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ABSTRACT

This dissertation focuses on the interaction of consumer and firm behavior with regulation in credit and health markets. Governments often implement a variety of regulations in these markets, which shape consumer and firm behavior in the marketplace and, ultimately, welfare. The extent to which such regulations achieve their intended goals is an empirical question. In this dissertation, I use a combination of economic models, large datasets and modern econometric techniques to inform this margin regarding three relevant policies.

The first chapter, co-authored with Alberto Sepúlveda, studies the effects of interest rate caps in credit markets. Interest rate caps are widespread in consumer credit markets, yet there is limited evidence on its effects on market outcomes and welfare. Conceptually, the effects of interest rate caps are ambiguous and depend on a trade-off between consumer protection from banks' market power and reductions in credit access. We exploit a policy in Chile that lowered interest rate caps by 20 percentage points to understand its impacts. Using comprehensive individual-level administrative data, we document that the policy decreased transacted interest rates by 9%, but also reduced the number of loans by 19%. To estimate the welfare effects of this policy, we develop and estimate a model of loan applications, pricing, and repayment of loans. Consumer surplus decreases by an equivalent of 3.5% of average income, with larger losses for risky borrowers. Survey evidence suggests these welfare effects may be driven by decreased consumption smoothing and increased financial distress. Interest rate caps provide greater consumer protection in more concentrated markets, but welfare effects are negative even under a monopoly. Risk-based regulation reduces the adverse effects of interest rate caps, but does not eliminate them.

The second chapter, co-authored with Juan Pablo Atal and Morten Sæthre, studies quality regulation in pharmaceutical markets.[1] Quality regulation attempts to ensure quality and to foster price competition by reducing vertical differentiation, but may also have unintended consequences through its effects on market structure. We study these effects in the context of pharmaceutical bioequivalence, which is the primary quality standard for

1. An early version of this paper was published in Atal (2016). This chapter is a complete version of that paper, and includes a theoretical model along with additional data and results that were not included in that earlier version.

generic drugs. Exploiting the staggered phase-in of bioequivalence requirements in Chile, we show that stronger quality regulation decreased the number of drugs in the market by 25%, increased average paid prices by 10%, decreased total sales by 20%, and did not have a significant effect on observed outcomes related to drug quality. These adverse effects were concentrated in small markets. Our results suggest that the intended effects of quality regulation on price competition through increased (perceived) quality of generics were overturned by adverse competitive effects arising from the costs of complying with the regulation.

The third chapter, co-authored with Carlos Noton and Benjamín Vatter, investigates the role of vertical integration between insurers and hospitals.[2] The welfare effects of vertical integration are ambiguous. Cost efficiencies and the elimination of double marginalization may offset increases in market power and incentives to raise rivals' costs. To study the effects of vertical integration between insurers and hospitals, we develop a model of bargaining and competition. Integrated firms have incentives to increase hospital prices to rivals to steer demand to integrated partners. We estimate the model using administrative data on claims and plans from Chile, where vertically integrated hospitals account for half of all admissions. Our estimates imply that steering incentives are significant and that vertical integration decreases welfare.

2. This chapter started after my co-authors and I realized that we were working independently but simultaneously on the same question and with the same data in July, 2016. After that, we decided to horizontally merge and this chapter is the result of that. Their initial project is published as Benjamín's masters thesis (Vatter, 2016).

CHAPTER 1

PRICE REGULATION IN CREDIT MARKETS: A TRADE-OFF BETWEEN CONSUMER PROTECTION AND CREDIT ACCESS

1.1 Introduction

Consumer credit penetration has increased steadily over recent decades and there is currently more that $41 trillion U.S dollars in household debt in the world, equivalent to around 40% of GDP across countries.[1] The growth of household debt has sparked a debate among researchers and policymakers about whether consumer credit is under- or over-supplied. The former argue that households are credit constrained due to market power or adverse selection, whereas the latter argue that moral hazard or behavioral biases induce households to borrow too much (Zinman, 2015).[2] This disagreement motivates a varied array of regulatory interventions that seek to increase or restrict credit access, and often coexist in the marketplace. While the regulation of consumer credit markets has been in the policy agenda for decades, its relevance increased substantially after the 2008 financial crisis (Campbell et al., 2011a,b).[3]

Interest rate regulation has historically been one of the main policy instruments in consumer credit markets (Temin and Voth, 2008). Several developed and developing countries implement some form of interest rate regulation nowadays, often adopting interest rate caps (Maimbo and Henríquez, 2014). On the one hand, regulators argue that interest rate caps limit lender usury and exercise of market power for loan pricing, as

1. Calculations based on data from the Global Debt Database by the International Monetary Fund (Mbaye et al., 2018), for 82 developed and developing countries with available data for 2016. Beyond the average, most countries display increases in household debt as a share of GDP over time and there is substantial cross-sectional dispersion. See Figure A.1 for an illustration of this evolution for a sample of countries.

2. Relevant examples of research providing evidence for households being credit constrained are Gross and Souleles (2002), Adams et al. (2009), Jappelli and Pistaferri (2010) and Mian and Sufi (2011), among others; whereas relevant examples of research that suggests households might be over-borrowing are Bertrand and Morse (2011), Stango and Zinman (2014), Bhutta and Keys (2016) and Beshears et al. (2018), among others.

3. For example, the U.S. government introduced the CARD Act in 2009 and then established the Consumer Finance Protection Bureau (CFPB) in 2010 to improve regulation and overall functioning of consumer credit markets, and European Commission has also taken steps in a similar direction (European Commission, 2015).

well as their ability to exploit consumers' behavioral biases. On the other hand, detractors argue that interest rate regulation makes risky borrowers unprofitable and therefore may limit credit access. Therefore, welfare implications of stronger interest rate regulation are potentially heterogeneous along borrower risk, as it benefits *protected* borrowers and harms *excluded* ones. Despite the ambiguity in its welfare effects, research analyzing this type of regulation is somewhat limited, at least partially due to a lack of comprehensive data and compelling research designs.

In this paper, we aim to make progress on understanding the consequences of regulating consumer credit markets by studying the equilibrium effects of interest rate caps on prices, credit access, loan performance, and consumer welfare. We exploit the Chilean consumer credit market for consumer loans as a setting, which is attractive because it combines policy variation in interest rate regulation with extensive administrative data. Interest rate regulation in this setting takes the form of interest rate caps, which vary across loan size, and were substantially strengthened between 2013 and 2015 for part of the market. Throughout that period, interest rate caps decreased by between 17 p.p and 24 p.p for loans smaller than $8,000, leaving larger loans unaffected.[4]

For our analysis, we combine this policy variation with comprehensive data on the supply and demand for consumer credit. This data includes administrative data on contracts, repayment behavior and credit histories for each consumer in the market, and data on loan applications for a large share of such contracts. Moreover, we complement this data with a survey that we designed and collected from a sample of borrowers in order to describe their shopping process and support the interpretation of welfare effects. We focus on unsecured consumer loans, a simple product that 15% of households hold (EFH, 2014). The average contract is roughly for a three-year loan of $6,800 with an interest rate of 23 p.p, and there is substantial dispersion in interest rates.

We start by providing evidence for the effects of interest rate regulation on the distribution of transacted interest rates in the market. We find that the policy change made interest regulation binding. At the onset of the policy change, in November 2013, as much

4. Unless otherwise noted, all monetary units are measured in U.S. dollars of December 31st, 2016. For reference, the exchange rate at that point was of $667.29 Chilean pesos per U.S. dollar, according to the Central Bank of Chile.

as 31% of contracts directly affected by it were offered at an interest rate higher than their corresponding interest rate cap by the time the policy was fully in place, in December 2015. We show that the policy shifted the distribution of interest rates downwards, and induced substantial bunching at the interest rate cap. One interpretation of this pattern is that banks hold market power, since under perfect competition banks would choose not to offer loans that were exposed to this regulation at rates below the interest rate cap. However, this interpretation is not conclusive, as the pool of applicants might also change under stronger interest rate regulation.

To provide evidence of the market-level effects of interest rate regulation, we exploit the variation across loan size and time in the intensity of interest rate regulation in a differences-in-differences framework. We find that the policy change had large effects on prices, quantities, and loan performance. Average transacted interest rates decreased by 9% (2.6 p.p) in response to the policy change. The quantity of credit in the market also decreased, as the number of loan contracts went down by 19%. Part of this effect stems from a decrease in loan applications driven by riskier borrowers. Both price and quantity effects are stronger for riskier borrowers, who were more exposed to interest rate caps due to risk pricing by banks. In particular, transacted interest rates for risky borrowers decreased by 11% (3.3 p.p) and the number of loans for them decreased by 24%. Indeed, the borrower pool became safer and default rates decreased by 18% (1.15 p.p).

Overall, this evidence suggests that interest rates caps have strong effects on credit markets, when binding. The trade-off between credit access and consumer protection is apparent in these results. On the one hand, our estimates imply that 151,027 loans for an amount of $361.6 million in loan contracts yearly were not signed due to stronger interest rate regulation. On the other hand, average monthly payments decreased by $3.26 in the regulated segment of the market, adding up to an aggregate reduction of $31.7 million in present value per year.

Motivated by this evidence, we develop and estimate an equilibrium model of the market for consumer loans, with two objectives. First, by developing a model we are able to estimate borrowers' willingness to pay for loans and banks' costs using the variation available in our data. Having those inputs, we can then develop a welfare analysis of

interest rate regulation. Second, the model allows us to study the relationship between the effects of interest rate regulation and the competitive environment, and the effects of counterfactual designs of interest rate regulation.

Our model consists of three stages that cover application, pricing and repayment. First, consumers decide whether to apply for loans or not given their credit needs. Application choices depend on expected approval probability, expected loan price, and application cost. Second, consumers shop across banks that offer homogeneous loans produced at heterogeneous costs. We model this process as an English auction, in which consumers shop across banks for the best contract offer. In equilibrium, the bank with the lowest cost signs the contract with the consumer at an interest rate that leaves the second-lowest cost bank with zero profits. The source of market power in our model is thus cost heterogeneity. This modeling choice has also been adopted in recent work on markets with bargained prices (Salz, 2017; Allen et al., 2018), and overcomes a common problem when working with contract-level data, which is that the econometrician only observes chosen contracts rather than the full choice set that consumers face.[5] By modeling the market as an auction, we rationalize observed contract prices as a function of banks' latent cost structure. Third, repayment risk is realized. The model incorporates both imperfect competition and adverse selection. The comparative statics of the model are in line with our evidence on market-level effects and support our interpretation of it.

