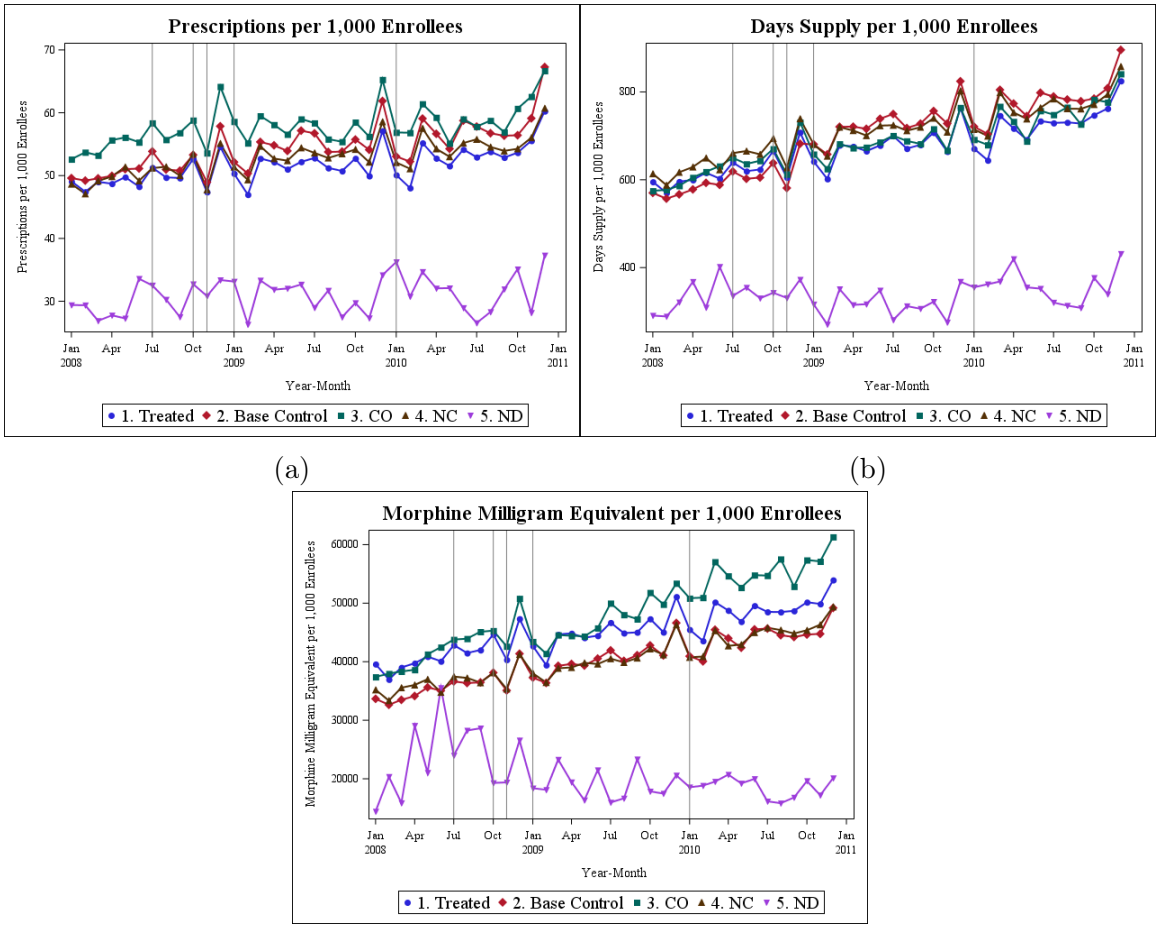

Figure 2.6: Alternative Control Group (Add South Carolina, Colorado, North Carolina, and North Dakota)

PDMPs that were approved by state legislatures before 2010 but came into effect after 2010. Including the PDMPs of 2011 (Kansas, Oregon, Alaska, Florida, New Jersey, Washington, and South Dakota) yields smaller effects on prescriptions and days supply but a larger effect on MME. Overall, the results are quite similar to the baseline, although there might be a slight anticipation effect as reflected by the smaller coefficients on prescriptions and days supply.[44] Adding the rest of the post-2010 PDMPs (Delaware and Wisconsin) lowers the magnitudes of the estimated effects further, although this is largely driven by Delaware.

Table 2.9: Alternative Control Groups, Add Post-2010 PDMPs

	$\hat{\beta}$	p-val	R^2	Obs
Panel A: Baseline (Panel C in Table 2.4)				
Log(Presc. per 1k)	-0.0281**	0.036	0.982	504
Log(Days Supp. per 1k)	-0.0379*	0.056	0.986	504
Log(MME per 1k)	-0.0834**	0.039	0.978	504
Panel C.1: Baseline + PDMP-11				
Log(Presc. per 1k)	-0.0250	0.119	0.982	756
Log(Days Supp. per 1k)	-0.0298	0.108	0.985	756
Log(MME per 1k)	-0.0962***	0.003	0.977	756
Panel C.2: Baseline + PDMP-11 + PDMP-Post11				
Log(Presc. per 1k)	-0.0177	0.271	0.981	828
Log(Days Supp. per 1k)	-0.0175	0.358	0.985	828
Log(MME per 1k)	-0.0755**	0.018	0.977	828
Panel C.3: Baseline + PDMP-11 + PDMP-Post11 (excl. DE)				
Log(Presc. per 1k)	-0.0215	0.144	0.982	792
Log(Days Supp. per 1k)	-0.0236	0.185	0.985	792
Log(MME per 1k)	-0.0853***	0.005	0.976	792

Notes: Each *row* is a separate regression. An observation is a state-month pair in the period of 2008-2010. Dependent variables are natural log of opioid utilization measures per 1,000 adults aged 18-64. "MME" stands for morphine milligram equivalent. Demographic variables, year-month and state fixed effects are included in each regression. p-values are based on the wild-bootstrap procedure in Cameron et al. (2008). $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$. PDMP-11 = KS, OR, AK, FL, NJ, WA, SD; PDMP-Post11 = DE, WI.

[44] An anticipation effect will lower opioid utilization in the later part of the 2008-2010 period, making the increase in opioid use smaller for the control group, which will make the effect of PDMPs seem smaller.

Finally, in Table 2.10 I explore the robustness of the results with respect to the states that are included in the treatment group. I drop each of the treated states, one at a time, and re-estimate the model. The results are all very similar to the baseline results, which suggests that there is no single state that is driving the overall results.

Table 2.10: Alternative Treatment Groups, Drop States One-by-One

	$\hat{\beta}$	p-val	R^2	Obs
Panel A: Baseline (Panel C in Table 2.4)				
Log(Presc. per 1k)	-0.0281**	0.036	0.982	504
Log(Days Supp. per 1k)	-0.0379*	0.056	0.986	504
Log(MME per 1k)	-0.0834**	0.039	0.978	504
Panel D.1: Drop CT				
Log(Presc. per 1k)	-0.0274***	0.002	0.983	468
Log(Days Supp. per 1k)	-0.0302*	0.080	0.987	468
Log(MME per 1k)	-0.1052**	0.042	0.977	468
Panel D.2: Drop AZ				
Log(Presc. per 1k)	-0.0389***	0.000	0.980	468
Log(Days Supp. per 1k)	-0.0447**	0.013	0.985	468
Log(MME per 1k)	-0.0961**	0.030	0.976	468
Panel D.3: Drop LA				
Log(Presc. per 1k)	-0.0272	0.138	0.98	468
Log(Days Supp. per 1k)	-0.0311	0.149	0.985	468
Log(MME per 1k)	-0.0989*	0.061	0.977	468
Panel D.4: Drop IA				
Log(Presc. per 1k)	-0.0375**	0.035	0.981	468
Log(Days Supp. per 1k)	-0.0388*	0.061	0.986	468
Log(MME per 1k)	-0.0951**	0.025	0.977	468
Panel D.5: Drop VT				
Log(Presc. per 1k)	-0.0347**	0.035	0.983	468
Log(Days Supp. per 1k)	-0.0493*	0.089	0.987	468
Log(MME per 1k)	-0.1027**	0.013	0.980	468
Panel D.6: Drop MN				
Log(Presc. per 1k)	-0.0295	0.196	0.981	468
Log(Days Supp. per 1k)	-0.0366	0.230	0.985	468
Log(MME per 1k)	-0.0794**	0.043	0.976	468

Notes: Each *row* is a separate regression. An observation is a state-month pair in the period of 2008-2010. Dependent variables are natural log of opioid utilization measures per 1,000 adults aged 18-64. "MME" stands for morphine milligram equivalent. Demographic variables, year-month and state fixed effects are included in each regression. p-values are based on the wild-bootstrap procedure in Cameron et al. (2008). $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

2.7.3 Falsification Test Results

Tables 2.11 and 2.12 contain the output from the falsification test regressions discussed in Section 2.5.1.

Table 2.11: Falsification Tests, Prescription Drug Regressions

	Log(Presc. per 1k)			Log(Days Supp. per 1k)		
Therapeutic Class	$\hat{\beta}$	p-val	R^2	$\hat{\beta}$	p-val	R^2
Vitamin D, NEC	-0.133***	0.000	0.970	-0.131***	0.002	0.956
Cath & Lax, Laxatives, Enemas	-0.126***	0.000	0.650	-0.132	0.196	0.537
Vitamin A & Derivatives	-0.059***	0.005	0.579	-0.294***	0.009	0.519
Sulfones, NEC	-0.050***	0.004	0.470	-0.266*	0.069	0.390
Antimalarial Agents, NEC	-0.047**	0.031	0.730	-0.110***	0.009	0.646
S/MM, Soaps/Cleansers/Antiseptics	-0.045	0.112	0.648	-0.076	0.172	0.603
Antiinflam S/MM Agnts & Comb, Misc	-0.041**	0.031	0.813	-0.049**	0.042	0.845
Antichol/Antimuscarinic/Antispas	-0.037	0.155	0.897	-0.051	0.345	0.802
Antiprut/Local Anest S/MM, NEC	-0.035	0.182	0.741	-0.109*	0.064	0.717
Anticonvulsants, Misc	-0.035**	0.020	0.914	-0.031*	0.094	0.889
Autonomic, Nicotine Preparations	-0.035	0.247	0.881	-0.084	0.218	0.820
S/MM Miscellaneous, NEC	-0.034	0.148	0.748	-0.093**	0.032	0.797
Eye/Ear/Nose/Throat Misc, NEC	-0.034	0.158	0.845	-0.062*	0.062	0.832
Antidiabetic Agents, Insulin	-0.032*	0.070	0.867	-0.066***	0.008	0.825
Antituberculosis Agents, NEC	-0.030*	0.100	0.686	-0.159	0.210	0.570
Antiallergic Agents	-0.028	0.318	0.877	-0.048	0.574	0.728
Psychother, Tranq/Antipsychotics	-0.027	0.165	0.838	-0.016	0.584	0.799
Repl Preps, Sodium Chlor Preps	-0.026*	0.097	0.602	-0.062	0.510	0.541
Antihistamines & Comb, NEC	-0.025	0.280	0.933	-0.001	0.970	0.847
Thy/Antithy, Antithyroid Agents	-0.025	0.303	0.561	-0.056	0.583	0.534
Diuretics, Loop Diuretics	-0.025	0.192	0.919	-0.028	0.318	0.882
Anti-infectives, Misc	-0.024	0.325	0.843	-0.046	0.341	0.685
Cardiac, Alpha-Beta Blockers	-0.022	0.317	0.728	-0.081	0.352	0.496
Antichol/Antiparkinsonian Agents	-0.022	0.170	0.611	-0.139	0.234	0.645
Antibiot, Cephalosporin and Rel.	-0.021	0.339	0.900	-0.047*	0.095	0.878
Alkalinizing Agents, NEC	-0.021	0.257	0.564	-0.075	0.547	0.442
(260-Missing Description)	-0.019	0.265	0.530	-0.135	0.317	0.524
Leukotriene Modifiers	-0.018	0.351	0.830	-0.047*	0.073	0.748
Antiifect, Sulfonamides EENT	-0.017	0.305	0.412	-0.026	0.811	0.348
Cell Stim/Proliferant S/MM, NEC	-0.016	0.517	0.827	-0.032	0.547	0.802
(254-Missing Description)	-0.016*	0.052	0.433	-0.295**	0.037	0.398
Antiinf S/MM, Antibiotics & Comb	-0.016	0.442	0.894	-0.009	0.760	0.863
Antihelmintic, NEC	-0.015	0.370	0.202	-0.110	0.170	0.214
Fluoride Preparations, NEC	-0.015	0.518	0.798	-0.103	0.285	0.675
Oxytocics, NEC	-0.013	0.355	0.347	0.007	0.872	0.543
Urinary Anti-infectives, NEC	-0.013	0.567	0.834	0.011	0.833	0.694
Caloric Agents, Nutrition Preps	-0.013	0.100	0.773	-0.114	0.300	0.768
Muscle Relax, Skeletal Central	-0.012	0.418	0.955	-0.044**	0.038	0.947
Histamine (H2) Antagonists, NEC	-0.012	0.597	0.909	-0.052	0.199	0.850
Muscle Relax, Skeletal, Misc	-0.012	0.508	0.743	0.004	0.970	0.723
Thy/Antithy, Thyroid Hormones	-0.011	0.220	0.962	-0.024**	0.046	0.954
Miotics, EENT, NEC	-0.011	0.173	0.189	-0.164	0.192	0.242