We estimate our model using data on loan applications, approvals, prices and repayment. On the demand side, we estimate that consumers facing lower approval probabilities are less likely to apply for loans; and that riskier borrowers have a higher willingness to pay for credit and are less price-sensitive than safe borrowers. In terms of repayment, we find that the main correlate of repayment is borrower risk score. Moreover, we find no compelling evidence of adverse selection along the extensive margin of loan applications, after conditioning on borrower risk scores. On the supply side, our cost estimates reveal

5. This approach provides both a reasonable characterization of the market and is convenient for empirical work in our setting. It is often the case in markets with bargained prices that only transacted prices are observed, while prices of the other options in consumers' choice sets remain unobserved. Some papers overcome this challenge by predicting prices using observed transactions, but this may be problematic due to selection concerns (e.g., Crawford et al. 2018). Our approach avoids this prediction step.

substantial cost heterogeneity that stems from differences across banks, banks' incumbency advantages over previously related borrowers, and idiosyncratic bank-borrower cost heterogeneity. Moreover, cost estimates reveal substantial bank market power: the average mark-up over bank marginal cost is of 29%, of which market power accounts for 90% and borrower risk only accounts for 10%.

Adopting a revealed preferences approach, we use our model to estimate welfare effects of interest rate regulation. We find that expected consumer surplus decreased by an average and median of $82.47 and $ 40.34 per month respectively, equivalent to 3.5% and 1.7% of average income. However, not all consumers in the market lose consumer surplus under stronger regulation: we find that 16.2% of consumers benefit from it, although the gains of this group are substantially smaller than the losses of those for whom expected consumer surplus decreases. Borrowers are heterogeneous in their exposure to interest rate regulation and in their willingness to pay, which generates substantial heterogeneity in consumer welfare effects. In particular, risky borrowers experience average decreases in expected consumer surplus three times those of safe borrowers, because they are more exposed to interest rate regulation in the presence of risk pricing, and because they display both higher willingness to pay and lower price sensitivity. Moreover, profits per consumer decrease by $2.41 per month, which adds up to 18% of profits in the market, and implies that overall welfare decreases.

Evidence from our survey allows us to complement and interpret our estimates of welfare effects. In particular, we study how the implications of economic hardships for households vary depending on whether they are able to access bank credit to deal with those hardships. We show that households that deal with hardships with bank credit are less likely to decrease consumption and register unpaid bills or loan payments. These results are consistent with our estimates of negative consumer welfare effects of interest rate regulation as reflecting that reduced credit access limits consumption smoothing and increases the risk of financial distress for households.

An important motivation for interest rate regulation is to protect consumers from the exercise of market power by banks. We exploit our estimated model to study how the effects of interest rate regulation vary across markets with different degrees of concentration. We

simulate equilibrium outcomes for a range of scenarios in which we sequentially merge banks, from the baseline market structure to the monopoly case. We find that adverse welfare effects are smaller in more concentrated markets, which suggests that the consumer protection role of interest rate regulation increases in less competitive markets. However, we find that stronger interest rate regulation decreases welfare even under a monopoly.

The design of interest rate regulation is strikingly simple in most countries. Few countries implement designs that go beyond having interest rate caps specific to a few loan size and type brackets.[6] The mismatch between unsophisticated regulation and sophisticated risk pricing by banks reinforces the trade-off between consumer protection and credit access by increasing the exposure of risky borrowers to interest rate caps. We use our estimated model to address the extent to which this mismatch exacerbates the potential for adverse effects. In particular, we study how risk-based interest rate caps, which combine the benefits of risk-based pricing in terms of dealing with borrower heterogeneity, with the potential of interest rate regulation in terms of limiting the exercise of market power by banks. In a simple example, we find that this design reduces the average welfare loss of interest rate regulation by 27%, without substantially increasing average loan prices or bank profit margins.

Overall, these results show that while interest rate regulation is meant to protect consumers facing high interest rates in the market, it mostly harmed consumers' credit access and overall welfare in this setting. We highlight that theoretical predictions regarding credit access and welfare are ambiguous and therefore interest rate regulation might improve outcomes in other settings. Regardless, our results inform the design of interest rate regulation for consumer credit by providing a conceptual framework that guides the mapping between the implications of interest rate regulation and market characteristics, such as market structure and borrower heterogeneity.

6. For example, several states in the U.S. have a single interest rate cap on consumer loans, and there is a federal interest rate cap at 36% for payday loans. In Europe, many countries have designs that impose caps at a mark-up over the average interest rate in the market, including Germany and Italy. Other countries, such as Belgium and France, have somewhat more sophisticated designs that allow the cap to vary by a few loan type and size brackets. In the case of the Chilean market, the design imposes differentiated interest rate caps for a small number of loan-size brackets. See Maimbo and Henríquez (2014) for further examples of interest rate regulation in credit markets across countries.

This paper contributes to different branches of the literature. First, it contributes to a literature that studies the effects of interest rate regulation, within which findings range between no effects and negative effects on credit access. However, the most recent research finds mostly negative effects on that margin when regulation has been binding (Bodenhorn, 2007; Temin and Voth, 2008; Benmelech and Moskowitz, 2010; Zinman, 2010; Rigbi, 2013; Fekrazad, 2016; Melzer and Schroeder, 2017). However, many of these papers focus on payday loans in the U.S. In some cases, they focus on a single lender or a single market. Moreover, most of the previous work adopts reduced form approaches and focuses on credit access as their main outcome. In this paper, we provide a conceptual framework for the equilibrium analysis of interest rate regulation, which allows for the estimation of welfare effects and exploits administrative data from a full market as an empirical application. Moreover, we emphasize the role of two pervasive attributes of credit markets, which are imperfect competition and borrower risk heterogeneity. Our empirical application is also studied by Hurtado (2015), SBIF (2017b) and Schmukler et al. (2018), all of which adopt reduced form approaches to analyze the effects of the policy change on credit access.

Second, this paper contributes to a recent literature on imperfect competition in selection markets. This literature emphasizes that the effects of different policies on selection markets depend on the degree of competition (Veiga and Weyl, 2016; Mahoney and Weyl, 2017; Lester et al., 2017). We relate to this literature by empirically studying the relationship between the effects of interest rate regulation and market structure. Recent research in this literature develops empirical models that allow for adverse selection, imperfect competition, and product differentiation (Einav et al., 2012, 2013; Kawai et al., 2018; Agarwal et al., 2018; Allen et al., 2018; Benetton, 2018; Crawford et al., 2018). In our model, we allow for adverse selection along the extensive margin of consumer credit and embed imperfect competition in bank cost heterogeneity. We then use this model to study equilibrium effects of interest rate regulation on different margins of the market including prices, quantities, loan performance and welfare.

Finally, this paper also contributes to other branches of the literature in household finance. First, we contribute to a recent literature that studies the effects of regulation on other margins of contract pricing in credit markets, also exploiting administrative data

(Agarwal et al., 2015; Benetton, 2018; Nelson, 2018). We focus on a key aspect of contract design: interest rates. Second, we also contribute to a literature that focuses on the welfare implications of access to expensive credit, which finds mixed effects (Melzer 2011; Morse 2011; Gathergood et al. 2018; Skiba and Tobacman 2018, among others). While most papers in this literature focus on payday lending, we study a segment of the market in which interest rates are lower and risk composition is safer than what common in payday lending. For our setting, we measure the welfare effects of a common class of regulation that affects credit access.

The remainder of the paper is organized as follows. In Section 1.2, we describe the setting and data we exploit for our empirical analysis. In Section 1.3, we provide evidence for the market-level effects of interest rate regulation. In Section 3.3, we develop an equilibrium model of supply and demand of consumer loans, and in Section 3.4 we estimate it. In Section 1.6, we use the estimated model to measure welfare effects and in Section 3.5 we use it to study outcomes under counterfactual competitive environments and policy designs. Finally, Section 3.6 concludes.

1.2 The Chilean Credit Market

Our empirical application focuses on the Chilean credit market, and we focus on unsecured consumer loans. Consumer loan contracts can be characterized basically by their interest rate, term and amount. Banks require no collateral on these loans.[7] Every year, more than 1.1 million contracts are signed in this market, adding up to more than 7 billion U.S. dollars in credit. While the consumer loan market is large, it is not the only source of consumer credit in this market. The two main alternative sources of consumer credit are credit cards and credit lines (SBIF, 2017b), both of which have increased its market penetration throughout the period we study.[8] These products are covered by the same interest rate

7. Consumer loan contracts impose prepayment penalties on borrowers. Borrowers may prepay part or all of the standing balance of a loan as long as the amount paid is higher than 25% of it. Upon prepayment, the borrower must pay a penalty equivalent to one month of interest on the prepaid balance.

8. Figure A.2 displays the evolution of household debt in credit cards and credit lines. Both products have increased its market penetration throughout the period we study both in terms of number of consumer and total amount of debt, although without a noticeable pattern around the policy change. Moreover, average

regulation described in Section 2.2.2 below. Payday loans, a relevant source of expensive credit in other countries, are not widely available in Chile. Moreover, informal lending is a relatively small segment of the market, and only 7% of households hold some form of informal debt (EFH, 2018).[9]

The market is concentrated, as the combined market share of the 3 largest banks is 56% and that of the 5 largest banks is 76%. We focus on the 15 banks that offer consumer loans in the consumer credit market, which covers 92% of consumer loan contracts (SBIF, 2017b).[10] The remaining 8% of market share consists of credit unions that offer loans paid through employers, which are a somewhat different product that we do not consider in our analysis.

Regarding risk assessment by banks, there are no market-wide risk scores in this setting such as FICO scores in the U.S. Instead, there are three sources of information that banks may use for risk assessment, namely: (i) comprehensive information on consumer covariates and credit history across all banks in the market that the regulator gathers and provides to banks; (ii) information banks may ask borrowers for when loans are requested; and (iii) risk scoring services provided by private firms. We have access to the first of these sources, which provides substantial information on borrower risk, as also emphasized by Foley et al. (2018) in their study of the role of information for bank lending using this same data. We use this data to construct measures of predicted default risk in Section 1.2.2.

Consumer debt is very common in Chile. The 2014 Survey of Household Finance (*Encuesta Financiera de Hogares*, EFH), conducted by the Central Bank of Chile, provides a

credit card and credit line balances across consumers—which combine both transactional and borrowing uses of these instruments in similar shares, according to industry sources—are less than a fourth of the average consumer loan in the market, which suggests these sources of credit are most often used to finance smaller expenses than consumer loans. In work in progress, we are analyzing the relationship between these markets in more detail.

9. This statistic covers several sources of informal credit, including family and friends, informal lenders, pawn shops, among others. Figure A.3 displays the evolution of the share of households that hold informal debt around the policy change, which has remained between 2% and 10% since 2007, and which does not display any noticeable pattern associated to the timing of the policy change. In fact, it remained almost constant between 2014 and 2017.

10. For comparison, this market structure is somewhat more concentrated than that in the U.S, where the average number of banks in a local market is around 45 (Aguirregabiria et al., 2017), but similar to those in Canada and the U.K, where 8 and 6 banks respectively dominate most of the credit the market (see Allen et al. 2018 and Benetton 2018, respectively).

picture of the relevance of consumer loans for household finance around the policy change (EFH, 2014). As much as 63% of households have some form of consumer debt and 15.4% have consumer loans. Among households with consumer debt, the average debt to income ratio is around 5 and every month households allocate 20.5% of income to debt repayment (SBIF, 2017a).[11]

1.2.1 Interest Rate Regulation

Interest rate regulation in the Chilean credit market is not new. Since 1929, several versions of interest rate caps on credit products have been in place.[12] We focus on a policy change enacted by Law 20,715, which aimed at further protecting low-income borrowers and providing access to credit at lower interest rates (SBIF, 2017b). This law was approved on December, 13th, 2013 and followed long-standing Law 18,010, which was in place since 1981 and subsequently modified in 1999. These laws cover virtually all credit market operations with a term of 90 days or more. The main policy tool determined by these laws is a set of interest rates caps that vary depending on loan size. These caps are called Conventional Maximal Rate (TMC, *Tasa Máxima Convencional*). The policy change we study changed both the definition of loan size brackets for interest rate caps and the formulas designed for their calculation. Interest rate caps are always measured in terms of annualized interest rates. Loan size brackets are defined in UF (*Unidades de Fomento*), an inflation adjusted monetary unit commonly used in Chile.[13]

Both before and after the policy change, interest rate caps can be summarized by a simple linear function of a lagged reference interest rate. In particular, the interest rate cap

11. In terms of utilization of loans, the share of households having consumer loans for different self-reported objectives varies as follows: 54% for household durables, 30% for clothing, 22% for debt consolidation, 11% for vehicles, 9% for medical treatment, 9% for home improvement and 5% for vacations (EFH, 2014).

12. For further detail on the evolution of interest rate regulation in the Chilean credit market, see Flores et al. (2005), Hurtado (2015), and SBIF (2017b).

13. According to the Central Bank of Chile, one UF was equivalent to 39.48 U.S. dollars on December 31st, 2016. Relevant policy thresholds are set at 50UF and 200UF. For reference, 50UF is equivalent to $1,970 and 200UF is equivalent to $7,880. We refer to these two thresholds as $2,000 and $8,000 respectively for expositional simplicity. All analyses, however, are conducted without such approximation.

for loan-size bracket ℓ at period t is given by:

$$\bar{\iota}_{\ell t} = \psi_{\ell} \tilde{\iota}_{\ell t-1} + \alpha_{\ell t} \tag{1.1}$$

such that caps $\bar{\iota}_{\ell t}$ are set as a combination of proportional and constant mark-ups over a reference rate $\tilde{\iota}_{\ell t-1}$. Before the policy change, only two loan size brackets were considered by the regulation, namely \$0-\$8,000 and \$8,000-\$200,000. For both brackets, the regulation considered $\psi_{\ell} = 1.5$ and $\alpha_{\ell t} = 0$. The reference rate $\tilde{\iota}_{\ell t-1}$ was calculated as a weighted average of interest rates for loans of size ℓ during the previous month.[14] Figure 1.1 displays the evolution of interest rate caps and shows that before the reform, interest rate caps were beyond 50 p.p and 25 p.p respectively for loans in the \$0-\$8,000 and \$8,000-\$200,000 size brackets respectively.

The reform under study made four changes to the previous regulation. First, it divided the \$0-\$8,000 size bracket into two, namely \$0-\$2,000 and \$2,000-\$8,000.[15] Second, it set $\psi_{0-8000} = 1$ while $\psi_{>8000} = 1.5$ remained unchanged. Third, it set constant mark-ups over the reference rate of $\alpha_{0-2000,t} = 21$ p.p and $\alpha_{2000-8000,t} = 14$ p.p. Fourth, the reference interest rate was set to be a weighted average of interest rates in the \$8,000-\$200,000 bracket for all size brackets. Therefore, only regulation for loans under \$8,000 was directly affected by the policy change. Moreover, the main qualitative effect of the policy was to move from a regulation based on proportional mark-ups to one based on constant mark-ups for those two size brackets, while leaving the regulation for loans in the \$8,000-\$200,000 bracket unchanged.

Had the policy been fully enacted by the December 2013, interest rate caps would have fallen instantaneously by 16.9 p.p and 23.9 p.p for loans in the \$0-\$2,000 and \$2,000-\$8,000 brackets respectively (SBIF, 2017b). Instead, the policy was phased in a staggered fashion to avoid such a sharp decrease. This transition was structured by an immediate fall of 6

14. Throughout the analysis, we ignore potential strategic incentives banks may have to increase interest rates to increase the reference rate $\tilde{\iota}_{\ell t-1}$.

15. This component of the design relates to considerations of risky borrowers being potentially excluded from the credit market by this regulation. Exclusion was indeed part of the discussion around the policy approval by the Chilean Congress. Allowing for a less strict regulation for the smaller loan size bracket aimed at reducing such concern.

p.p and 8 p.p respectively followed by quarterly decreases of 2 p.p for $\alpha_{\ell t}$. Under such a calendar, the policy was fully in place by December 2015. Figure 1.1 displays the evolution of interest rate caps after the reform. The reduction in caps for the \$0-\$2,000 and \$2,000-\$8,000 size brackets is stark, and the difference between the former and the latter is of 7 p.p. However, the cap on larger loans has remained roughly constant over the period of study. We exploit these features as identifying variation to study the effects of this regulation below.

1.2.2 Data

We use large administrative datasets collected by the regulator of the Chilean credit market, the Superintendence of Banks and Financial Institutions (*Superintendencia de Bancos e Instituciones Financieras*, SBIF). The data covers the period between January 2013 and December 2015, which subsumes the roll-out of the policy change in interest rate regulation described above in Section 2.2.2. Our population of interest is that of potential borrowers. We define potential borrowers as the broad set of consumers with some relationship with the consumer credit market, defined as having used any product offered by banks, ranging from checking accounts to mortgages. This set covered 2.5 million consumers in January 2013, as much as 25% of the working-age population in the country. We observe demographics, income and credit history for each potential borrower. We exploit two main administrative datasets: one that contains every loan contract signed and one that provides a large sample of loan applications by borrowers. In both cases, we merge borrower demographic information and credit histories.

We complement administrative data with a household survey that we designed and administered. We exploit this data to provide complementary evidence for our model assumptions and to aid the interpretation of the estimates of consumer welfare effects we obtain from our model. In particular, we collect data from 1,003 consumers who applied for loans at least twice between 2013 and 2015, and were rejected by at least one bank in that period. The objective of this sampling strategy was to target a population of risky borrowers that were likely to be affected by the policy change we study and, at the same

time, were familiar with the market. The survey collects information about financial literacy, familiarity with credit market, search and application behavior in the credit market, and the evolution of household finance over the period of interest.

Loan Contracts Dataset

The first dataset we employ is a registry of all consumer loan contracts signed in the Chilean credit market during the period of study.[16] This dataset has several features. First, both borrower and bank identifiers are available for each loan contract in the data, along with relevant information on contract characteristics including interest rate, amount, and term. Second, the dataset tracks the performance of each loan contract, which allows us to observe loan defaults and their timing. Third, the dataset provides relevant borrower attributes including age, gender, income, and county of residence. Fourth, the dataset collects the full credit history of each borrower in the system. These variables include amount of consumer and mortgage debt held, and amount of debt in 90-day default. Importantly, this is the same dataset that the regulator provides to banks for borrower risk assessment, and covers the relationships between each borrower and all banks in the market. In the absence of market-wide risk scores in the Chilean credit market, we exploit this information to construct risk scores for our analysis in Section 1.2.2. The fact that banks employ this same information when assessing borrower risk reinforces our approach. We measure all monetary variables in U.S. dollars and all interest rates in annualized terms.

Applications Dataset

The second dataset we utilize for our analysis covers a large sample of consumer loan applications for the period of study. While the loan contracts dataset covers the entire market, the coverage of the applications dataset is only partial.[17] We link the applications

16. We restrict our interest to loan originations. While renegotiation is available in this segment of the market, in practice very few borrowers renegotiate their contracts. The share of renegotiations is 6% and does not change across years in our sample.

17. Banks' reporting practices for this dataset were not as rigorous as those for the contracts dataset, as this was a new requirement for them. In particular, three banks did not report to this data to SBIF.

and contracts datasets using borrower identifiers, and we are able to match application events for 64.5% of loan contracts in the data. Given we observe all loan contracts in the contracts dataset, the implication of this partial coverage is that we are unable to observe some rejected applications. For each application in the dataset, we observe the identity of the bank and the borrower, the application date, the loan size and term for which the borrower applies, and the outcome of the application. Whenever the application is approved by the bank, we also observe the interest rate. For each application, we merge the same borrower attributes and credit history available for the loan contracts dataset.

We organize the applications dataset by constructing application events, and develop all our analysis using this definition of applications. We construct application clusters of a given borrower across—potentially—multiple banks in a short period of time. Concretely, we define an application event as a set of applications by a borrower such that no pair of applications are more than 30 days apart. We then merge these application events with loan contracts using borrower and bank identifiers.

Measuring Credit Default Risk

We exploit the availability of data on loan performance, consumer covariates, and credit history to estimate credit default risk. In particular, we estimate a logit model of default using data for the period before the policy. The model we estimate uses an indicator for loan default over the term of a loan as dependent variable, and a rich vector of borrower covariates x_i determined before signing the contract as independent variables. This is a standard risk scoring model (Ohlson, 1980). We consider different sets of variables in x_i, starting with borrower income and leverage, then adding borrower credit history variables, and finally borrower demographics and macroeconomic controls. This set of features is similar to that employed by Liberman et al. (2018) for estimating borrower risk for the Chilean consumer credit market.

Table A.1 displays estimates of different specifications of this model.[18] Overall, results

18. For the rest of the paper, we use results from a model that splits all continuous covariates in twenty bins and includes dummies for such bins as regressors. The objective of this more flexible model is to accommodate potential non-linearities in the relationship between covariates and default.

point in the expected directions: borrowers with higher income and lower leverage default less frequently. Regarding credit history variables, borrowers with more consumer debt, without previous consumer loans, and with more consumer debt under default are more likely to default; while borrowers with more mortgage debt and whose mortgage debt is not in default are less likely to default. In terms of demographics, both older and female borrowers are less likely to default. The model has reasonable predictive accuracy; it predicts 69% of loan defaults correctly out of sample. We construct our measure of credit default risk as the fitted probabilities from this model, such that riskier borrowers are those with higher risk scores in this scale. For the rest of the paper, we refer to the income risk model as that in column (1) of Table A.1 and as the history risk model as that in column (5) of Table A.1. Figure A.4 displays relationships between relevant market outcomes and our measures of predicted risk. Figures A.4-a and A.4-b display negative relationship between predicted risk and approvals, while Figures A.4-c and A.4-d display positive relationships between interest rates and predicted risk. Finally, Figures A.4-e and A.4-f display positive relationships between realized and predicted default.