Falsification Tests, Prescription Drug Regressions (continued)

	Log(Presc. per 1k)			Log(Days Supp. per 1k)		
Therapeutic Class	$\hat{\beta}$	p-val	R^2	$\hat{\beta}$	p-val	R^2
Coag/Anticoag, Anticoagulants	-0.011	0.611	0.688	-0.016	0.563	0.718
Antiplatelet Agents, NEC	-0.011	0.552	0.946	-0.024	0.387	0.920
Cardiac, Antiarrhythmic Agents	-0.009	0.674	0.817	0.043	0.615	0.718
Estrogens & Comb, NEC	-0.008	0.508	0.980	-0.030*	0.062	0.967
Parasympathomimetic, NEC	-0.008	0.668	0.685	-0.075	0.500	0.541
(257-Missing Description)	-0.008	0.603	0.931	-0.026	0.252	0.869
Biological Response Modifiers	-0.008	0.706	0.815	0.039	0.446	0.779
Cholelitholytic Agents, NEC	-0.008	0.621	0.564	-0.007	0.949	0.567
Vitamin Bs w/Vitamin C, NEC	-0.007	0.450	0.590	-0.131	0.274	0.585
Hypotensive Agents, NEC	-0.007	0.720	0.935	-0.045	0.131	0.892
Antiinfect, Antibiotics, EENT	-0.007	0.778	0.410	0.018	0.643	0.424
Multivit Prep, Multivit Minerals	-0.007	0.610	0.934	0.155	0.120	0.909
Antiinf S/MM, Antivirals & Comb	-0.007	0.799	0.470	-0.054	0.605	0.402
Vasodilating Agents, NEC	-0.007	0.778	0.861	0.023	0.749	0.704
Depig/Pig/S/MM Depigment Agents	-0.007	0.645	0.432	-0.098	0.401	0.455
Hematopoietic Agents, NEC	-0.006	0.764	0.391	-0.089	0.555	0.439
Antiinfect, Antiinflam EENT	-0.005	0.848	0.686	-0.003	0.963	0.598
Vitamin K Derivatives, NEC	-0.005	0.390	0.222	0.000	0.994	0.360
Digestants & Comb, NEC	-0.005	0.746	0.531	-0.107	0.346	0.651
Vaccines, NEC	-0.005	0.934	0.747	0.004	0.974	0.589
Sympatholytic Agents NEC	-0.005	0.662	0.390	-0.057	0.597	0.461
Mydriatics, EENT, NEC	-0.004	0.741	0.171	0.024	0.847	0.239
Sulfonamides & Comb, NEC	-0.004	0.847	0.894	-0.007	0.858	0.679
Antiinfect, Antivirals, EENT	-0.004	0.670	0.176	-0.040	0.639	0.229
Antibiot, Aminoglycosides	-0.003	0.858	0.588	0.030	0.730	0.639
Folic Acid & Derivatives, NEC	-0.003	0.894	0.747	0.018	0.652	0.717
Adrenals & Comb, NEC	-0.003	0.833	0.876	-0.029*	0.057	0.881
Ammonia Detoxicants, NEC	-0.003	0.893	0.509	0.046	0.742	0.366
Antianemic, Iron Preparations	-0.002	0.878	0.812	0.038	0.781	0.737
S/MM Misc, Vaginal Lubricants	-0.002	0.771	0.728	-0.071	0.489	0.648
Antiinflam Agents EENT, NEC	-0.002	0.914	0.867	-0.005	0.798	0.834
Antidiarrhea Agents, NEC	-0.002	0.842	0.507	-0.038	0.699	0.573
Diuretics, Carb Anhydrase Inhib	-0.002	0.919	0.553	-0.120	0.386	0.408
Immunosuppressants, NEC	-0.001	0.945	0.844	-0.030	0.432	0.692
Antiinf S/MM, Antiinf Local Misc	-0.001	0.965	0.725	-0.005	0.921	0.678
Antiemetics, NEC	-0.001	0.973	0.655	0.061	0.185	0.659
Antidiabetic Agents, Misc	0.000	0.992	0.956	-0.026*	0.062	0.950
Antibiotics, Misc	0.000	0.998	0.478	0.004	0.931	0.453
Roentgenography, NEC	0.000	0.914	0.365	0.000	0.916	0.410
Antihyperlipidemic Drugs, NEC	0.001	0.912	0.973	-0.017*	0.076	0.966
Eyewash/Eyestrm/Lubr/Tear, NEC	0.001	0.967	0.674	0.001	0.987	0.570
Hemorrheologic Agents, NEC	0.002	0.87	0.456	-0.014	0.917	0.482
Gonadotropins, NEC	0.002	0.958	0.682	-0.014	0.908	0.566
Multivit Prep, Multivit Plain	0.002	0.917	0.814	0.047	0.674	0.675
Antiinfectives, Misc EENT	0.002	0.766	0.201	0.063	0.363	0.236
Antibiot, Penicillins	0.002	0.896	0.880	-0.014	0.509	0.850
Phosphodiesterase Inhibitors	0.002	0.900	0.946	0.024	0.438	0.880
Anxiolytic/Sedative/Hypnotic NEC	0.002	0.914	0.820	0.013	0.721	0.754
Contraceptive, Oral Comb, NEC	0.003	0.700	0.978	-0.011	0.322	0.962
Serums, NEC	0.004	0.429	0.688	-0.037	0.578	0.685

Falsification Tests, Prescription Drug Regressions (continued)

	Log(Presc. per 1k)			Log(Days Supp. per 1k)		
Therapeutic Class	$\hat{\beta}$	p-val	R^2	$\hat{\beta}$	p-val	R^2
Keratoplastic Agents S/MM, NEC	0.004	0.81	0.338	0.014	0.904	0.403
Vascular 5HT1 Agonist, NEC	0.005	0.776	0.854	-0.017	0.650	0.721
Anesthetics, Local EENT, NEC	0.006	0.730	0.532	-0.034	0.737	0.487
Pituitary Hormones, NEC	0.006	0.672	0.462	0.045	0.733	0.493
Unclassified Agents, NEC	0.006	0.719	0.918	-0.024	0.330	0.904
(261-Missing Description)	0.007	0.705	0.769	0.009	0.793	0.737
Gastrointestinal Drugs Misc, NEC	0.007	0.536	0.968	0.008	0.586	0.962
Keratolytic Agents S/MM, NEC	0.008	0.637	0.611	0.035	0.776	0.571
Caloric/Nutrition/Dietary Misc	0.009	0.388	0.806	0.212*	0.068	0.775
Vitamin Bs & B Complex, NEC	0.009	0.688	0.860	0.128	0.209	0.708
Cardiac, Calcium Channel	0.009	0.407	0.983	-0.012	0.370	0.971
Cardiac, Beta Blockers	0.010	0.355	0.961	-0.016	0.156	0.959
Cardiac Drugs. NEC	0.010	0.346	0.985	-0.003	0.772	0.980
Antivirals, NEC	0.010	0.734	0.911	-0.015	0.541	0.969
Cough/Cough/Cold Comb, NEC	0.011	0.759	0.937	-0.018	0.867	0.905
Cardiac, ACE Inhibitors	0.011	0.234	0.967	-0.019*	0.059	0.969
Anticholinergic, NEC	0.011	0.617	0.765	0.030	0.591	0.687
Antibiot, Antifungal	0.011	0.601	0.887	-0.038	0.318	0.741
Potassium Removing Resins, NEC	0.011*	0.064	0.341	0.075	0.298	0.454
Enzyme Preps, Topical S/MM, NEC	0.011	0.188	0.259	0.067	0.332	0.354
Caloric Agents, Amino Acid Preps	0.012	0.188	0.454	0.225	0.121	0.467
Progestins, NEC	0.013	0.552	0.731	-0.015	0.618	0.743
Antimanic Agents, NEC	0.013	0.591	0.663	0.017	0.777	0.639
S/MM Misc, Astringents	0.014	0.457	0.428	0.081	0.523	0.429
Multivit Prep, Multivit Iron	0.014	0.234	0.821	-0.069	0.54	0.852
Antiinf S/MM, Antifungals & Comb	0.015	0.501	0.914	0.003	0.923	0.877
Mucolytics, Cold Comb, NEC	0.016	0.134	0.329	0.232*	0.057	0.341
Sympathomimetic Agents, NEC	0.016	0.434	0.751	-0.004	0.838	0.778
Repl Preps, Potassium Supp	0.018	0.341	0.956	0.001	0.978	0.934
Antiinf S/MM, Scabic/Pediculic	0.019	0.387	0.447	0.059	0.577	0.401
Parathyroid Hormones, NEC	0.020	0.131	0.575	0.073	0.602	0.571
Muscle Rel, Smooth-Genitour NEC	0.020	0.322	0.846	0.024	0.510	0.791
Vitamins & Comb Misc, NEC	0.021***	0.004	0.631	0.250***	0.008	0.666
Antibiot, Erythromycin & Macrolide	0.022	0.434	0.915	-0.012	0.692	0.895
Diuretics, Potassium-Sparing	0.023*	0.099	0.953	-0.024	0.258	0.898
Quinolones, NEC	0.023	0.237	0.897	0.018	0.432	0.872
Antigout Agents, NEC	0.024	0.305	0.802	0.027	0.436	0.757
Muscle Rel, Smooth-Respiratr NEC	0.025	0.107	0.803	0.175	0.185	0.631
Repl Preps, Calcium Supp	0.025**	0.044	0.349	0.187	0.172	0.414
Psychother, Antidepressants	0.026***	0.001	0.975	0.005	0.584	0.969
Emoll/Moist/Demul/Protect S/MM	0.027	0.281	0.712	0.033	0.753	0.587
Multivit Prep, Multivit Prenatal	0.027	0.185	0.951	0.022	0.567	0.907
Diuretics, Thiazides & related	0.028**	0.025	0.964	0.006	0.692	0.959
Phosphorus Removing Agents, NEC	0.029**	0.025	0.677	0.106	0.428	0.600
Antidiabetic Ag, Sulfonylureas	0.029*	0.098	0.931	-0.011	0.630	0.885
(263-Missing Description)	0.032***	0.008	0.651	0.201**	0.045	0.645
Cath & Lax, Laxatives, Saline	0.032	0.110	0.778	0.073	0.397	0.711
Ovulation Stimulants, NEC	0.034*	0.077	0.544	0.044	0.572	0.586
CNS Agents, Misc.	0.038	0.104	0.874	0.045	0.294	0.834
(262-Missing Description)	0.040*	0.098	0.683	0.074	0.183	0.440