1.2.3 Descriptive Statistics

The contracts dataset contains more than 3.3 million loan contracts for 2013-2015. Table 2.2 displays summary statistics for this dataset. Average annualized interest rates are around 23 p.p, but more than 10% of the loans in the dataset have rates higher than 35 p.p, which is partly what motivated the implementation of the regulation under study.[19] The average loan in the sample is about $6,700 and 33 months long, and has a monthly payment of $266, with substantial variation in these attributes.[20] Regarding the distribution of loan size across size brackets defined by the regulation in place, the share of loan contracts in the year before the policy change was 30.8%, 41.5% and 27.7% respectively for loans in the

19. Most of the price dispersion is cross-sectional. While there is variation in the funding cost of banks through time, only 1.2% of the variation in interest rates can be explained by monthly dummies. See Figure A.5 for the evolution of bank funding cost through our period of study. On the other hand, there is substantial heterogeneity across banks in interest rates: bank and month dummies jointly explain as much as 25.4% of the variation.

20. Monthly payments are calculated using the formula $p = \frac{L\iota(1+\iota)^T}{(1+\iota)^T-1}$, where L is loan amount, ι is the interest rate, and T is loan term.

$0-$2,000, $2,000-$8,000 and $8,000-$200,000 brackets. In terms of loan performance, 5% of borrowers default on payments during the first year of the loan and 11% through the loan term. The average predicted default risk is 0.11, and most of the borrowers are under 0.2.

There is substantial variation in the population of borrowers in this market. The average borrower has an annual income of $18,685 and is almost 44 years old. Moreover, 40% of borrowers are female. Most of loan contracts in our data are signed by consumers that had previously dealt with banks in the consumer credit market, and 76% of them are signed with a bank that the borrower has previously used for banking.[21] In terms of credit history, the average consumer holds $7,022 in consumer loans and $12,447 in mortgage debt. The median borrower in the contracts dataset takes out only one consumer loan throughout our sample period, although there is a group of borrowers that take several loans and the average borrower takes 1.8 loans. Finally, borrowers in the system hold relationships with multiple banks, and the average borrower is a customer of three banks.

Our applications dataset collects almost 3.7 million application events, and every month we observe 2% of potential borrowers in the market applying for a loan. Both loan amount and term are slightly larger on average in the applications dataset than those in the contracts dataset. In terms of outcomes, as much as 90% of application events end with an approval, whereas 10% of application events end with a rejection.

Additionally, there is substantial heterogeneity in market structure across local markets. We define local markets geographically as the 54 provinces in the country to provide a description of the competitive environment. The average market has 8 banks and 43 branches, although there is wide dispersion in both across markets. Most markets are dominated by a few banks. In particular, in the average market the top three banks hold 66% of market share in terms of loan contracts, and the top five banks hold 83% of it.

1.2.4 Descriptive Facts about the Chilean Consumer Credit Market

In this section, we document relevant descriptive facts of the Chilean consumer credit market in order to motivate our analysis in the rest of the paper. We focus on the relationship

21. We refer to a borrower as previously related to a bank whenever they had any product offered by that bank in the past, ranging from checking accounts to mortgages.

between relevant outcomes and behavior and two important borrower attributes, namely borrower risk score and previous relationships with banks. We focus on the period before the policy change for this descriptive analysis.

First, we focus on the main correlates of loan application behavior. Column (1) in Table 1.2 displays results from a regression of an indicator for application on borrowers risk score and a set of fixed effects for the pool of potential applicants. Estimates from such regression show that the likelihood of application increases with borrower risk score, suggesting that observably riskier borrowers are more likely to select into the market. Additionally, they also show that potential applicants with previous experience in the market are also more likely to apply for loans.

The second aspect we study is how relevant borrower attributes influence approval decisions by banks. Column (2) in Table 1.2 displays results from regressions of an indicator for loan application approval on borrower covariates. Estimates from this regression show that banks are less likely to approve applications from borrowers with higher predicted default risk, which is also documented by Figure A.4-a. Moreover, they also show that banks are more likely to approve applicants with which they hold a previous relationship.

Third, we show that previous relationships affect bank choice by borrowers. Figure A.14-a shows that there is substantial variation in the number of bank-borrower previous relationships, and few contracts are signed by borrowers new to the system. Figure A.14-b shows that the likelihood of signing a given loan contract with a previously related bank is high, and that it increases with the number of previous relationships and decreases with borrower risk. This pattern may relate to the fact that applications from previously related borrowers are approved more often, perhaps because previous relationships make applications less costly for borrowers and banks.

Additionally, we study the determinants of loan interest rates. Banks engage in risk pricing in the market through offering higher loan prices to observably riskier borrowers. Column (3) in Table 1.2 displays results of regressions of interest rate margins over banks' funding cost on borrower and contract covariates. Interest rates are increasing in borrower predicted default risk, as also documented by Figure A.4-b. Additionally, we find that even after conditioning on contract attributes, borrower default risk, and other covariates,

previous relationships affect prices: borrowers who have a previous relationship with a bank receive loan prices that are lower on average.

Moreover, there is substantial price dispersion in the market, as displayed by Figure A.15. After residualizing interest rate margins of interacted month, bank, location, loan size, term and borrower risk fixed effects, as much as 26% of the variation in interest rate margins remains unexplained. Therefore, there is substantial price dispersion even within remarkably narrow segments of the market, consistent with evidence from U.S. credit markets (Woodward and Hall, 2012; Stango and Zinman, 2016). The standard deviation of residualized interest rate margins remains high at 3.9 p.p, slightly less than a third of the unconditional standard deviation in interest rate margins.[22] One potential source of price dispersion within observably similar contracts is discretion of banks' loan officers and bargaining over prices.

Finally, we show that riskier borrowers are more likely to default, as expected. Column (4) in Table 1.2 shows the results from a regression of an indicator of loan default on borrower and contract covariates. Estimates from this regression show that observably riskier borrowers are more likely to default on loan payments, conditional on contract amount and term.

We use these facts in the rest of the paper. In particular, these facts are useful to interpret our findings for the effects of interest rate regulation in Section 1.3, in terms of heterogeneity across borrower risk. Moreover, we rely on them to motivate the structure and assumptions for the model we develop in Section 3.3.

1.3 The Effects of Interest Rate Regulation

In this section, we study the effects of interest rate regulation on market outcomes in the Chilean credit market. As described in Section 2.2.2, this policy strongly decreased interest rate caps on loans, differentially so across loan size. Using different approaches,

22. Evidence from our survey complements this fact by showing consumers are aware of this price dispersion. Figure A.16-a shows that consumers in the market perceive substantial price dispersion conditional on loan terms. In particular, the average perceived range of monthly payments in the market in our survey is 26%.

we provide evidence for price, quantity, and risk composition effects. Throughout this section, we emphasize heterogeneity in effects across borrower risk. In particular, we split the sample according to the median predicted default risk before the reform and estimate effects for low- and high-risk borrowers.

1.3.1 Evidence from the Evolution of Interest Rates

The policy change we study reduced interest rate caps between December 2013 and December 2015 for loans smaller than $8,000. As a first piece of evidence, we visually inspect the evolution of interest rates. The left column of Figure 1.2 displays the evolution of the distribution of interest rates for loans of $0-$2,000, $2,000-$8,000 and $8,000-$20,000, along with the evolution of the interest rate cap for each of those groups, for the period around the policy change. There are two relevant aspects to these figures. First, interest rate caps were mostly not binding within the treated size brackets.[23] Second, interest rate caps became increasingly binding for these groups after December 2013. On the other hand, the extent to which interest rates for loans larger than $8,000 were binding did not change noticeably over the period of study.

To further document the price effects of interest rates caps, we compare the distribution of interest rates before and after the policy change. The right column of Figure 1.2 shows the distribution of interest rates for the month before the policy change with that for the same month exactly two years after, when the policy was fully in place.[24] The policy displaced a substantial share of the density downwards for treated loan-size brackets, inducing bunching of interest rates at the interest rate caps. As much as 42% and 23% of loans of $0-$2,000 and of $2,000-$8,000 were exposed to the policy, respectively. In contrast, only 8% of loans of $8,000-$20,000 were exposed to it, and only marginally so. One interpretation for this response is as suggestive evidence of imperfect competition in this market. Had

23. Previous research has shown that interest rate caps may play the role of focal points for collusion in credit markets (Knittel and Stango, 2003). The fact that caps for loans smaller than $8,000 were not binding before the policy change suggests this regulation was not playing such role in this setting, at least for that period.

24. The period we utilize for this exercise covers the second half of November and the first half of December of both 2013 and 2015. The pattern we find remains the same when focusing on longer time periods.

there been perfect competition, banks would have not offered exposed loans after the policy was in place, as those loans would be unprofitable. However, this interpretation is not conclusive, as the pool of applicants might have also changed between the two periods we analyze in response to the policy change. Overall, these patterns suggest that banks had market power, which allowed them to charge interest rates above expected costs.

Exposure also varies across borrower risk. Figure 1.3 displays exposure by borrower risk within each policy size bracket. As much as half and a third of high-risk borrowers signing loan contracts for loans of \$0-\$2,000 and \$2,000-\$8,000 were exposed to the policy, compared to only 26% and 11% for low-risk borrowers. These patterns suggest that exposure to interest rate regulation was increasing in borrower risk, which is as expected in the presence of risk pricing.

This evidence suggests that as interest rate caps were strengthened, the distribution of interest rates responded by bunching below the interest rate cap, and that riskier borrowers were more affected by the policy. This is not surprising and simply implies that the regulation was enforced. The magnitude of the effects is large and understanding its implications for other outcomes is important. We address these aspects in the rest of the paper.

1.3.2 Effects on Market Outcomes

The policy change we study provides two useful sources of variation to estimate the effects of interest rate regulation. First, it provides variation across time. Before December 2013, regulation was not binding for loans in \$0-\$8,000, but it became increasingly binding as the reform was phased in. Second, it provides variation across loan size. Regulation became more binding for loans of \$0-\$2,000 than for loans of \$2,000-\$8,000, and for loans of \$2,000-\$8,000 than for those of \$8,000-\$20,000, which remained essentially untreated. We exploit these two sources of variation. For our analysis, we aggregate the data to measure market-level effects. In particular, we construct bins for loan size and term indexed by k, and aggregate the data at that level.[25]

25. Concretely, we define loan size bins in intervals of 50 UF (\$2,000) and employ a clustering algorithm to classify loan term in 8 bins, which adds up to 80 loan-type bins, indexed by k. We then compute averages

Evolution of Policy Effects

We start by studying the evolution of outcomes of interest around the policy change. We estimate differences-in-differences models that decompose effects though time. The objective is to provide a first approximation of the effects of the policy change, while also addressing concerns related to trends in the outcomes of interest leading to the policy change that could be correlated with the policy itself. We start by estimating the equation:

$$y_{krt} = \sum_{\tau} D_k \beta_{r\tau} + \alpha_{kr} + \delta_{rt} + \varepsilon_{krt} \tag{1.2}$$

where y_{krt} is the outcome of interest for product bin k and risk group r in month t; D_k indicates whether loans in k are smaller than \$8,000 and thus affected by the policy change; α_{kr} are fixed effects that control for unobservable shocks specific to a loan size, term and risk group, but are constant through time; and δ_{rt} are fixed effects that control for unobservable shocks specific to a month and risk group but are constant across loan size and term. The coefficients of interest are $\beta_{r\tau}$, which measure the difference in the outcome of interest between loans affected by the policy change and the comparison group for borrowers of risk r, τ months after the policy change.[26]

Figure 1.4 displays results from equation (1.2) for relevant outcomes, separately for low- and high-risk borrowers. Figure 1.4-a shows that average interest rate in the market decreased after the policy change. Figure 1.4-b shows that the effect is concentrated on the upper part of the distribution of interest rates, as the effect on the 90th percentile of interest rates is stronger than that on the average, and becomes apparent earlier after the policy change than that on the average. Moreover, Figure 1.4-c shows that the number of loan applications by high-risk borrowers decreases, whereas Figure 1.4-d shows that the number of loans in the market also decreased, and substantially more so than applications. Figure 1.4-e shows that the average risk score in the market decreased after the policy

or aggregate levels of the outcomes of interest for each bin and month. To study heterogeneous effects, we implement the same procedure but separately for low- and high-risk borrowers.

26. We control for seasonal patterns specific to loan size for quantity outcomes by removing month-of-the-year fixed effects from the time series of each product type bin k, before estimating equation (1.2).

change, such that the borrower pool became safer. Finally, Figure 1.4-f shows that a measure of expected mark-up also decreases after the policy change, although less than interest rates, given the decrease in default risk.[27]

These results share two important patterns across all outcomes. First, estimates display flat trends leading to the policy change and a steady decrease in interest rates after it, which suggests that loans of $8,000-$20,000 evolve similarly to loans that were directly treated, reinforcing the extent to which the former serves as a comparison group for the latter in this analysis. We further exploit that in a more extensive regression analysis in the next section. Second, estimated effects are larger for high-risk borrowers than for low-risk borrowers, which suggests that the former were more affected, consistent with their higher exposure discussed above.

This set of results readily suggests that both prices and quantities decreased under stronger interest rate regulation, which is consistent with the policy change having effects both in terms of consumer protection and credit access.

Regression Analysis

In this section, we exploit more granular variation in interest rate caps to estimate its effects on market outcomes. We define the following treatment intensity variable to exploit time variation in regulation within each size bracket, and to ease the interpretation of the results:

$$\Delta^{\bar{\iota}}_{\ell,t} \equiv (\bar{\iota}_{\ell,0} - \bar{\iota}_{\ell,t}) - (\bar{\iota}_{>8000,0} - \bar{\iota}_{>8000,t}) \tag{1.3}$$

for each of the treated size brackets $\ell \in$ {$0-$2,000, $2,000-$8,000}. The first term in equation (1.3) is the change in the interest rate cap for loan-size bracket l between current month t and baseline month $t = 0$ at December 2013. The second term in equation (1.3) is the change in the interest rate cap for the comparison group, i.e. loans larger than $8,000.

27. We compute expected mark-up as $m_{krt} = \frac{1}{N_{krt}} \sum_{i \in \mathcal{I}_{krt}} [\iota_i(1 - d_i) - f_t]$, where m_{krt} is the average mark-up for loans in bin k for borrowers of risk r; ι_i is the interest rate charged to borrower i; d_i is the predicted default probability of such borrower; f_t is the funding rate faced by banks; and $\mathcal{I}_{krt}$ is the set of borrowers of risk r taking loans k in month t. This is a proxy of average mark-up in the market that does not account for other components of cost than risk and funding. We develop more comprehensive measures of mark-ups using our model in the second part of the paper.

Subtracting the second term removes variation in economic conditions that influences interest rate caps, and thus isolates the policy variation that we exploit. Figure A.6 displays the evolution of these treatment intensity variables.

Using these variables, we estimate the following specification:

$$y_{krt} = \sum_{\ell} \beta_{\ell(k),r} \Delta^{\bar{\iota}}_{\ell(k),t} + \delta_{kr} + \phi_{krm(t)} + \gamma_{rt} + \varepsilon_{krt} \tag{1.4}$$

where y_{krt} is the outcome of interest for product bin k for borrower of risk r for month t; δ_{kr} is a set of fixed effects that controls for unobservable shocks specific to a loan size and term and borrower risk bin, but constant through time; $\phi_{krm(t)}$ is a set of fixed effects that controls for unobservable shocks specific to a product type, borrower risk bin and month-of-the-year $m(t)$; and similarly γ_{rt} is a set of fixed effects that controls for unobservable shocks specific to a borrower risk bin and month but constant across loan size and term. The coefficients of interest are $\beta_{0-2000,r}$ and $\beta_{2000-8000,r}$. Given how the treatment variable $\Delta^{\bar{\iota}}_{\ell,t}$ is constructed, these coefficients measure the effect of *reducing* interest caps by 1 p.p. on the outcome of interest for each policy size bracket respectively. We then compute full effects by scaling up these estimates by the full change in interest caps. All regressions are weighted by the number of loans in each product bin before the policy was implemented. Finally, standard errors are clustered at the product bin level to allow for potential correlation in errors within bins across time.

We study three sets of outcomes. First, we estimate effects on interest rates, focusing on maximum and average rates. Second, we focus on quantity by estimating effects on the number of applications, number of loans and credit volume. Third, we focus on risk selection, loan performance and expected profitability by estimating effects on borrower risk scores and income, on 90-day loan default in the first year, and on expected mark-ups. In each case, we estimate regressions both across all borrowers and separately for low- and high-risk borrowers.

Effects on Interest Rates. Stronger regulation reduced interest rates, consistent with evidence in Section 1.3.1. Table 1.3 displays estimates of equation (1.4) for maximum and

average interest rates. We find pass-through of interest rate caps to maximum interest rates was high. Effects from a 1 p.p decrease in interest rate caps range from 0.96 p.p for low-risk borrowers to 1 p.p for high-risk borrowers for loans of $0-$2,000; and from 0.66 p.p for low-risk borrowers to 0.8 p.p for high-risk borrowers for loans of $2,000-$8,000. Full effects are large and close to the total change in the interest rate cap, particularly for riskier borrowers. These results verify that the policy was enforced, and that it was more binding for smaller loans and riskier borrowers.

Average interest rates decreased as a result of stronger regulation, as displayed in Table 1.3-B. We estimate that reducing interest rate caps by 1 p.p decreases average interest rates by 0.23 p.p and 0.07 for loans of $0-$2,000 and $2,000-$8,000, respectively. These effects are heterogeneous across borrower risk. The effects on low-risk borrowers are smaller at 0.13 p.p and 0.03 p.p, while those on high-risk borrowers are much larger at 0.26 p.p and 0.11 p.p respectively. The full effects on average interest rates were 3.8 p.p and 1.7 p.p for loans of $0-$2,000 and $2,000-$8,000.[28]

Effects on Quantity Outcomes. Interest rate regulation may affect borrower application behavior. On the one hand, it weakly reduces interest rates upon approval and thus induces marginal borrowers to take loans. On the other hand, banks may be less willing to approve applications if they are constrained in terms of pricing, which may deter borrower applications if applying is costly. The latter should be more relevant for observably riskier borrowers. Table 1.4-A displays estimates of equation (1.4) for the number of applications. We find no statistically significant effects on average, nor for low-risk borrowers. However, we find suggestive evidence that risky borrowers apply less often for loans under stronger regulation. In particular, a 1 p.p decrease in interest rate caps reduced applications by 1% and 0.4% for loans of $0-$2,000 and $2,000-$8,000, although the latter is not statistically significant. These estimates imply that the full policy decreased applications by risky

28. Note that these estimates measure the effect on the average interest rate, regardless of whether the loans were exposed to the policy. The effect on loans not exposed to the policy should arguably be close to zero, which suggests that effects on exposed loans should be larger. In absence of quantity effects, we would expect a perfect pass-through of changes in interest rate caps to the average interest rate of exposed loans, In that case, the ratio between our estimates and shares of exposed loans per group in Figure 1.3 would be equal to one. However, such calculation yields around 0.55 p.p and 0.32 p.p respectively for loans of $0-$2,000 and $2,000-$8,000, which readily suggests the policy had quantity effects.

borrowers by 15% and 9% for loans in each size bracket.

How did this change in interest rate regulation affect equilibrium quantities? Tables 1.4-B and 1.4-C display estimates of equation (1.4) for number of loans and credit volume. We find that reducing interest rate caps by 1 p.p reduced the number of loans by 2% and 0.5% respectively for loans of $0-$2,000 and $2,000-$8,000. Again, we find substantial heterogeneity across borrower risk. For low-risk borrowers, we estimate decreases of 0.8% and 0.2% for loans of $0-$2,000 and $2,000-$8,000 respectively; whereas for high-risk borrowers, estimates are almost three times larger, at 2.5% and 0.7% respectively. Results are similar quantitatively for credit volume. Full effects of the policy change are large. We estimate that the number of loans decreased by 27.6% and 11.9% for loans of $0-$2,000 and $2,000-$8,000 respectively. Effects are particularly large for high-risk borrowers, at 33.9% and 15.8% respectively. The fact that the effects on the number of loans and credit volume are substantially larger than those on applications suggests that a large share of the estimated reduction in quantity comes from rejections.

Effects on Risk Selection, Loan Performance and Profitability. How do changes in applications and approvals affect the borrower pool? Table 1.5-A displays results from estimating equation (1.4) for ex-ante borrower risk measures. The policy change improved the borrower risk pool. A reduction of 1 p.p in the interest rate cap decreases average borrower predicted default rate by between 0.07 p.p and 0.04 p.p for loans of $0-$2,000 and by around 0.02 for loans of $2,000-$8,000, depending on the measure of predicted risk. The full policy decreased borrower predicted default risk by between 1.14 p.p and 0.7 p.p for loans of $0-$2,000, and by between 0.49 and 0.35 p.p for loans of $2,000-$8,000. Relatedly, we find that average borrower income increases with stronger regulation.

We now turn to estimate effects on loan performance. Effects on interest rates and screening could affect loan performance. On the one hand, lower interest rates may increase loan repayment by reducing moral hazard (Holmstrom and Tirole, 1997; Adams et al., 2009). On the other hand, a better borrower pool—due to stronger risk selection—may also lead to improvements in loan performance. Results in Table 1.5-B show that loan performance did in fact improve as a result of the policy. Reducing interest rate caps by

1 p.p decreased the share of loans under 90-day default in their first year by 0.09 p.p and 0.04 p.p respectively for loans of $0-$2,000 and $2,000-$8,000. This effect is higher among high-risk borrowers than among low-risk borrowers. The full policy was able to reduce the average share of loans under 90-day default in their first year by 1.52 p.p and 0.88 p.p, equivalent to 22.5% and 14.6% of their baseline levels.