Falsification Tests, Prescription Drug Regressions (continued)

	Log(Presc. per 1k)			Log(Days Supp. per 1k)		
Therapeutic Class	$\hat{\beta}$	p-val	R^2	$\hat{\beta}$	p-val	R^2
Antitussives/Cold Comb, NEC	0.041	0.213	0.882	0.000	0.999	0.849
Anticonv, Hydantoin Derivatives	0.043*	0.059	0.737	0.139	0.196	0.568
Antibiot, Tetracyclines	0.052**	0.012	0.698	0.042*	0.069	0.722
Pharmaceutical Aids/Adjuv, NEC	0.053*	0.071	0.743	0.068	0.533	0.735
Vitamin Bs w/Iron/Other Min NEC	0.056***	0.002	0.803	0.419***	0.000	0.815
Cardiac, Cardiac Glycosides	0.060***	0.003	0.839	-0.004	0.959	0.662
Antineoplastics S/MM, NEC	0.070***	0.002	0.493	0.435***	0.001	0.468
Cath & Lax, Laxatives, Stimulant	0.170***	0.000	0.730	0.133	0.256	0.700

Notes: Each row shows the regressions of the number of prescriptions per capita and the days supply per capita for a given therapeutic class of drugs. Demographic variables, year-month and state fixed effects are included in each regression, which is estimated on 504 observations. An observation is a state-month pair in the period of 2008-2010. p-values are based on the wild-bootstrap procedure in Cameron et al. (2008). $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

Table 2.12: Falsification Tests, Medical Procedure Group Regressions

	Log(Procedures per 1k)		
Medical Procedure Group	$\hat{\beta}$	p-val	R^2
Other home health services	-0.183***	0.003	0.903
Other medicine procedures	-0.160***	0.001	0.777
Destruction, facial lesion	-0.134***	0.000	0.540
Other preventive medical services	-0.127**	0.014	0.701
Durable medical equipment	-0.120***	0.000	0.883
Psychiatric diagnostic services	-0.117***	0.001	0.774
Blood chemistry, Rx monitor	-0.116***	0.003	0.723
Other ENT services (non-surgical)	-0.105**	0.026	0.943
Physical medicine: unlisted/other	-0.102*	0.073	0.841
Injections: immunizations	-0.101**	0.011	0.969
Other lab & path procedures	-0.100*	0.090	0.827
Chemotherapy injections	-0.099*	0.081	0.789
Ophthalmic diagnostic services	-0.088***	0.002	0.913
Physical medicine: manipulation	-0.081**	0.021	0.842
Transurethral surgery	-0.076	0.201	0.264
Other toxicology tests	-0.074**	0.013	0.943
Blood test: Hgb/Hct	-0.074**	0.028	0.941
Home health PT/OT/ST	-0.068	0.353	0.681
EKG stress test	-0.061*	0.082	0.724
Other minor respiratory procedures	-0.054*	0.092	0.646
Minor hemic & lymphatic procedures	-0.053	0.183	0.156
Other chemistry tests	-0.053***	0.002	0.974
Laparoscopy, hysteroscopy	-0.052	0.472	0.812
Dental: basic restorative	-0.050	0.127	0.444
Chiropractic services	-0.049**	0.024	0.974
Transportation services	-0.046	0.304	0.764
Psychotherapy, family	-0.043	0.283	0.888
Nerve conduction tests/EMG	-0.043	0.549	0.583
Allergy therapy	-0.04*	0.065	0.973
CT scan, extremities	-0.039	0.206	0.170
Other urinalysis	-0.038	0.105	0.896
Physical medicine: other modes	-0.036	0.273	0.913
Blood test: sedimentation rate	-0.032	0.230	0.899
General eye exams	-0.031	0.400	0.981
Performance tracking codes	-0.029	0.373	0.652
Physical medicine: hot/cold packs	-0.029	0.544	0.916
X-ray, abdomen	-0.028	0.388	0.760
Anesthesia services	-0.026	0.253	0.899
Other radioimmunoassays (RIA)	-0.025	0.702	0.963
Arthrocentesis, large joint	-0.025	0.442	0.762
Nail debridement/avulsion	-0.025	0.363	0.778
Echocardiogram	-0.025	0.453	0.882
Dx ultrasound, pregnancy	-0.024	0.454	0.857

Falsification Tests, Medical Procedure Group Regressions (continued)

	Log(Procedures per 1k)		
Medical Procedure Group	$\hat{\beta}$	p-val	R^2
Decompression, carpal tunnel	-0.023	0.433	0.498
Destruction of warts	-0.022	0.397	0.809
Destruction, non-facial lesion	-0.021	0.614	0.803
Dx ultrasound, other	-0.019	0.387	0.864
Bacterial culture, screening	-0.019	0.545	0.863
Preventive care visits	-0.018	0.252	0.942
Specialty drugs	-0.017	0.715	0.802
Office visits, new patient	-0.017	0.263	0.927
Speech/hearing therapy	-0.016	0.693	0.336
Physical medicine: other procedures	-0.015	0.390	0.956
Spirometry	-0.015	0.593	0.853
Cystourethroscopy	-0.015	0.644	0.547
Mammograms	-0.014	0.480	0.899
Office visits, established patient	-0.014*	0.086	0.950
Acne surgery	-0.013	0.329	0.581
Other major ear/auditory procedures	-0.013	0.678	0.212
Other radiology procedure	-0.013	0.693	0.604
Other minor cardiovascular procedures	-0.013	0.754	0.850
Venograms	-0.012	0.490	0.230
Nuclear medicine, diagnostic	-0.012	0.786	0.870
Major hemic & lymphatic procedures	-0.011	0.802	0.171
PTCA- percutaneous angioplasty	-0.010	0.656	0.379
Major maternity procs & related care	-0.010	0.801	0.279
Other neurology dx services	-0.009	0.799	0.755
CT scan, abdomen/pelvis	-0.008	0.795	0.644
Thyroid function tests (RIA)	-0.008	0.664	0.954
Pulmonary function tests	-0.007	0.848	0.725
Blood count, automated	-0.007	0.638	0.973
Excision of breast tissue	-0.007	0.868	0.169
Other microbiology tests	-0.007	0.807	0.938
Bronchoscopy	-0.006	0.839	0.152
Other minor musculoskeletal surgery	-0.005	0.906	0.720
Repair of inguinal hernia	-0.004	0.939	0.19
Medical supplies and devices	-0.004	0.869	0.849
X-ray, extremities	-0.003	0.853	0.783
Allergy testing	-0.003	0.941	0.530
Lymphangiograms	-0.003	0.851	0.149
Pap smear	-0.003	0.889	0.932
EKG monitoring	-0.002	0.970	0.471
Minor endocrine system procedures	0.000	0.882	0.159
Routine urinalysis	0.000	0.980	0.971
Vaginal deliveries	0.000	0.985	0.216
Surgical pathology	0.000	0.995	0.864
Dental: diagnostic & preventive	0.000	0.985	0.364

Falsification Tests, Medical Procedure Group Regressions (continued)

	Log(Procedures per 1k)		
Medical Procedure Group	$\hat{\beta}$	p-val	R^2
Nuclear medicine, therapeutic	0.001	0.961	0.252
Respiratory therapy	0.001	0.988	0.566
Physician telephone/online visits	0.001	0.855	0.305
Other hematology tests	0.002	0.965	0.813
Dilation & currettage	0.002	0.930	0.191
Dental: other	0.002	0.468	0.222
Other consults, location unspecified	0.002	0.351	0.316
Lab tests, organ/disease panel	0.002	0.874	0.974
Psychotherapy, individual	0.003	0.835	0.987
Case management services	0.003	0.850	0.714
Physical medicine: elec stimulation	0.004	0.853	0.869
Other minor ear/auditory procedures	0.005	0.876	0.602
Immunology tests	0.005	0.854	0.960
Other minor urinary procedures	0.005	0.905	0.677
Magnetic resonance (NMR/MRI)	0.006	0.808	0.758
Myelograms/discograms	0.006	0.793	0.500
Blood test: prothrombin time	0.007	0.774	0.862
Dx radiology, misc/other	0.007	0.828	0.766
Blood chemisty tests, automated	0.007	0.331	0.279
Bacterial culture, urine	0.007	0.763	0.952
Incision & drainage of cyst	0.007	0.829	0.517
Injections: therapeutic/IV	0.007	0.781	0.931
Dental: major restorative	0.008	0.341	0.226
Aortograms	0.009	0.358	0.426
Other maternity procs & related care	0.010	0.839	0.718
PET scan	0.010	0.751	0.501
Minor male genital procedures	0.011	0.690	0.407
Other major cardiovascular procedures	0.011	0.853	0.328
Other minor digestive procedures	0.011	0.783	0.204
Other major breast surgery	0.012	0.795	0.33
Bronchospasm evaluation	0.012	0.700	0.673
Gastroenterology services (non-surgical)	0.012	0.695	0.548
Major endocrine system procedures	0.013	0.734	0.169
Other minor skin & breast surgery	0.013	0.606	0.778
Other major respiratory procedures	0.015	0.822	0.461
Skin lesion injection	0.016	0.502	0.811
CT scan, spine	0.018	0.602	0.526
Cesarean section deliveries	0.018**	0.028	0.278
Major nervous system procedures	0.018	0.770	0.561
Other major urinary procedures	0.019	0.700	0.211
Definitive bacterial culture	0.020	0.502	0.909
X-ray, spine/pelvis	0.020	0.360	0.876
Major male genital procedures	0.021	0.517	0.250
Venipuncture (draw blood)	0.022	0.116	0.975

Falsification Tests, Medical Procedure Group Regressions (continued)

	Log(Procedures per 1k)		
Medical Procedure Group	$\hat{\beta}$	p-val	R^2
X-ray, chest	0.022	0.306	0.891
Other major surgery procedures	0.022	0.563	0.282
Other major eye/ocular procedures	0.024	0.618	0.332
Non-invasive peripheral vascular studies	0.025	0.445	0.745
Arthrocentesis, sm/med joint	0.026	0.404	0.588
Thyroid function tests (non-RIA)	0.027	0.306	0.962
Minor female genital procedures	0.028	0.396	0.541
X-ray, OB/Gyn	0.028	0.203	0.318
Cholecystograms/cholangiograms	0.029	0.135	0.476
Other anatomic pathology services	0.029	0.659	0.511
Therapeutic radiology	0.030	0.778	0.433
Antibiotic sensitivity studies	0.031	0.256	0.861
Blood count, manual	0.032	0.252	0.814
Other minor eye/ocular procedures	0.033	0.276	0.480
EKG	0.033	0.107	0.950
Dx ultrasound, abdominal	0.033	0.290	0.692
Angiograms	0.033	0.426	0.430
Cataract removal	0.034	0.487	0.459
Upper GI endoscopy	0.039	0.428	0.496
Major female genital procedures	0.040	0.446	0.540
X-ray, GI tract	0.041	0.189	0.564
Colposcopy	0.041	0.104	0.473
Clinical path, consultation	0.042**	0.012	0.580
ENT diagnostic services	0.042	0.270	0.963
Other major musculoskeletal surgery	0.043	0.488	0.457
Laryngoscopy	0.045	0.134	0.666
Unmapped codes	0.046	0.586	0.702
Colonoscopy	0.046	0.194	0.652
X-ray, head & neck	0.047	0.137	0.797
Office visits, emergency	0.050	0.161	0.949
Physical medicine: ultrasound	0.052	0.112	0.777
CT scan, head & neck	0.053	0.112	0.650
Cardiac catheterization	0.056	0.451	0.640
Specimen handling	0.058	0.176	0.934
CT scan, chest	0.059*	0.086	0.473
Therapeutic psychiatric services	0.059*	0.076	0.924
Physical medicine: testing	0.061**	0.026	0.910
Minor nervous system procedures	0.063*	0.055	0.575
Other medical services	0.066	0.206	0.692
Other non-surgical pulmonary services	0.067*	0.081	0.788
Outpatient consults	0.072**	0.028	0.873
Inpatient consults	0.088***	0.002	0.595
Other major digestive procedures	0.091	0.195	0.485
Other major skin surgery	0.096**	0.040	0.576

Falsification Tests, Medical Procedure Group Regressions (continued)

	Log(Procedures per 1k)		
Medical Procedure Group	$\hat{\beta}$	p-val	R^2
Emergency room visits	0.098***	0.000	0.933
General ophthalmology services	0.110***	0.000	0.957
Office visits, other	0.127**	0.034	0.678
Other cardiovascular procedures	0.128**	0.031	0.509
Psychotherapy, group	0.129**	0.028	0.726
Other injections/noninjectables	0.194***	0.000	0.893
Facility visits	0.262***	0.000	0.682
Dialysis	0.325***	0.000	0.834

Notes: Each row shows the regression of the number of procedures per capita for a given medical procedure group. Demographic variables, year-month and state fixed effects are included in each regression, which is estimated on 504 observations. An observation is a state-month pair in the period of 2008-2010. p-values are based on the wild-bootstrap procedure in Cameron et al. (2008). $^{***}p < 0.01$, $^{**}p < 0.05$, $^{*}p < 0.1$.

Chapter 3

Empirical Evidence on Conditional Pricing Practices: A Review *

(Co-authored with Julie Holland Mortimer)

3.1 Introduction

Conditional pricing practices (CPPs) allow the terms of sale between a producer and a downstream firm to vary based on whether the downstream firm meets a set of conditions that the producer specifies. The conditions may require a downstream firm to accept minimum quantities or multiple products, to purchase a minimum share of its requirements, or even to deal exclusively with one producer. Payment from the producer to the downstream firm may take the form of a discount at the time

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of purchase, a rebate paid after a period of time, or marketing support and training. CPPs cover a wide variety of arrangements and are in widespread use throughout many industries.

CPPs have often been challenged in court over the years, but there is no consensus among lawyers, judges, or academics on how they should be analyzed. Fundamentally, adjudication seeks to determine whether a given CPP harms or benefits competition. Under U.S. law, this means determining whether a practice reduces or improves consumer welfare. To a large degree practitioners' efforts to evaluate this question have rested on two approaches: applying theoretical models of the potential mechanisms behind CPPs or using prior litigated arrangements as precedent. In this article, we consider the effects of CPPs through a third lens: empirical research analyzing a variety of CPPs across several different industries.

Empirical research provides unique insight into understanding the effects of CPPs that is complementary to the insights gained through theoretical analysis and litigated arrangements. Theoretical models predict a wide range of mechanisms through which CPPs may affect welfare, and there may be multiple theoretical models that could be relevant for analyzing any given CPP. Court cases are selected through the process of litigation and may not be representative of the wider population of such arrangements. Empirical research addresses these limitations, while simultaneously highlighting the wide variety of settings in which CPPs are used. A limitation of the empirical literature, however, is that it cannot necessarily address the full range of potential settings or arrangements that one may ultimately want to analyze. Relatedly, the heterogeneity highlighted in this literature does not necessarily lend itself to

a single unifying framework to adjudicate future CPPs.

Some arrangements that can be described as CPPs include: vertical rebates, which can be structured as "loyalty discounts" or "all units discounts" (AUDs); vertical bundling, which includes "full-line forcing" (FLF) contracts and bundled discounts; and exclusive dealing.[1] The term "exclusive dealing" may be used to describe a loyalty discount with a 100 percent market share requirement.[2]

Table 3.1 presents a selected group of CPPs and the range of industries they cover, based on a review of judicial decisions and empirical research. Vertical rebates have been used, for example, in the truck transmission, microprocessor, and confections industries. Vertical bundling has been employed in markets for video rentals, tape products, and some pharmaceutical products, among others. Exclusive dealing has been used in the video game, smartphone, and auto refrigerant equipment industries.[3] A much richer set of arrangements is employed across many more industries in reality.

A brief review of cases involving CPPs illustrates the difficulties that courts have faced in adjudicating these legal disputes and the concomitant lack of consensus on an appropriate analytical framework. In *LePage's Inc. v. 3M*, 3M was the dominant player in the market for branded tape products but was facing competitive pressure

[1]We refer to a vertical rebate as a loyalty discount if it is conditional on a customer buying a specified share of its overall requirements from the supplier (i.e., a market-share requirement) and an AUD if it is conditional on a quantity requirement.

[2]The Third Circuit defines exclusive dealing as "an agreement in which a buyer agrees to purchase certain goods or services only from a particular seller for a certain period of time." The Third Circuit further clarifies that even though an agreement to deal exclusively is a prerequisite to exclusive dealing, an express exclusivity requirement is not necessary. ZF Meritor, LLC v. Eaton Corp., 696 F.3d 254 (3d Cir. 2012). Some authors also require that an exclusive dealing arrangement include an explicit or implicit threat that the seller will refuse to deal with the buyer unless the buyer accepts the arrangement, an "all-or-nothing" clause (Klein and Lerner, 2016).

[3]An arrangement with a very high, but not 100%, market share requirement can be considered de facto exclusive dealing.

Table 3.1: Analyses of Conditional Pricing Practices

	Product Coverage	Nature of Restriction	Downstream Competition
Judicial Decisions:			
Truck Transmissions (*ZF Meritor v. Eaton Corp.)*	Single	Share	Standard
Auto Refrigerant Equip. (*SPX Corp. v. Mastercool Inc.)*	Single	Exclusive	Standard
Tape Products (*LePage's v. 3M)*	Multiple	Quantity	Standard
Boat Engines (*Concord Boat v. Brunswick Corp.)*	Single	Share	Standard
Anticoagulants (*Eisai v. Sanofi-Aventis)*	Single	Share	[1]
Cephalosporins(*SmithKline v. Eli Lilly)*	Multiple	Quantity	[1]
Microprocessors (three Intel cases)*	Single§	Share	Standard
Hospital Services (*Cascade Health Solutions v. PeaceHealth)*	Multiple	Share	[2]
Catheters (*Southeast Missouri Hospital v. C.R. Bard Inc.)*	Multiple	Share	[2]
Airline Reservations (two British Airlines cases)†	Single	Quantity	Standard
Mobile Phones (Korean Fair Trade Commission fine)‡	Multiple	Share	Standard
Empirical Research:			
Confections (Conlon and Mortimer (2019a))	Multiple	Quantity	[3]
Video Rentals (Ho et al. (2012a,b))	Multiple	Quantity	Standard
Video Games (Lee (2013))	Single	Exclusive	[4]
Smartphones (Sinkinson (2020))	Single	Exclusive	[5]
Beer (Sass (2005), Asker (2004, 2005), Chen (2014))	Multiple	Exclusive	Standard

Notes: "Product coverage" indicates whether the arrangement governs purchases of a single product or requires the purchase of multiple products. "Nature of the restriction" describes the condition that a downstream firm must meet to qualify for payment. "Share" indicates a market share requirement. "Quantity" indicates a minimum (or maximum) quantity requirement used in AUDs, FLF contracts, or other arrangements. "Exclusive" indicates exclusive dealing. "Downstream competition" is noted as "Standard" when downstream firms compete on price. Alternative forms of downstream competition vary by industry and are described as follows:

[1] Product administered to patients in hospitals. Insurers reimburse hospitals for a patient's treatment.

[2] Insurers reimburse hospitals for services associated with patient treatment.

[3] Retail prices rarely vary across products or time.

[4] Gaming consoles are durable; consumer demand responds to current and expected future prices.

[5] Service providers subsidize the purchase price of a handset when a consumer agrees to a two-year service plan.

* Cases are: *FTC v. Intel, AMD v. Intel,* and *Intel v. Commission.*

§ The FTC Intel case has a multiproduct aspect to it.

† Cases are: *Virgin Atlantic v. British Airlines* and *British Airlines v. Commission.*

‡ Fine levied against Qualcomm. See press release "Qualcomm's Abuse of Market Dominance," Korea Fair Trade Commission, July 23, 2009, available at `http://eng.ftc.go.kr/bbs.do`.

from private label tape manufacturer LePage's.[4] 3M responded by entering the private label tape market and offering retailers discounts on bundles consisting of private label tape and other of its office products. LePage's could not match this strategy because of its limited product line, and claimed that its rival's pricing scheme was exclusionary. 3M argued that its conduct was not anticompetitive because it did not sell transparent tape below cost. The Third Circuit rejected 3M's argument and ruled in favor of LePage's despite the absence of below-cost pricing. However, the ruling

[4]LePage's Inc. v. 3M, 324 F.3d 141 (3d Cir. 2003).

has been criticized for failing to provide sufficiently clear guidance regarding when bundled rebates violate antitrust law.

The Ninth Circuit adopted a different approach in *Cascade Health Solutions v. PeaceHealth*.[5] There, the plaintiff and the defendant were the only health care providers in Lane County, Oregon. Whereas Cascade Health offered only primary and secondary care, PeaceHealth offered tertiary care as well. PeaceHealth offered insurance companies substantial discounts if they made it their sole provider of all three levels of health services. In response, Cascade Health challenged the practice as exclusionary. In a break with the Third Circuit's reasoning, the Ninth Circuit determined that the conduct could not be condemned as anticompetitive absent a showing that the defendant had lowered prices below "an appropriate measure of cost." Using a "discount attribution test," it ruled in favor of the defendant and reversed the district court's decision.

The lack of agreement on the correct principles for adjudicating conditional pricing practices applies not only to multiproduct discounts but to single-product loyalty discounts as well. *ZF Meritor v. Eaton Corp.* was a lawsuit brought against the dominant manufacturer of heavy-duty truck transmissions by a rival firm.[6] The contention was that long-term contracts that the defendant signed with the four major truck manufacturers amounted to de facto exclusive dealing. These contracts provided rebates to the truck manufacturers if they satisfied a high minimum-share purchase requirement, treated Eaton's products preferentially in their sales catalogs,

[5]Cascade Health Sols. v. PeaceHealth, 515 F.3d 883, 899–900 (9th Cir. 2008).
[6]ZF Meritor, LLC v. Eaton Corp., 696 F.3d 254 (3d Cir. 2012).

and priced Eaton's products lower than the plaintiff's products. The Third Circuit ruled against the defendant after applying a rule-of-reason analysis and declining to employ a price-cost test because it found that price was not the primary method of exclusion.

The outcome was different in *Eisai Inc. v. Sanofi Aventis*.[7] In that case, the defendant offered hospitals a discount on its drug Lovenox if they made 90 percent or more of their total anticoagulant drug purchases from Sanofi. Eisai had exclusive distribution rights to Pfizer's competing product, Fragmin, and alleged that Sanofi's conduct bundled customers' contestable and incontestable demand for Lovenox and amounted to de facto exclusive dealing. Because Eisai's claims related primarily to the alleged de facto exclusive dealing aspect of Sanofi's conduct and not to its pricing practices, the Third Circuit analyzed the conduct under the rule of reason rather than applying a price-cost test. It concluded that there was no evidence of either restriction of consumer choice or substantial anticompetitive effect and upheld summary judgment in favor of the defendant.

One reason for the lack of agreement on the appropriate framework of analysis of CPPs is that there is no consensus in the theoretical literature either. Economists have found both procompetitive and anticompetitive justifications for these arrangements. However, empirical analyses that give more credibility to one theory or another are relatively scarce.