Finally, we study effects on banks' expected mark-ups on signed contracts. Effects on this outcome will combine effects on interest rates with effects on the composition of the borrower risk pool. We find that expected mark-ups decrease as a result of stronger interest rate regulation, as displayed in Table 1.5-C. However, these effects are smaller than those on interest rates, which is driven by the fact the composition of the borrower pool is safer and, therefore, banks' expected costs decrease on average. Overall, these results suggest that interest rate regulation indeed constrains the exercise of market power by banks.

Robustness Exercises

The analysis we develop exploits policy variation across loan size and time to estimate the effects of interest rate regulation on market outcomes. The main concern regarding our empirical strategy is that the policy change affected relative regulation across loan size, which might induce substitution across loan size brackets. In principle, given regulation becomes stronger for loans of $0-$8,000, we might expect consumers to substitute towards that group, which would imply our quantity effects are attenuated. However, baseline variation in interest rate caps limits such incentives, as the interest rate for loans larger than of $8,000 is lower than that for loans of $0-$8,000 throughout the period of study, as shown in Figure 1.1. On the other hand, banks may attempt to offer multiple loans of smaller sizes below $8,000 rather than a single one of size larger than $8,000 in order to charge higher interest rates. However, we find no evidence of such behavior in the data, as more than 98% of borrowers take only one loan in months in which they borrow, and that share remains unchanged throughout the period of study, as displayed by Figure A.7.

We develop a number of robustness exercises to assess the assumptions underlying this strategy. We provide a summary of the main results from these exercises in this section,

and leave an extended discussion for Appendix A.1. We already showed in Section 1.3.2 that trends leading to the policy change are similar across groups. In a similar vein, we study whether placebo policies shifted along loan-size space relative to the actual policy change could generate effects similar to our estimates. Finding evidence of effects from placebo effects would be suggestive of substitution concerns. Appendix A.1.1 shows that effects from such placebo policies are generally smaller and close to zero. Second, we study whether the distribution of loan size and term changes around the policy change for loans larger than $8,000, and Appendix A.1.2 shows we find no evidence of such pattern. Third, we study whether the distribution of loan application loan amount changes around the policy thresholds, which could reflect substitution across size brackets by borrowers. Appendix A.1.3 shows there are no such patterns in the data. Fourth, we study whether alternative definitions of the comparison group affect our results, and show in Appendix A.1.4 the latter remain similar across a range of comparison groups. Overall, this evidence suggests that our main results are robust to concerns about substitution across loan size brackets. Finally, we study heterogeneity in estimated effects across banks to verify whether our results are driven by any particular bank. In Appendix A.1.5, we show that effects for most banks display the same patterns of our results.

1.3.3 Discussion

In this section, we provided evidence for equilibrium effects of interest rate regulation on market outcomes. We find that the policy change we study had strong effects. Average interest rates decreased across the market, while the number of loan contracts also decreased substantially. Effects are particularly large for risky borrowers, who were more exposed to the policy change, as they were charged higher interest rates before the policy change due to risk pricing. These results are consistent with recent research that also finds quantity effects from this regulation such as Benmelech and Moskowitz (2010). Additionally, we find improvements in the borrower pool risk and loan performance, which is in contrast to Rigbi (2013), who finds no effect on loan performance.

Overall, our estimates imply that 151,027 loan contracts per year were deterred by

stronger interest rate regulation, equivalent to 19% of the number of loans signed during the year before the policy change and $361.6 million in consumer loans.[29] Our estimates of price effects imply that interest rates decrease on average by 9%, which translates into an average decrease in monthly payments across loans of $3.26. The present value of reduced monthly payments during the year before the policy change is $31.7 million.[30] These results provide a picture of the magnitude of aggregate effects, but do not allow to assess welfare effects.

While this analysis is informative of the effect of interest rate regulation on equilibrium outcomes, several questions remain unanswered. First, the welfare implications of the combination of policy effects we estimate are unclear. In order to develop a welfare analysis, we require knowledge about consumers' willingness to pay and banks' costs. Second, as we emphasized at the beginning of the paper, market power may have a role in determining the effects. However, it is hard to assess this argument using observational data given the endogeneity of market structure. Third, we also emphasized that interest rate regulation is remarkably unsophisticated in most markets. Nevertheless, this analysis does not allow us to draw conclusions on how alternative designs would affect market outcomes. We develop and estimate an equilibrium model for the consumer credit market in the next sections to address these aspects.

1.4 An Equilibrium Model of Applications, Pricing and Repayment

We develop and estimate an equilibrium model of applications, pricing and repayment in the consumer credit market. The ultimate goal is to estimate the model. Several modeling choices aim at moving from a theoretical model to an empirical one that can be estimated

29. This aggregate effect is obtained by calculating the share of the credit volume originated during the year before the policy change that would be deterred by the policy for each treated policy size bracket according to estimates across risk bins in columns (1) and (4) of Table 1.4. We report the total across both policy loan-size brackets.

30. This amount is calculated by computing counterfactual monthly payments using an interest rate adjusted downwards by average price effects in column (4) of Table 1.3. Then, we compute the difference between those monthly payments and actual monthly payments. We compute the present value of that difference using a discount rate of 5% and the term of each loan contract. Finally, we aggregate across loan contracts actually signed during the year before the policy change was implemented.

using the data available for our setting. We discuss these choices after developing the model, in Section 2.6.6.

1.4.1 Model

Setup

There are N consumers, denoted by i. There are J banks, denoted by $j \in \mathcal{J}$, where $\mathcal{J}$ is the set of banks in the market. The model is static, and we focus on the choice consumers face in a given month. Consumers choose whether to apply for loans of a given amount and term (L_i, T_i), determined in a previous stage that we do not model. Conditional on (L_i, T_i), contracts are homogeneous and only differ by their monthly payments, which vary across banks due to cost heterogeneity.[31] Therefore, consumers shop across banks for the lowest monthly payment. The price and bank signing a contract with a consumer are determined as the outcome of an English auction, as in Allen et al. (2018). The structure and timing of the model are summarized in Figure A.17.

Borrowers. Consumers are endowed with observable characteristics x_i and unobservable characteristics ε_i, such that (x_i, ε_i) summarize consumer type. The vector x_i collects all publicly available information in the market, including risk scores, borrower income, and borrower credit history, among others, whereas $\varepsilon_i = (\varepsilon_{Ai}, \varepsilon_{Si})$ are potentially correlated application and repayment shocks that follow a joint distribution F_ε and are private information of borrowers. Let ε_{Ai} be realized at the application stage, and ε_{Si} be realized at the repayment stage.

Borrowers decide whether to shop for loans. If they shop for loans, they incur an application cost $\kappa(z_i)$ that depends on cost shifters z_i, and shop across banks. If they do not to shop for loans, they obtain their outside option. Let the indirect utility from a contract

31. We write the model in terms of monthly payments for convenience, as it simplifies the derivation of optimal pricing by banks.

and the outside option be:

$$\begin{aligned} u_{Ci} &= v_C(x_i, L_i, T_i) - p_i \\ u_{Oi} &= v_{Oi} \end{aligned}$$

where $v_C(x_i, L_i, T_i)$ is the indirect utility of a contract, which depends on borrower and loan attributes; p_i is the monthly payment offered to borrower i; and v_{Oi} is the indirect utility of the outside option. Borrowers choose to apply for loans by comparing the expected value of both options, given by:

$$\begin{aligned} u_{Ai} &= P_{Ci} \underbrace{\int u_{Ci} f_{p|C}(p) dp}_{\text{Value of approval}} + (1 - P_{Ci}) \underbrace{u_{Oi}}_{\text{Value of rejection}} - \kappa(z_i) + \varepsilon_{Ai} \\ u_{NAi} &= u_{Oi} \end{aligned}$$

where P_{Ci} is the probability that the application is approved by some bank in the market, and where the borrower integrates the value of a loan contract over the density of loan prices they face conditional on approval, we denote by $f_{p|C}(p)$. Both P_{Ci} and $f_{p|C}(p)$ are equilibrium objects which borrowers know. Finally, ε_{Ai} is a shock to the utility that borrowers obtain from applying for a loan relative to not applying.

Given this structure, a borrower decides to apply for a loan whenever its expected utility is higher than that of remaining out of the credit market. The application probability is:

$$P_{Ai} = \Pr\left(P_{Ci} \int (u_{Ci} - u_{Oi}) f_{p|C}(p) dp - \kappa(z_i) + \varepsilon_{Ai} \geq 0 \right) \tag{1.5}$$

from where it is clear that application decisions are driven by: (i) the approval probability, (ii) the expected gains from a loan contract relative to the outside option, (iii) the density of loan prices conditional on approval, (iv) an application cost that borrowers face, and (v) a shock to the utility of application. Let a_i indicate that borrower i applies for a loan and define $\mathcal{A}$ as the set of loan applicants. We set the utility of the outside option to $u_{Oi} = 0$ for the remainder of the paper, such that u_{Ci} is the utility of a loan contract relative to the borrower's outside option.

Conditional on applying for loans, the borrower solves a discrete choice problem to choose which bank to sign a loan contract with, which implies that utility from a loan contract is:

$$u_{Ci} = \max_{j \in \mathcal{J}} \quad v_C(x_i, L_i, T_i) - p_{ij} \iff p_i = \min_{j \in \mathcal{J}} p_{ij}$$

such that bank choice is driven solely by monthly payment, given there is no differentiation across banks in terms of the utility they provide to borrowers. As we further detail below, all differentiation is concentrated in banks' costs.

Loan Repayment. After signing a loan contract, repayment is realized. Let $s_i \in [0, 1]$ measure the share of payments made by borrower i relative to the total number of monthly payments in the contract:

$$s_i = s(x_i, L_i, T_i, \varepsilon_{Si}) \tag{1.6}$$

which is a function of borrower characteristics and non-price contract terms. Moreover, repayment is increasing in the repayment shock ε_{Si}. There is adverse (advantageous) selection in the model if application and repayment display a negative (positive) correlation through unobservables to banks, i.e. through ε_{Ai} and ε_{Si}.

Banks. We model competition among banks to attract borrowers as an English auction.[32] Banks are heterogeneous in the cost of serving borrowers. There are three components of cost: (i) funding cost f_i; (ii) bank-borrower match-value ω_{ij}, which is an i.i.d. shock from a distribution G_ω that is unobserved to borrowers and may make it less costly for a bank to serve some borrowers than others; and (iii) repayment risk. We combine the first two components in $m_{ij} = f_i - \omega_{ij}$. In terms of repayment risk, banks observe x_i and application choices a_i, which they employ to estimate repayment risk when pricing contracts.

Bank profits are given by the difference between a stream of repayments with repayment risk and a stream of monthly bank costs. Let $\varphi(T_i) \equiv \frac{1}{r}(1 - \exp(-rT_i))$ be a present value operator that discounts a stream of payments for T_i months at a discount rate r, and

32. Modeling the interaction between consumers and banks as an English auction provides a reasonable characterization of the market, and is convenient for empirical work. We discuss this choice in detail in Section 2.6.6.