In this article, we provide background regarding the theoretical literature addressing CPPs and review the existing empirical literature. We identify market features

[7]Eisai Inc. v. Sanofi Aventis U.S., LLC, 821 F.3d 394 (3d Cir. 2016).

that affect the likelihood that a CPP will have an adverse impact on consumer welfare. We find that anticompetitive effects are more likely when CPPs are used by a dominant firm and when buyers have limited capacity to carry multiple products from suppliers. The existence of substitute products or alternative distributors can also influence the effect of conditional pricing on competition. These market characteristics are just a few of the factors that should be considered in analyzing CPPs. Furthermore, the empirical analyses reveal that different arrangements have different exclusionary effects and should be studied in conjunction with the characteristics of the specific markets in which they are used. The wide array of arrangements and market settings precludes broad generalizations and suggests that the effects of conditional pricing can differ case by case, based on the specifics of the CPP and the market.

3.2 Theoretical Background

Interest in CPPs has generated a large volume of scholarly work, but there is no consensus on their predominant competitive effect or on an appropriate analytical framework to use in a litigation setting. In this section, we review the prevalent points of view on these questions in the theoretical literature, first for single-product and then for multiproduct CPPs.

3.2.1 Single-product Conditional Pricing Practices

Klein and Lerner (2016) view single-product loyalty contracts as a commitment device that allows a seller and a buyer to achieve a mutually beneficial equilibrium off the demand curve.[8] Without commitment, a seller facing a downward-sloping demand curve sells the quantity at which marginal cost equals marginal revenue and charges the price indicated by the demand curve. However, it can do better by writing a contract that offers a lower price only if the buyer agrees to purchase a larger quantity. The seller is better off because it sells a sufficiently larger quantity to offset profit forgone through the lower price. The buyer also benefits because the discount it receives on the units it would purchase absent the contract and the additional units it buys at the discounted price outweighs the negative surplus on units that it values at less than the discounted price. The buyer can move off the demand curve because it is not a final consumer but rather a downstream firm that subsequently resells the product as a component in a different product or service. The Klein-Lerner model assumes that final consumers are unlikely to substitute to a competing product based on a preference for a single component, thus conferring a degree of loyalty on the buyer.[9] The buyer can exploit this loyalty to shift purchases from one seller to another. Sellers compete for these sales-shifting services and compensate the buyer through the loyalty discount.[10]

[8] A similar procompetitive justification for conditional pricing is given in Murphy et al. (2015).

[9] For instance, patients will not change the hospital they go to because it does not carry the patient's preferred brand of blood-clotting drug. Similarly, a truck buyer will not go to a rival manufacturer only because it offers different transmissions.

[10] The authors assume that any disadvantages to consumers from increases in the list (non-contract) price, by either the seller or its competitor, are outweighed by the benefits from the contract. In order for the contract to be procompetitive, the model must implicitly assume that at least some portion of the discount is passed on to consumers. This need not always be the case (e.g., if consumers are

Despite this procompetitive justification for loyalty contracts, Klein and Lerner acknowledge that such contracts can also be used anticompetitively. In their prescriptive analysis, they distinguish between two types of contract terms: performance and incentive. Performance terms stipulate the conditions that a buyer needs to meet, such as market share, preferential treatment of certain products, and retail pricing requirements. Incentive terms specify what happens if the buyer does not satisfy the performance requirements: for example, it may forfeit the discount or may even face restricted supply. The authors argue that if a discount is the only incentive mechanism, the contract resembles predatory pricing, and a "discount attribution test" safe harbor can be applied.[11] However, if the loyalty contract includes non-price incentive terms, such as a threat to restrict or terminate supply, or if the list price is much higher than what would prevail absent the contract, the authors recommend a rule-of-reason analysis.[12]

Other scholars, such as Salop (2017) and Wright (2013), recognize that many CPPs resemble both predatory pricing (through discount terms) and exclusive dealing (through exclusivity or near exclusivity requirements), but argue that a rule-of-reason standard better captures the various mechanisms through which CPPs may affect consumer welfare. In a predatory pricing setting, a firm lowers its price below

locked in).

[11]The test applies the full amount of discounts on the contestable portion of sales and compares the discounted price to marginal cost, where contestable sales are those for which the rival can "reasonably compete." If the discounted price is lower than marginal cost, the loyalty discount is likely predatory, and the next step in the analysis is to determine if the dominant firm will be able to eventually recoup its "investment."

[12]The authors explain that before weighing pro- and anticompetitive effects, the analysis needs to determine whether the contractual arrangement constitutes de facto exclusive dealing, which is the case when the contract gives the buyer no economic choice but to accept the offered terms if the buyer wants to deal with the seller.

cost, drives equally efficient rivals out of the market, then raises its price to a supra-competitive level and recoups the profit lost while it was pricing below cost. The mechanisms that harm consumer welfare are the exclusion of rivals and subsequent higher prices. This is why predatory pricing analyses proceed by comparing price to cost and, if price is lower, assessing whether the alleged predator can recoup its "investment."

Unlike predatory pricing, exclusive dealing can lead to exclusion of rivals without below-cost pricing.[13] Moreover, exclusive dealing can also impair competition without inducing full exclusion in the market. By restricting rivals' access to vital inputs or a sufficient customer base, exclusive dealing may effectively raise their costs, forcing them to increase their prices and reducing the competitive constraint they can impose.[14] Similarly, rivals' ability to compete can be limited if they are relegated to a niche position in the market with limited access to customers. A reduced customer base can also diminish rivals' incentives to invest and innovate, which in turn may lead to less investment and innovation by the dominant firm. Exclusive dealing requires a different analytical framework than predatory pricing because it can lead to competitive harm in more varied ways. Steven Salop follows this logic to argue that a rule-of-reason standard of adjudication is appropriate because it can account for the

[13]The theoretical literature on the exclusionary effects of exclusive dealing is sizeable. Authors in the tradition of the "Chicago School" have argued that exclusive dealing cannot lead to exclusion of an equally efficient rival because compensating the downstream firm for accepting the arrangement makes it unprofitable for the upstream firm to offer it in the first place. See, e.g., Bork (1993) and Posner (1976). Other authors have used models with scale economies and externalities across buyers to show that anticompetitive exclusion is possible. See, e.g., Aghion and Bolton (1987), Rasmusen et al. (1991), Segal and Whinston (2000), and Whinston (2008).

[14]Through these effects, exclusive dealing arrangements fit into the "raising rivals' costs" paradigm Salop (2017).

various mechanisms through which harm can occur.[15]

While exclusive dealing can induce foreclosure or raise rivals' costs, this need not automatically translate into consumer harm. For consumer harm to result, the seller employing the arrangement needs to have "power over price." Such power may not exist if competitors are not significantly disadvantaged by the conduct, if there is sufficient competition from non-foreclosed competitors, or if there are substitute products. Another reason why consumers may not be harmed by exclusive dealing is that such arrangements can induce efficiencies. For example, exclusive dealing can intensify competition among suppliers, provide incentives for better products, service, or increased promotion, or reduce free riding (Salop, 2017). These procompetitive effects should be accounted for when evaluating the overall competitive effect of exclusive dealing.

There is also a growing theoretical literature that focuses specifically on the effects of CPPs that are not as restrictive as exclusive dealing.[16] This literature has found conditions under which loyalty discounts, among a broader group of vertical practices, can lead to foreclosure (Asker and Bar-Isaac, 2014; Chen and Shaffer, 2014). However, conditional pricing, and AUDs in particular, need not necessarily reflect an exclusionary motive but may instead provide a more effective way to price discrim-

[15]Salop (2017) also discusses various reasons why the price-cost test can give too many false positives and false negatives, which make it unsuitable as a method to analyze alleged anticompetitive conduct related to CPPs.

[16]Although it is often assumed implicitly that loyalty discounts are a weaker, but also cheaper, way of foreclosing rivals than exclusive dealing, the opposite may be true under some circumstances. In particular, loyalty discounts may allow an incumbent firm to achieve a desired level of foreclosure more flexibly but also at a higher cost because it needs to over-compensate buyers that accept the arrangement. Thus, there are situations in which loyalty discounts are preferred to exclusive dealing and can achieve a higher level of foreclosure (Chen and Shaffer, 2016).

inate than a menu of two-part tariffs (Kolay et al., 2004). Furthermore, AUDs can mitigate moral hazard and improve efficiency by providing incentives to upstream and downstream firms to make investments that boost both firms' profits.[17] The conditional discount may encourage a retailer to expand output while still compensating the upstream firm for its investment (O'Brien, 2017). Similarly, upstream firms can use market share discounts to induce selling effort from downstream retailers (Mills, 2010).

3.2.2 Multiproduct Conditional Pricing Practices

Multiproduct CPPs (or bundled discounts) condition a buyer's discount on its purchase of multiple different products.[18] As with single-product CPPs, there is no established consensus on how multiproduct CPPs should be analyzed. Some courts and scholars have recommended using a predatory pricing-based price-cost test (Greenlee et al., 2008). Others, however, have pointed out that multiproduct CPPs can have exclusionary effects even without below-cost pricing and are best assessed as forms of tying (Carlton et al., 2008).[19]

Tying can have both exclusionary and nonexclusionary rationales and can either increase or decrease consumer welfare. Firms can tie products to attain efficiencies in production stemming from scale economies, to price discriminate, or to achieve greater product differentiation (Carlton et al., 2008). While these exemplify nonexclusionary

[17]For example, these investments can take the form of advertising by the upstream firm and in-store promotional effort by the downstream firm.

[18]Some authors use the term "bundling" to describe selling packages of multiple units of the same product and the term "tying" for selling packages of different products. Others do not stick to this convention and use "bundling" for selling different products together (Shy, 1995).

[19]Carlton et al. (2008) point out additional weaknesses of the price-cost test.

uses of tying, bundled discounts can also be used anticompetitively in a variety of ways. For instance, a firm with a monopoly in a market for a primary good that also supplies a complementary good in a duopoly market can use tying to extend its monopoly power to the complementary good market by denying scale to its rival.[20] A firm can also use tying to strengthen its market power in the primary market by excluding producers of complementary goods, thus making it harder for firms that need access to the complements to compete in the primary market.[21] Finally, an incumbent firm may tie its products in an effort to deter the entry of a firm producing a superior good in the complementary market, thus eliminating the threat that the (potential) entrant may eventually challenge the incumbent in the primary market.[22]

3.3 Empirical Evidence

Economic theory suggests that conditional pricing can have both positive and negative effects on competition. In reality, both types of effects are likely to occur simultaneously, so that the net impact of a given CPP becomes an empirical question. Empirical work is also helpful for establishing the means by which CPPs affect consumer welfare. As discussed in the preceding section, below-cost pricing is one mechanism that can lead to rival exclusion, but not the only one. Furthermore, even

[20] An important requirement is that the complement can be used without the monopoly good. Otherwise the monopolist can achieve the same or higher profit without tying, i.e., tying is a feasible but not necessarily profitable monopolization strategy. This is an example of the "one monopoly rent" critique (Carlton et al., 2008).

[21] This can also be seen as an example of raising rivals' costs.

[22] Carlton et al. (2008) examine the conditions under which a bundled discount is likely to be anticompetitive. These include situations in which rivals face economies of scale, the discounting firm has market power, the price of the tied good increases for consumers that do not buy the tying good, and rivals exit or face increased marginal costs because of the bundled discount. Greenlee et al. (2008) examine the impact of bundled discounts when the adjacent market is perfectly competitive.

in the presence of foreclosure, consumer welfare may not be harmed. Thus, empirical work that investigates the net impact and the different mechanisms through which CPPs affect competition can inform both the courts' general attitude towards these types of arrangements and the particular framework for analyzing their impact.

Although existing case law illustrates the issues raised by CPPs and the methods used to analyze them, it does not necessarily reflect the competitive effects of CPPs in general because of sample selection bias.[23] For this reason, independent empirical research is essential for shedding light on the impact of these practices. Such inquiries, however, face a variety of challenges, which makes them scarce. First, data are often proprietary and difficult to obtain. Second, when data are available, lack of variation in prices and choice sets often hinders demand estimation. Third, supply-side estimation becomes problematic when agents' actions are endogenous or difficult to observe. Finally, the extremely wide variety of arrangements and institutional settings makes it difficult to generalize results and extrapolate from one industry or type of arrangement to another.

Despite these challenges, economists have made progress in empirically assessing the impact of CPPs. Earlier work primarily consists of "reduced-form" analyses. But more recently researchers have used "structural" models, which allow one to conduct counterfactual experiments and study more closely the mechanisms that

[23]That certain instances of conditional pricing end up in court suggests that these cases may be more likely to be anticompetitive, because plaintiffs expend the effort to litigate. Possible selection bias from relying on litigated cases is discussed in Ippolito (1991). Sample selection bias is an issue independent of whether courts reach the correct conclusion about alleged anticompetitive effects. There is a tradeoff between maximizing the probability that courts adjudicate a given practice correctly and having predictable and easily implementable, albeit more frequently incorrect, court decisions. A thorough evaluation of this tradeoff is beyond the scope of this article.

affect consumer welfare.[24] There have been empirical studies of the effects of CPPs, including loyalty discounts, AUDs, and exclusive dealing. We organize the discussion of these studies by industry and other market features that have implications for the competitive effects of CPPs.

3.3.1 Confections and Beer

Confections and beer are traditional food and beverage manufacturing industries. New product introductions are relatively infrequent, but there are plenty of imperfect substitutes. In the studies we consider, CPPs are offered by dominant firms, and there is evidence that the arrangements may foreclose rivals under certain conditions. However, the estimated impact on consumer welfare is positive in the case of confections and negative but small in the case of beer.

Confections

Conlon and Mortimer (2019a) study the efficiency and foreclosure effects of an AUD used by the dominant firm in the vending channel of the confections industry. The main upstream players are Mars, Nestle, and Hershey. The dominant firm, Mars, offers a per-unit rebate on the total quantity purchased in a given fiscal quarter. To qualify for the rebate, a vending operator needs to meet or exceed a quarterly purchase

[24]Structural models typically specify the behavior for both firms and consumers. If these behavioral models are correct, the researcher can estimate parameters of the objective functions of firms and consumers that are robust to policy changes. Knowledge of these "primitives" allows the researcher to conduct counterfactual analyses; thus, one can change a particular feature of the world and predict what market outcomes will be. The parameters estimated in reduced-form models may lack policy robustness and need not reveal anything about agents' objective functions, ruling out the ability to explore counterfactual simulations. However, reduced-form analyses do not require explicit behavioral models of agents in the market.

target customized for that vendor, which applies to the total across all varieties of Mars candy; it must also satisfy a facing requirement, which specifies that it carry at least six Mars products in each vending machine.

The effect of the AUD on firm profits and consumer utility is theoretically ambiguous. On one hand, the AUD requirements can induce the retailer to restock its vending machines more frequently and reduce the likelihood of a product stocking out.[25] The increased level of effort increases consumer welfare because it ensures customers can buy their top choice of candy. The AUD also mitigates downstream moral hazard, which occurs when the retailer lacks the incentive to exert the level of effort optimal for the manufacturer. For example, if a Mars product is out of stock and customers are forced to substitute to a competing product with a higher margin, the retailer will not restock. This leaves Mars worse off and the retailer and the competing manufacturer better off. The AUD rebate effectively increases the retailer's margin on the Mars products, creating an incentive for it to restock more frequently.[26]

On the other hand, AUDs can also have anticompetitive effects. The rebate, quantity threshold, and facing requirement can induce a retailer to replace Hershey or Nestle products with Mars candy bars. Such foreclosure reduces the profits of Mars's competitors, but the impact on consumers is unclear. Whether consumer welfare

[25] A stockout occurs when no units of a product remain available for purchase. When the vendor restocks a machine, it replenishes all products, not just the ones that the firm offering the AUD manufactures.

[26] The increased level of retailer effort tends to increase the profits of the dominant manufacturer, whose products are likely to stock out first, and decrease the profits of competing manufacturers. However, this may not be true under all circumstances. If the initial frequency of restocking is so low that Mars, Hershey, and Nestle products all stock out between visits, then an increase in the level of retailer effort can increase the profits of all upstream firms. Conlon and Mortimer (2019a) provide evidence that this does not occur in the market they study, so that increased retailer effort increases Mars profits, while decreasing Nestle and Hershey profits. Mars can induce a similar effect with an unconditional discount, but this approach is not as profitable.

increases or decreases depends on whether consumers like the Mars product(s) better than the Nestle or Hershey products that are displaced.

An important feature of the setting, as it relates to the impact of the AUD on consumer welfare, is that downstream prices are the same across products and rarely vary over time. The reasons for the lack of pricing variation are technical difficulties in providing change and the fact that service contracts sometimes require the vendor to commit to a price structure over a multi-year period. Thus, the AUD can affect consumer welfare through product availability and assortment, but not through retail prices.

To assess the impact on consumer welfare and firm profits, Conlon and Mortimer (2019a) combine a model of demand for different candy bars and a model of restocking. The demand model estimates consumer preferences for different products, while the restocking model estimates the optimal time between restocking visits for the retailer, weighing the cost of a visit against the benefits of extra sales from avoiding a stockout. The authors focus on a representative vending machine carrying five base candy products of the seven supplied and estimate the optimal level of retailer effort and the optimal assortment choice for the last two candy products under different vertical payment structures.[27]

Based on this model, the authors analyze the welfare impact of the AUD. Absent the AUD, the retailer's optimal assortment is to carry two Hershey products, Reese's Peanut Butter Cups and Payday, in addition to the five base products. The motivating

[27]The base products are Snickers, Peanut M&Ms, Twix, Plain M&Ms (owned by Mars), and Raisinets (owned by Nestle).

factor is that even though demand for the Hershey products is slightly lower than for the Mars replacement products, the profit margin on the Hershey products is higher. When Mars offers the AUD, the combination of the per-unit rebate, the quantity threshold, and the facing requirement induces the retailer to increase its restocking frequency and to stock two Mars products (Three Musketeers and Milky Way) instead of Hershey products in the last two slots. This increases the profits realized by the retailer and Mars, but decreases Hershey's and Nestle's profits. The impact on Hershey's bottom line is especially stark because it loses distribution for two products. Further analysis reveals that as long as the marginal cost per candy bar is above 13 cents, there is no price above marginal cost that Hershey can charge in the presence of the AUD that would convince the retailer to carry its products. Furthermore, Hershey has no incentive to offer an AUD of its own because this will only decrease its profits in the event that the retailer accepts it. Thus, there is evidence of foreclosure.[28]

Despite the presence of foreclosure, the authors find that consumers are not harmed by the AUD. Retail prices are assumed to be fixed, so consumer welfare is affected only by the increased level of effort and by the changed assortment. While more retailer effort has an unambiguously positive effect for consumers (by decreasing the number of stockouts and thus increasing availability), the effect of changes

[28]These analyses assume that wholesale prices remain unchanged in the counterfactual world without the AUD. While all three firms can adjust their prices in real life, such adjustments make finding an equilibrium a very difficult problem computationally. The authors conduct an additional analysis in which Hershey's and Nestle's wholesale prices are fixed, but Mars's is not. In this case, Mars lowers its price to undercut Hershey and ensure that the retailer carries two Mars products in the last slots. Once again, Hershey is foreclosed, since it cannot offer a price above marginal cost that would induce the retailer to carry its products.

in product assortment can be either positive or negative. In the Conlon and Mortimer setting, consumers are better off when the retailer carries Three Musketeers and Milky Way (Mars products) than when it carries Reese's and Payday (Hershey products) and maintains the same restocking frequency. Thus, the estimate of the overall effect of Mars's AUD on consumer welfare is positive.[29]

Beer

The beer industry is another traditional manufacturing industry dominated by a small number of major producers and many smaller ones. Products are differentiated, but there are many close substitutes. The market is characterized by a three-tier vertical structure composed of brewers, distributors, and retailers.[30] Some of the largest brewers, such as Anheuser-Busch and Miller, enter into exclusive agreements with their distributors.[31] The effects of these arrangements are studied in three articles. Sass (2005) summarizes the theoretical literature on exclusive dealing and uses reduced-form analyses to determine which theory best describes the observed market outcomes. Using structural models, Chen (2014) and Asker (2016) study the welfare effects of exclusive dealing and analyze whether such arrangements lead to foreclosure of rivals.

[29]Price-cost tests are not designed to shed light on product availability and consumer preferences, which in this case determine the effect of conditional pricing on consumer surplus. Therefore, a price-cost test would be uninformative about the competitive impact of the AUD in this setting.

[30]In most states, owning firms across different tiers is either expressly prohibited or restricted (Chen, 2014; Asker, 2016).

[31]Perhaps the most famous campaign to boost a company's number of exclusive distributors is Anheuser-Busch's "100% share of mind." This campaign was started in 1997 and offered distributors discounts, extended credit, and marketing support in exchange for carrying only Anheuser-Busch products (Sass, 2005; Asker, 2016).

Sass (2005) organizes the rationales for using exclusive dealing into three types: to align distributors' incentives with those of the upstream firm; to foreclose rivals; or to dampen competition among producers. Each of these rationales leads to predictions about the effect of exclusivity on prices and output, which the author evaluates using data from a survey of 391 U.S. beer distributors. Reduced-form analyses indicate that exclusive dealing tends to increase the prices charged by the implementing brewers and distributors, as well as total quantity sold. At the same time, there is no evidence that exclusivity increases the prices of rival brewers and distributors. These results suggest that efficiency-enhancing motives are an important rationale for the use of exclusive dealing in this industry.[32]

Asker (2016) provides further evidence on the effects of exclusive dealing in the market for beer. He focuses on the greater Chicago area, in which the exclusive contracts used by Anheuser-Busch and some other upstream firms raised concerns about the potential foreclosure of rival brewers. Combining a model of consumer demand for beer and a supply-side model of brewer profit maximization, Asker calculates brewer and distributor marginal costs.[33] The results show that brewers that use exclusive dealing enjoy both a cost and a service advantage over their rivals. These advantages can stem from investments that the brewers make in their exclusive distributors or from two types of foreclosure: cost-based or promotion-based.[34] The article develops

[32]The analysis in Sass (2005) does not include a formal evaluation of the impact of exclusive dealing on consumer welfare. The findings suggest an efficiency-enhancing motivation, but it is unclear whether the net effect of higher prices (if higher wholesale prices are passed on to consumers) and increased quantity will be positive or negative.

[33]Distributors in the model are "passive" in that it is not them, but brewers, that set the prices charged to retailers. This feature of the model is supported by the fact that brewers provide strong guidelines to distributors about preferred wholesale prices.

[34]Cost-based foreclosure occurs if a rival cannot access low-cost distributors because of the exclusive arrangement, while promotion-based foreclosure occurs if a rival cannot access the distributors

tests for each type of foreclosure. The idea behind the test for cost-based foreclosure is to compare the distribution costs of brewers that do not employ exclusives, in markets with and without exclusive distributors. Assuming distribution costs are identically distributed across markets (in the statistical sense), if foreclosure occurs, these brewers will face higher costs of distribution on average in markets with exclusives. Specifically, they will not be able to access the most cost-efficient distributors.[35] By contrast, this will not necessarily be the case if brewers use exclusive arrangements to protect investments they have made in their distributors.[36] The test for promotion-based foreclosure is based on the same reasoning. Once implemented, the two tests indicate that cost and promotional advantages are not caused by exclusivity-induced foreclosure and support the conclusion that exclusive beer distribution in metropolitan settings should not raise antitrust concerns.