$\varphi(S_i) \equiv \frac{1}{r}(1 - \exp(-rS_i))$ be a present value operator that discounts a stream of payments for $S_i = s_i T_i$ months, where S_i is repayment length by borrower i. The expected profit from a given loan contract at price p_{ij} is:

$$E_\varepsilon[\pi_{ij}] = E_\varepsilon[\varphi(S_i)]p_{ij} - \varphi(T_i)(f_i - \omega_{ij})$$

where repayment risk and funding cost depend only on borrower-specific attributes, while match-value ω_{ij} depends on bank-borrower attributes. Therefore, the role of ω_{ij} is to introduce cost heterogeneity across banks and can be thought of as a term measuring the match-value of a potential contract. For instance, ω_{ij} could capture bank-borrower relationships and bank convenience in local markets, among other features. Conditional on x_i, banks with higher ω_{ij} face a lower cost of signing a loan contract with borrower i and can therefore offer such contract at a lower price.

A bank offers a contract to a borrower if $E_\varepsilon[\pi_{ij}] \geq 0$, and otherwise rejects the borrower. Expected profits are decreasing in borrower repayment risk at a given price, and thus observably riskier applicants are less likely to be approved. Borrowers' application choices and banks' approval decisions are related. Given banks observe x_i and know F_ε, they make inference about borrower unobservable repayment shock ε_{Si} from application choices. Banks incorporate that information in their approval decision when computing conditional expected repayment.[33]

Interest Rate Regulation. We introduce interest rate regulation in the form of an interest rate cap, which induces caps on monthly payments. In particular, banks are not allowed to charge monthly payments higher than $\bar{p}_i$.

Equilibrium

Equilibrium in this model is characterized by the pool of applicants, and loan approvals and prices. In absence of interest rate regulation, the outcome of an English auction in this

33. In particular, banks compute $E_\varepsilon[\pi_{ij}] = E_\varepsilon[\pi_{ij}|a_i = 1, x_i]$. This implies that, conditional on x_i, application choices reveal information about ε_{Ai}. Given banks know F_ε, a signal about ε_{Ai} is informative about repayment risk ε_{Si}.

setting is that the lowest cost bank wins the auction with a bid $b_{i(1)}$ such that the second lowest cost bank is indifferent between getting the loan contract or not at that price.[34] The solution to:

$$E_\varepsilon[\pi_{i(2)}] = E_\varepsilon[\varphi(S_i)]b_{i(1)} - \varphi(T_i)m_{i(2)} = 0$$

is thus the equilibrium unconstrained price:

$$p_i^u = \frac{\varphi(T_i)}{E_\varepsilon[\varphi(S_i)]}(f_i - \omega_{i(2)}) \tag{1.7}$$

which is increasing in repayment risk and funding cost, and decreasing in match-value of the closest competitor.[35] This price yields equilibrium expected profits $E_\varepsilon[\pi_{i(1)}] = \varphi(T_i)(m_{i(2)} - m_{i(1)}) = \varphi(T_i)(\omega_{i(1)} - \omega_{i(2)})$, from where it becomes clear that the source of banks' market power in this model is given by cost advantages.

Under interest rate regulation, there are three potential outcomes for an applicant. If not binding, then the bank offers the contract at the unconstrained price in equation (1.7). If regulation is binding, however, the unconstrained price is higher than the price cap, $\bar{p} < p_i^u$.[36] In this case, the lowest cost bank offers the contract at price $p_i = \bar{p}$ as long as $E_\varepsilon[\pi_{i(1)}] = \bar{p} - \frac{\varphi(T_i)}{E_\varepsilon[\varphi(S_i)]}m_{i(1)} \geq 0$. Finally, if the cost of the lowest cost bank is high enough as to make lending at the cap unprofitable, $E_\varepsilon[\pi_{i(1)}] = \bar{p} - \frac{\varphi(T_i)}{E_\varepsilon[\varphi(S_i)]}m_{i(1)} < 0$, all banks reject

34. As usual in the treatment of auction models, the notation $x_{(m)}$ indicates the mth order statistic of x.

35. This expression of the unconstrained equilibrium price can be rewritten as:

$$p_i^u = \underbrace{\frac{\varphi(T_i)}{E_\varepsilon[\varphi(S_i)]}}_{\text{Risk adjustment}} \underbrace{(f_i - \omega_{i(1)})}_{\text{Mg. Cost}} + \underbrace{\omega_{i(1)} - \omega_{i(2)})}_{\text{Mark-up}} \tag{1.8}$$

where it is clear that unconstrained loan prices are comprised by risk-adjusted cost and a mark-up determined by the cost advantage of the bank signing the contract relative to its closest competitor.

36. Note that for interest rate regulation to be binding, it must be the case that only one bank $j \in \mathcal{J}$ has a cost below the price cap. Otherwise, competition by other banks would drive price below the cap, making the latter non-binding.

the borrower. Therefore, equilibrium prices under interest rate regulation are:

$$p_i^* = \begin{cases} p_i^u & \text{if } p_i^u \leq \bar{p} \\ \bar{p} & \text{if } \frac{\varphi(T_i)}{E_\varepsilon[\varphi(S_i)]} m_{i(1)} \leq \bar{p} < p_i^u \\ \cdot & \text{if } \bar{p} < \frac{\varphi(T_i)}{E_\varepsilon[\varphi(S_i)]} m_{i(1)} \end{cases} \tag{1.9}$$

The distribution of equilibrium prices determines application decisions by borrowers, which in turn determines the equilibrium set of applicants, $\mathcal{A}^*$. In this equilibrium, (i) borrowers optimally make application choices given both the application approval probability and the distribution of prices they face in the market, and their application costs, while (ii) banks optimally make price offers in a competitive environment given both their costs and the pool of loan applicants.

Effects of Interest Rate Regulation

Application Behavior. What are the implications on the demand side? Stronger regulation affects borrower application behavior by (i) reducing the approval probability of an application, and by (ii) weakly reducing prices conditional on approval. These incentives jointly determine the effect of interest rate regulation on borrower application behavior:

$$\frac{du_{Ai}}{d\bar{p}_i} = \underbrace{\frac{\partial P_{Ci}}{\partial \bar{p}_i} \int u_{Ci} f_{p|C}(p) dp}_{\text{Credit access } (\geq 0)} + \underbrace{P_{Ci} \frac{\partial}{\partial \bar{p}} \int u_{Ci} f_{p|C}(p) dp}_{\text{Consumer protection } (\leq 0)} \tag{1.10}$$

which is ambiguous and depends on which incentive dominates. If the approval probability decreases sharply in response to stronger regulation but expected prices conditional on approval do not respond as strongly, then borrowers will likely apply for loans less often. In the opposite case, if the effects on approval probability are small relative to those on expected prices, borrowers will likely apply for loans more often. Finally, if regulation is not binding, then it should not affect the approval probability nor expected prices, and therefore should not affect application behavior.

Banks' Lending. We consider how interest rate regulation affects pricing and approval incentives for banks. The effect of stronger interest rate regulation on banks' expected profits depend on whether it is binding. For loan applicants who were already in the market, profits decrease under stronger interest rate regulation whenever it is binding, and are unaffected whenever it is not binding. There are two possible scenarios for the former set of applicants, as banks may either: (i) choose to sign those contracts as long as they yield non-negative profits; or instead (ii) choose to reject them if they yield negative profits at the lower interest rate cap. Given borrower expected profitability is decreasing in observable risk at a given price, the probability that a bank decides to reject an application under stronger interest rate regulation is increasing in observable risk.

Heterogeneity across Consumers. Borrowers can be classified in four sets according to the effects of interest rate regulation. First, consumers who remain in the market under stronger regulation and are offered contracts at a lower price are *protected* and increase their consumer surplus. That is, the policy is a transfer from banks to borrowers in the amount of the change in the interest rate cap. Second, consumers who become *excluded* from the market either by being discouraged from applying for loans or by having their applications rejected under stronger regulation. Third, consumers who enter the market because of stronger regulation are *included*. These are consumers that experience an improvement in their expected loan prices due to stronger interest rate regulation without a strong enough decrease in their approval probability, such that regulation induces them to enter the market and apply for loans. Finally, consumers for whom stronger regulation does not change their approval probability nor their expected loan prices are *unaffected*.

Welfare Effects. The effects of stronger regulation on expected consumer surplus are ambiguous and determined by the same forces as the effects on application behavior in equation (1.10). The effect on expected consumer surplus will have the same sign as that on application behavior. From an ex-post perspective, effects combine increases in consumer surplus for protected and included borrowers, with decreases in consumer surplus for

excluded borrowers, and decreases in bank profits. The overall effect is ambiguous.[37]

Market power and selection are relevant for welfare effects. First, note that in a setting without market power, there would be no borrowers that are protected by the policy, as all marginal borrowers would become unprofitable for banks under stronger interest rate regulation. Second, if there is selection into the market on observable risk and the willingness to pay for loans is correlated with risk, then the direction of selection will matter for welfare implications, given (observably) riskier borrowers are more likely to be excluded.

Loan Performance. If stronger interest rate regulation improves the borrower pool risk through rejecting marginally (observably) riskier borrowers, then the aggregate default rate in the market decreases under stronger regulation. In this model, where prices do not directly affect repayment, the effect of interest rate regulation on aggregate loan performance is thus purely compositional.

1.4.2 Model Discussion

The model provides a framework to study the effects of regulation in consumer credit markets. It accommodates a variety of the features common to these markets that we documented in Section 3.2.3, such as price dispersion, risk pricing, the role of previous relationships for approvals and pricing, among others. However, it also has limitations that we discuss.

Static Demand. We model potential borrowers' application choices as a static problem. Theoretical models of demand for credit often involve intertemporal optimization problems where the trajectory of interest rates determines the optimal trajectory of borrowing and saving. However, that class of model only yields closed form solution in restricted cases, which often fail to accommodate heterogeneity in loan contracts (Attanasio et al., 2008).

37. Previous research on the welfare effects of price caps predicts mostly adverse effects on consumer surplus for perfectly competitive markets (e.g., Glaeser and Luttmer 2003; Bulow and Klemperer 2012). In contrast, our model predicts ambiguous effects on consumer surplus, because we study an imperfectly competitive market.

We instead focus on the static problem where a borrower chooses whether to finance credit needs by applying for loans or not. Previous empirical research of loan demand also adopts this static approach (e.g., Alessie et al. 2005; Attanasio et al. 2008; Einav et al. 2012). This assumption might not be appropriate for large loans such as mortgages, for which consumers often shop over long periods of time and for which evidence shows that consumers react dynamically to market conditions (Mian and Sufi, 2009). However, it is likely appropriate for markets for smaller loans, such as consumer loans in our setting. In fact, evidence from our survey suggests that borrowers spend a median of only 7 days searching for consumer loans, as displayed in Figure A.16-b. Moreover, as much as 66% of the respondents say that they search credit "quickly" in response to financing needs. These patterns suggest that focusing on static choices is meaningful in our context.