Asker (2004) also conducts two counterfactual analyses in which exclusive dealing is banned. In the first, the cost advantage from using exclusive dealers is attributed entirely to additional brewer investment in the distributor. A ban on exclusives in such a case eliminates the cost benefits brewers enjoyed by using exclusive dealers. As a result, Anheuser-Busch's and Miller's prices to distributors and retailers increase. These increases are passed on to consumers. Overall, Asker estimates that the ban would lead to a 20 percent decrease in consumer welfare, retailer profits, and total brewer profits. In the second counterfactual, the cost advantage is attributed entirely

most adept at selling its product.

[35]The distribution of distributor costs will be truncated from the left.

[36]Brewers may use exclusives both to foreclose rivals and to protect their investments in their distributors. Even if this is the case, it will not affect the underlying logic of the proposed foreclosure tests because they focus on the distributors used by brewers that do not use exclusive contracts.

to foreclosure. Removing exclusive dealing leads to lower costs for brewers that do not use exclusives, increasing consumer surplus, retailer profits, and brewer profits by 40 percent. The results indicate the potential benefits that an intervention by an antitrust authority can bring if foreclosure is present. However, given that the test results provide no support for the foreclosure hypothesis, the author concludes that the most likely outcome of an intervention is a welfare loss.

Chen (2014) offers additional insights into the impact of exclusive dealing by examining the effect of Anheuser-Busch's exclusive arrangements on microbrewers' entry decisions in northern California markets. This setting allows her to consider foreclosure effects in both metropolitan and rural areas, which complements Asker's results.[37] Chen's analysis uses a model of consumer demand for beer combined with a model of a microbrewer's decision to enter a market, which depends on the expected demand for its product and on the entry decisions of other microbrewers.[38] The demand and entry models recover the impact of exclusivity on the fixed cost and probability of entry. The results highlight two facts. First, the interdependence of firms' entry decisions is important. There are substantial spillover effects of entry into a market: the more microbrewers there are in a market, the easier it is for others to enter, and the harder it is for another firm to deter entry. Strategic interactions are also important because they affect the estimates of the impact of Anheuser-Busch's exclusive arrangements. Chen finds that if strategic interactions are not

[37]Foreclosure in this setting occurs if a microbrewer cannot obtain distribution at a particular store because of exclusive dealing. This definition differs from Asker's, which focuses on the cost-efficiency or marketing aptitude of distributors.

[38]The article studies only the entry decisions of specialty brewers. The large national brewers enter essentially all markets.

taken into consideration, there are no estimated foreclosure effects from exclusivity. However, when such interactions are accounted for, the results provide a more nuanced picture: foreclosure is present in rural areas, outside of the Bay Area and Sacramento counties. Where a foreclosure effect is present, exclusivity decreases the probability of a specialty beer producer's entry by six percentage points–a substantial effect given a base entry probability of 28 percent. A possible reason for the presence of this effect is that there are relatively fewer distributors in rural counties compared to metropolitan areas.[39]

Despite finding foreclosure in some areas, Chen concludes that foreclosing rivals is not the main motivation behind the use of exclusive distributors. Counterfactual simulations show that banning exclusivity does not have a big impact on entry behavior because at most one additional brewer enters a market. Furthermore, the consumer welfare benefit of the expanded product variety is negligible. Even if all specialty beers are stocked, the potential increase in consumer welfare remains fairly inconsequential.[40] The likely reason for such a limited impact is the presence of many substitute products and the fact that many of the specialty brewers are fringe firms that cater to a small segment of the market. Moreover, as small players in the market, microbreweries have minimal impact on equilibrium prices. Finally, demand substitution estimates indicate that by foreclosing a specialty brewer, Anheuser-Busch can sell

[39]Thus, the existence of a foreclosure effect does not contradict Asker (2016), who finds no foreclosure in greater Chicago. In that area, it seems that the relative abundance of distributors helps prevent foreclosure. In particular, even though Anheuser-Busch uses eight and Miller uses five exclusive distributors, there are 29 other distributors to serve the rest of the brewers.

[40]In particular, a ban on exclusives will lead to a $15 increase in consumer surplus per store per quarter. The potential increase if all specialty brewers are stocked at a given store is $510 per store per quarter. These results assume exclusive dealing has no procompetitive effects. If it does, banning exclusive dealing may increase consumer welfare less or may even decrease it.

at most 31 additional six-packs per store per quarter, a negligible amount for a firm of its size. Such a strategy to increase sales seems inefficient. Together with the rest of the results, this suggests that foreclosure is more likely to be a side effect rather than the main rationale for using exclusive dealing, and suggests an efficiency-inducing motivation.

It is possible to draw some conclusions from the analyses of AUDs in the confections industry and exclusive dealing in the beer industry. First, CPPs can lead to foreclosure of rivals, but need not cause substantial (or any) harm to consumers. Second, the dimensions on which consumer welfare can be affected are retail prices, product availability, and product variety. Third, foreclosure is more likely when there are fewer distributors available. Thus, even though Hershey is foreclosed by Mars's AUD from accessing a particular retailer, it may be able to find other distributors in the same area. Fourth, the existence of many close substitutes attenuates the effect of changes in product variety and availability on consumer welfare. For instance, in Conlon and Mortimer's article, the change in product variety actually benefits consumers, while in Chen's article the exclusion of specialty beers decreases consumer surplus only minimally.

3.3.2 Video Rentals

The movie industry differs from traditional manufacturing industries in that the product is an information good. Having "consumed" the content of the product, a consumer does not need to obtain it again (Ho et al., 2012b). This feature forces producers

to continually update their products. Given this constant "churn," firms are only as good as their last few products. As their product lines change, firms face different incentives to use conditional pricing.

The use of a "full-line force" (FLF) contract in the video rental industry and its welfare impacts are the focus of Ho et al. (2012a) and Ho et al. (2012b). The wide spread of the Internet and advances in information technology in the late 1990s, which facilitated tracking transactions from a distance, allowed movie distributors to offer rental stores two new contract types, revenue sharing (RS) and FLF, in addition to traditional linear pricing. RS and FLF contracts are similar in that they offer lower upfront prices per tape in exchange for a portion of the revenue and a commitment to buy a minimum (or a maximum) number of tapes. The difference between the two arrangements is that an FLF contract offers more generous per-tape prices and revenue-sharing terms in exchange for the rental store's agreeing to carry all movies that the distributor releases over a year. This bundling feature, together with the minimum and maximum purchase requirements, is what makes an FLF contract a form of conditional pricing.[41]

The authors estimate a flexible demand system and a model of retailers' choices of titles and vertical arrangements, and use these to analyze the competitive effects of an FLF contract.[42] Theoretically, there are three potential effects. An efficiency

[41]Minimum and maximum purchase requirements specify the number of tapes a rental store must purchase to satisfy the contract. Such quantity requirements have similar effects as resale price maintenance (RPM). In particular, under certain conditions, a seller can use a minimum purchase requirement to achieve the effect of a maximum RPM, or a maximum purchase requirement to achieve the effect of a minimum RPM (Tirole, 1988).

[42]Ho et al. (2012a) also analyze the distributors' decisions to offer FLF contracts and finds that, for all but one distributor, their real-world decisions are profit-maximizing. Ho et al. (2012b) discuss the welfare implications of using FLF contracts.

effect occurs when an FLF contract allows a rental store to keep a higher level of inventory of a given title, increasing its availability to consumers. A market coverage effect is observed when a store signs an FLF contract with a distributor and carries more titles from that distributor than it would otherwise. Finally, a leverage effect is present if a rental store drops titles from one distributor when it enters into an FLF contract with another.

The findings indicate that FLF contracts have a positive effect on consumer surplus.[43] First, the results indicate that the leverage effect is negligible; the number of titles that a rental store takes from competing distributors barely changes when it signs an FLF contract. This is not obvious and is perhaps a bit surprising because one might expect the costs of holding the tapes of the additional movies taken under the FLF contract to force rental stores to drop titles by rival distributors. However, the empirical evidence suggests that the advantageous terms of the FLF contract generate savings that stores use to purchase additional titles from competing distributors. Second, the article finds that the market coverage effect is substantial. The bundling aspect of the FLF contract induces stores to carry more movies by an FLF distributor than they would otherwise. The effect is bigger for relatively "weak" film distributors because stores carry many of the stronger distributors' titles even without an FLF contract.[44] The negligible leverage effect and the strong market coverage

[43]We focus on the impact on consumer surplus because it is the measurement relevant for antitrust analysis under U.S. law. However, the effect on total welfare can be negative if the profit losses to a distributor are larger than the gains to rental stores and consumers. This can happen if the distributors that do not offer FLF contracts in the real world offered FLF contracts in a counterfactual scenario. In such a case, the losses from lower upfront tape prices may outweigh the gains from selling more titles (Ho et al., 2012b).

[44]Indeed, it is these relatively weak distributors that benefit from offering FLF contracts. The stronger movie distributors do not benefit and do not offer FLF contracts in the real world.

effect expand the assortment of titles, which increases consumer surplus.[45]

Third, the analysis also finds that there is a positive efficiency effect, driven by the fact that lower upfront per-tape prices ameliorate the double marginalization problem.[46] The impact of this efficiency effect is particularly large for titles that a store would have taken under linear pricing in the absence of an FLF contract.[47] Furthermore, the efficiency effect under an FLF contract is much larger than what revenue-sharing terms can achieve, because stores purchase the most popular titles under linear pricing to avoid sharing the revenue. The increased holdings of inventories induced by an FLF contract improve the availability of products, which further increases consumer surplus.[48]

A more detailed look at the FLF contract reveals the different mechanisms through which its terms affect consumer welfare. The bundling aspect is the main factor driving the market coverage effect. By forcing a store to forgo taking a title under linear pricing, bundling also strengthens the efficiency effect. The lower upfront price, the revenue-sharing terms, and the minimum purchase requirement also induce firms to buy larger inventories. Finally, the bundling term strengthens the leverage effect, while the lower upfront price and revenue sharing weaken it.

[45]The effect on consumer surplus is nevertheless constrained by the fact that rental stores are predicted to carry the most popular titles even without FLF contracts. Thus, the additional movies that stores take as a result of the FLF contract tend to cater to smaller audiences with idiosyncratic preferences, which contributes only marginally to the estimate of overall consumer surplus

[46]Double marginalization occurs when an upstream firm sells inputs to a downstream firm with a markup and the downstream firm charges final consumers a markup as well. This is suboptimal for the upstream firm because the downstream firm purchases fewer inputs compared to what a vertically integrated firm would choose.

[47]This is true because the drop in the upfront price is much larger under linear pricing than under RS.

[48]The authors assume that retailers do not re-optimize their rental prices when they adopt an FLF contract. This assumption rules out impacts on consumer surplus through the retail price channel.

Aside from the terms of the FLF contracts, there are a few other factors that determine their overall competitive effect. First, movie distributors introduced the FLF contract to augment existing pricing options available to rental stores rather than to replace them. As long as rental stores can obtain the same linear prices, the additional vertical pricing option likely benefits rental stores and final consumers.[49] Furthermore, linear prices can "discipline" the terms of the FLF contract because stores can choose linear pricing if they are not satisfied with their terms. Second, one of the factors driving the negligible leverage effect is the low cost of holding inventory. The authors explain that a store effectively faces no capacity constraints because it can display titles spine-forwards or put additional tapes in a storage room. If this were not so, the cost of storage would be higher, possibly giving rise to a leverage effect that could harm consumers. Last, the lack of anticompetitive effect, together with the fact that non-dominant distributors offer FLF contracts, reinforces the idea that such contracts are less likely to harm competition when used by weaker, rather than dominant, players.

3.3.3 Ocean Shipping

Ocean shipping differs from all other industries considered in this article because it enjoys partial exemption from antitrust laws. In particular, ocean carriers are allowed to participate in legal cartels, called "conferences," and to engage in price and quantity

[49]Of course, it is also possible that distributors simultaneously introduce an FLF contract and raise linear prices to force rental stores to accept the FLF contract. Such a strategy can have anticompetitive effects. A similar situation is analyzed by Greenlee et al. (2008).

fixing.[50] The impact of the conferences' preferred form of pricing, dual-rate loyalty contracts, is analyzed by Marín and Sicotte (2003). Under this form of conditional pricing, a cartel offers its customers a lower rate for shipping services as long as they do not use the services of non-cartel carriers. If customers do not satisfy the exclusivity requirement, they must pay the higher, non-contract rate.[51]

The use of dual-rate contracts was the focus of a protracted legal and legislative battle that lasted from the late 1950s to the early 1960s. Proponents of the contracts argued that they allowed carriers to provide stable rates and regular shipping services of high quality. Opponents, on the other hand, claimed that the main purpose of the contracts was to deter entry and augment cartel members' market power.

The authors identify seven court actions and legislative developments that affected the likelihood of the dual-rate contracts remaining legal. If the purpose of the contracts was to prevent entry and raise rates without providing a substantial benefit to customers, any event that casts doubt on the continuing legality of dual-rate contracts should harm the financial prospects of cartel members and improve them for customers (i.e., exporting firms). This in turn should be reflected in these firms' stock returns. The authors conduct an event study and confirm that the stock indexes of ocean shippers and net exporting industries moved in opposite directions during the seven selected periods.[52] This leads them to conclude that loyalty contracts enhanced

[50] Ocean shipping benefits from antitrust exemptions not only in the United States but in European and other countries as well.

[51] In some cases, customers that break the contract must pay even larger damages.

[52] The authors focus on net exporting industries because they surmise that a decrease in rates brought about by a ban on dual-rate contracts should benefit exporting firms but harm importing firms, thus benefiting the industry on net.

market power but did not lead to efficiencies that were passed on to customers.[53] Even though these results may not currently apply to the ocean shipping industry because the legal framework has been amended since the 1960s, they provide evidence of the potential negative effect of conditional pricing on competition in an industry that enjoys some protection from antitrust laws.[54]

3.3.4 Smartphones and Video Games

Network effects are a distinctive feature of the mobile telecommunications and video games industries.[55] This characteristic encourages rivals to compete for larger customer bases. The competition for customers can be a motivating factor in firms' decisions to use conditional pricing.

Smartphones

Sinkinson (2020) analyzes the competitive effects of exclusive contracts in the telecommunications industry. His study focuses on the agreement between AT&T and Apple for exclusivity for the first-generation iPhone, which attracted a lot of attention when it was announced in 2007. Opponents of the deal were concerned that it would lead to higher prices and limited choice for consumers, while proponents claimed that it

[53]The authors conduct a similar analysis with net importing industries, whose stock indexes should move in the same direction as those of the ocean shippers. It provides weaker support for the hypothesis that loyalty contracts are used for exclusionary purposes. The authors speculate that a possible reason for this is that the largest firms in net importing industries drive movements in the industry indexes and are also large exporters who might benefit from abolishing dual-rate contracts.

[54]Cartel members can coordinate their actions and achieve the outcome of a much larger firm or even a monopolist (Tirole, 1988). Thus, it may be possible to extend the results for the ocean shipping industry to other industries dominated by a single large firm under certain conditions.

[55]Positive network externalities, or network effects, exist when a good or service becomes more valuable as more people use it (Tirole, 1988).

would encourage wireless carriers to innovate.

Sinkinson builds a model in which exclusivity allows a carrier to differentiate the handset-network bundles it offers consumers not only through the quality of wireless service but also through product variety.[56] This additional differentiation may allow a carrier to charge a higher markup. Furthermore, if prices are strategic complements, the higher price on the differentiated bundle leads to higher prices on all other bundles in equilibrium.[57] This effect is known as "softening of price competition." If demand for handsets is less sensitive to price than demand for wireless service, softened price competition for wireless service can increase a carrier's profits sufficiently to compensate the handset manufacturer for the forgone opportunity to sell to other wireless carriers.

The author estimates a model of consumer demand that accounts for the durable nature of the good and uses it to simulate counterfactual scenarios and measure the effects of exclusivity. The first analysis calculates AT&T's and Verizon's willingness to pay for the exclusive contract by comparing each firm's profits when it obtains exclusive rights to sell the iPhone to its profits when its rival obtains the exclusive rights. The outcome is that AT&T has higher willingness to pay only after equilibrium price adjustments are taken into account, which underscores the importance of modeling the equilibrium price changes. The results are driven by the fact that AT&T offers lower quality service than Verizon and without the iPhone it attracts

[56]The model in Sinkinson (2020) builds on the theoretical model developed in Rey and Stiglitz (1995).

[57]In game theory, players' actions (usually choice of price or quantity) are strategic complements if an increase by one player leads the other players to increase their strategic variable as well (Tirole, 1988).

fewer customers and has to cut its monthly service fees. At the same time, Verizon's higher quality network insulates it from price competition and makes it less dependent on the iPhone in the counterfactual. Thus, exclusivity raises retail prices and limits consumer choices, which decreases consumer welfare. Restricting choice by making the iPhone available on only one carrier harms consumers that switch to AT&T to get the iPhone by forcing them to pay early termination fees (if they are on a two-year contract) and by reducing the quality of their network (if they switch from a carrier with a higher-quality network). Non-AT&T consumers who would have purchased the iPhone from their carrier absent the exclusive deal are also harmed by being constrained to using a less preferred handset.[58]

Another counterfactual reveals that manufacturers of Android-based smartphones would make approximately $1.4 billion less in profits if the iPhone were available on all carriers. This demonstrates that the exclusive contract between AT&T and Apple created strong incentives for entry into the smartphone market. The article does not estimate the net welfare effect of exclusivity because the change in the likelihood of entry brought about by the exclusive contract cannot be estimated given the available data. The counterfactual analyses, however, demonstrate that exclusivity can generate powerful competing forces by restricting choice and softening price competition, which harms consumers in a static setting, and by creating entry and innovation incentives, which benefit consumers in a dynamic setting.

[58]The exclusive dealing arrangement between Apple and AT&T can be seen as a way to raise rivals' costs by foreclosing their access to an important input, which limits their ability to differentiate the network-handset bundles they offer. However, the existence of substitute handsets and the ability of the other carriers to differentiate their offerings through exclusive contracts of their own limits the impact of AT&T's exclusivity.

Video Games and Consoles

Lee (2013) conducts another study of the effects of exclusivity in an industry with network effects–video games. The industry is comprised of console manufacturers, which produce the platforms needed to play games; developers, which create games; and publishers, which bring games to market. A title can become exclusive to a particular console as a result of vertical integration, a contract, or a voluntary decision by the developer.[59] The author focuses specifically on the industry's sixth generation, during which Sony released PlayStation 2 (PS2), the successor to the highly successful PlayStation, while Nintendo and Microsoft entered the market a year later with their own platforms, Game Cube (GC) and Xbox (XB).[60] This setting allows the author to empirically analyze the possible pro- and anticompetitive effects of exclusivity. In the context of the video game industry, theory predicts that exclusive arrangements can limit consumer choice and lead to entry deterrence and rival foreclosure but also that they can encourage investment, solve coordination problems, and help entrants gain a foothold in an established industry.

The author estimates a model of dynamic consumer demand for both video games and consoles that takes into account the fact that consumers are forward looking and platforms are durable goods; and a model of hardware adoption by software developers that weighs the costs and benefits of exclusivity and multihoming. Modeling both

[59]Video games created by a vertically integrated entity are called "first-party" titles, while those produced by independent developers are called "third-party" titles. In some cases, the console manufacturer and the developer sign a contract that makes a title exclusive to the particular console in exchange for financing. In other cases, the developer voluntarily makes the title exclusive if the "porting" costs of making the title compatible with other platforms ("multihoming") exceed the benefits of reaching a wider audience.

[60]Over that year Sony sold 5 million PS2 consoles.

sides of the market allows agents to react to past and future actions of other agents, which is an important feature of consumer and firm behavior. Based on these models, the author analyzes the set of market outcomes that would have been obtained absent exclusive arrangements. The counterfactuals indicate that a ban on exclusives benefits the incumbent firm at the expense of entrants, while also increasing consumer surplus.[61] Hardware and software sales increase by 7 percent and 58 percent, respectively, both driven by higher PS2 and lower GC and XB sales of consoles and titles. Consumer welfare increases by $1.5 billion.

Two facts are driving the counterfactual results. First, in the real world GC and XB have a higher-quality stock of exclusive titles than PS2. As a result, PS2 benefits more by gaining access to its rivals' exclusive titles than by retaining exclusivity over its own. Second, as the incumbent, PS2 has a larger installed base, which attracts developers that want to reach a wider audience. As almost all hit titles become available for PS2 following the ban on exclusives, the incentive to purchase competing consoles disappears. The two factors together lead to a large increase in sales of PS2 consoles and titles at the expense of GC and XB. The same mechanisms also drive the gains in consumer surplus. PS2 owners get access to a much wider range of hit titles, while most consumers who own multiple platforms can play their preferred games on PS2 without needing to purchase additional consoles.[62]

[61] We focus on the results from the counterfactual in which all titles are free to re-optimize the set of consoles to support. In addition, Lee considers two other counterfactuals as well: one in which PS2 loses its exclusive titles while GC and XB keep theirs; and another in which all titles are forced to be compatible with all consoles. In all three counterfactuals, banning exclusives increases consumer welfare.

[62] The counterfactual analysis is "partial," which means that the quality and set of products are kept fixed and that platform manufacturers are not strategic. Modeling all these decisions is computationally infeasible, but the author conducts robustness checks in which he varies the price of

The telecommunications and video game industries illustrate how firms can use exclusivity to differentiate themselves and expand their customer base. In particular, exclusive arrangements can lead to higher prices and limit choice, while also creating entry incentives and helping entrants compete against an incumbent. Evaluating the impact of such conduct is particularly challenging because it requires weighing short-term harm against possible long-term benefits to consumers.

3.4 Conclusion

The reviewed empirical articles demonstrate the range of competitive effects that CPPs can have. FLF contracts are estimated to have a positive effect on consumer welfare in the video rental industry. In the confections industry, AUDs can have exclusionary effects, but they also motivate the downstream firm to exert more effort and may benefit consumers through better availability and variety of products. Exclusive dealing can similarly foreclose rivals in the beer industry, but only in rural areas where there are presumably fewer available distributors. Despite the presence of foreclosure, this conduct has only a very small negative impact on consumer welfare and is likely to have an important efficiency-inducing motivation. The impact of exclusivity is more difficult to evaluate in the video game and smartphone industries because it leads to short-run consumer harm while simultaneously creating entry incentives that can have beneficial effects in the long run. Finally, loyalty discounts can

consoles, the quality of first-party titles, and the magnitude of the porting costs. The results indicate that the prohibition of exclusives is still detrimental to entrants and beneficial to consumers, although consumer welfare gains are substantially diminished in some cases.

have anticompetitive effects as suggested by the case of ocean shipping.

The reviewed articles reveal not only the variety of possible competitive effects, but also the importance of the specific form of the arrangement at issue and the particular market characteristics in influencing these effects. CPPs are more likely to be anticompetitive when dominant firms employ them, when market features force firms to drop competitors' products to comply with the arrangement, and when substitute products or alternative distributors are not widely available. This list of characteristics that affect the likelihood and degree to which CPPs can have anticompetitive effects is by no means exhaustive. Rather, it points out only some of the market features that should be considered when evaluating the impact of conditional pricing. Although the wide variety of arrangements and the diversity of market characteristics makes reaching general conclusions about the competitive effects of CPPs very challenging, the agencies and courts would do well to draw on the findings of the existing empirical work.

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