Exogenous Loan Amount and Term. We assume that loan amount and term are determined in a previous stage not in the model, which is in line with modeling loan demand as a response to shocks, but imposes a strong constraint on consumer behavior. This assumption allows for a convenient application equation that becomes a binary choice. Moreover, the fact that we analyze the implications of interest rate regulation and we find no effects of the policy change on the distribution of loan size in our analysis in Appendix A.1.2, suggests that not modeling this substitution dimension might be a reasonable assumption for our purpose and setting. Finally, the extent to which loan size and term signal borrower cost should be captured by including (L_i, T_i) in our repayment equation.

Bank Competition as English Auction. We model equilibrium interest rates as the result of an English auction, where banks compete for borrowers who bargain with banks for lower interest rates. The appeal of this approach is that it provides a tractable model that accommodates price dispersion and imperfect competition. Moreover, it avoids the need to specify the prices of all alternatives in consumers' choice sets, which are unobserved to us.[38] This approach has been recently used for modeling markets with bargained

38. An alternative approach previously adopted to study choice in credit markets is to model the game between borrowers and banks as a Bertrand-Nash game with posted prices and to predict the prices that competing banks would offer to each borrower using information from signed contracts (e.g., Crawford et al.

prices (Salz, 2017; Allen et al., 2018), and is isomorphic to modeling the market as a standard Bertrand game where firms produce homogeneous goods with heterogeneous costs (Beckert et al., 2018). Under this framework, the source of bank market power in our model is cost heterogeneity, which translates into interest rates being set at a mark-up over expected costs, similar to the interpretation of market power in Petersen and Rajan (1995). Additionally, survey evidence shows that 89% of borrowers considered more than two banks when shopping for loans in their last search for loans, and the median borrower considered three banks as shown by Figure A.16-c. This evidence suggests that borrowers indeed interact with several banks in their shopping process.[39]

Search Frictions. We do not model other sources of market power such as search frictions, which have been the focus of recent research on credit markets (Woodward and Hall, 2012; Agarwal et al., 2018; Allen et al., 2018; Galenianos and Gavazza, 2018). A first implication of this assumption is that we disregard potential effects that interest rate regulation may have on search effort. As suggested by Fershtman and Fishman (1994) and Armstrong et al. (2009), price caps reduce price dispersion and thus in turn search effort, which may lead to unintended effects such as increases in equilibrium prices. Our model does not account for such channel. A second implication is that our estimates of the model might understate the amount of bank market power.

Moral Hazard. Loan price p_{ij} does not enter into the repayment equation, which implies the model rules out moral hazard in the form suggested by Holmstrom and Tirole (1997). We depart in this aspect from recent work on credit markets, such as Adams et al. (2009). While restrictive, this assumption substantially simplifies the analysis of bank pricing. Moreover, recent experimental evidence in Castellanos et al. (2018) suggests moral hazard might not be a first order concern in consumer credit markets.

2018). Modeling the game between borrowers and banks as an English auction avoids that prediction step.

39. The fact that the sample selection for our survey requires consumers to have applied for loans before suggests that our survey data might be representative of a set of consumers with a relatively more intense search behavior than the average consumer. However, note that these statistics are for a given application event.

1.5 Econometric Model

The model is summarized by equations (1.5), (1.6), and (1.9) for applications, repayment, and pricing. The structural objects of interest on the demand side are the parameters in the indirect utility function of consumers, $u_{Ci}(x_i, L_i, T_i, p_i)$; the parameters in the application cost, $\kappa(z_i)$; the parameters in the repayment equation, $s(x_i, L_i, T_i, \varepsilon_{Si})$; and the joint distribution of application and repayment shocks, F_ε. On the supply side, we are interested in the distribution of banks' costs, G_ω.

We estimate the model using the following observables available in our data. First, we observe borrower covariates x_i, application shifters z_i, funding cost f_i, relationships with banks r_{ij}, and application choices a_i for all borrowers. Second, we observe loan amount and term for each applicant, (L_i, T_i). Finally, we observe loan monthly payment and repayment for each approved applicant, (p_i, s_i). In this section, we specify the model and state relevant statistical assumptions, and then develop an identification discussion before moving towards estimation.

1.5.1 Model Specification

Application and Repayment. We specify the net indirect utility of a contract as a linear function of borrower attributes x_i, loan amount, term, and prices; and the application cost as a linear function of shifters z_i. In particular, we specify the application probability in equation (1.5) as:

$$P_{Ai} = \Pr\left(P_{Ci} \int (x_i'\delta_X + \delta_L L_i + \delta_T T_i - \delta_p p) f_{p|C}(p) dp - z_i'\kappa + \varepsilon_{Ai} \geq 0\right) \tag{1.11}$$

where x_i is a vector of borrower covariates that includes the borrower risk score, income, debt to income ratio, default to debt ratio, gender, and age along with market and month dummies. Additionally, z_i is a vector of application shifters that includes the total number of banks' branches in the local market where the borrower is located, and the number of previously related banks of the borrower in the previous year. We provide a discussion of the role of these application shifters in our identification discussion below.

In terms of loan repayment, we adopt the same specification as Einav et al. (2012) for the loan repayment share. In particular, we let the repayment share in equation (1.6) be the following function of borrower covariates and contract terms:

$$s_i = \min\{\exp(x_i'\alpha_X + \alpha_L L_i + \alpha_T T_i + \varepsilon_{Si}), 1\} \tag{1.12}$$

which has the advantages that: (i) it is bounded in the unit interval, and that (ii) it accommodates the possibility of a mass point at full repayment, something we do observe in the data. The vector x_i in this specification is the same as that in the application equation above.

Moreover, we specify the joint distribution of application and repayment shocks F_ε as a bivariate normal:

$$\begin{pmatrix} \varepsilon_A \\ \varepsilon_S \end{pmatrix} \sim N\left(\begin{matrix} 0 \\ 0 \end{matrix}, \begin{matrix} \sigma_A^2 & \rho\sigma_A\sigma_S \\ \rho\sigma_A\sigma_S & \sigma_S^2 \end{matrix} \right) \tag{1.13}$$

where ρ determines the extent of adverse or advantageous selection in the market. In particular, $\rho < 0$ implies adverse selection, as riskier borrowers are more likely to apply for loans; whereas $\rho > 0$ implies advantageous selection, as then safer borrowers are more likely to apply for loans. Moreover, σ_A^2 and σ_S^2 are respectively the variance of application and repayment shocks, and we normalize σ_A^2 to 1. While restrictive, assuming a normal distribution has the advantage of providing a closed form relationship between the conditional and unconditional distributions of interest, something that related previous work has also exploited (e.g., Einav et al. 2012; Crawford et al. 2018). Note that, under this specification, the demand side of the model takes the form of a standard selection model with a normality distributional assumption (Heckman, 1979).

Banks' Costs. We specify the cost function of banks as $m_{ij} = f_i - L_i\omega_{ij}$ such the bank-borrower idiosyncratic component is measured per loan unit. For the match-value component of cost, we assume that it follows an i.i.d. extreme value distribution, $\omega_{ij} \sim T1EV(\delta_{ij}, \sigma_\omega)$. We parametrize the location parameter of this distribution as $\delta_{ij} = \tau_j + \gamma r_{ij}$,

where τ_j is a bank-specific intercept, and r_{ij} is an indicator for a previous relationship between borrower i and bank j. Bank fixed effects τ_j allow for banks to hold cost differences that are constant across borrowers. Allowing for cost to depend on previous relationships is motivated by differences in approvals and interest rates between previously related and non-related borrowers that are documented in Table 1.2. Therefore, the parameter γ captures the potential incumbency advantage that banks previously related to a loan applicant hold relative to non-related banks.[40] Finally, we denote the idiosyncratic component of ω_{ij} as $\varepsilon_{\omega ij}$, which captures variation in cost at the borrower-bank level, which could be driven by heterogeneity in banks' services in local markets or relationships between borrowers and local branch officers.[41]

This specification of banks' costs is consistent with several of the facts in Section 3.2.3. In particular, we specify cost heterogeneity in ways suggested by these facts, namely by: (i) allowing for expected default cost to vary across borrowers according to borrower observables, (ii) allowing bank costs for a given borrower to vary across banks, and (iii) allowing bank costs to depend on previous relationships with borrowers, thus introducing the potential for incumbency advantages.

1.5.2 Identification

We discuss how variation in the data identifies the model and describe our identification assumptions. We assume that borrower covariates, loan amount and term, and application cost shifters (x_i, L_i, T_i, z_i) are exogenous. The main identification assumption is conditional independence between the idiosyncratic component of cost shocks ω_{ij}, and application and repayment shocks $(\varepsilon_{Ai}, \varepsilon_{Si})$. Formally, this is:

$$\varepsilon_{\omega ij} \perp\!\!\!\perp (\varepsilon_{Ai}, \varepsilon_{Si}) | (x_i, L_i, T_i) \tag{1.14}$$

40. The advantage of assuming an extreme value distribution for the match-value component of cost is that it provides closed form expressions for distributions of order statistics of ω_{ij}, which are useful for estimation as discussed below. We summarize these properties in Appendix A.2.4. Proofs for these results are available in Froeb et al. (1998).

41. As an example of the cost heterogeneity captured by $\varepsilon_{\omega ij}$, Drexler and Schoar (2014) use data from a large Chilean bank to show that loan officer turnover has sizable effects on loan approval and borrower default behavior.

which implies that the idiosyncratic component of banks' costs is unrelated to unobservable determinants of application and repayment behavior, once borrower observables are accounted for. The economic implication of the assumption is that banks do not have any informational advantage relative to the econometrician in terms of the determinants of borrower application and repayment behavior, that affects banks' costs and, therefore, pricing. While restrictive, this assumption relies on the fact that our detailed dataset is the same dataset that the regulator provides to banks for pricing purposes. Note that this assumption does not imply that banks' costs are invariant to borrower attributes and application behavior. In fact, banks' consider observable risk for pricing and also infer unobservable risk from applications. Under this assumption, we can treat identification and estimation of the demand and supply sides of the model separately.

Applications and Repayment. The demand side of the model has the structure of standard selection models, where application is the selection equation and repayment is the outcome equation, and where the correlation between the unobservable components of them has the interpretation of adverse or advantageous selection. Parametric and non-parametric identification of this model is established in Heckman (1979) and Das et al. (2003), respectively. The latter emphasizes the importance of exclusion restrictions for identification. In that line, we exploit two application cost shifters in z_i as exclusion restrictions. First, we include the number of branches in the local market as a measure of local bank density, which should reduce application cost. This shifter is in line with papers that exploit distance as a shifter of school applications (e.g., Walters 2017). Second, we include the number of previously related banks as a measure of previous experience dealing with banks, which should make the application process easier to navigate and decrease application cost. Both of these variables arguably shift application choices, but they are are unlikely to directly affect the utility that borrowers obtain from loans or their repayment behavior.

Given the model specification and our identification assumption, the intuition for how variation in the data identifies the demand side of the model is as follows. Application responses to variation in (x_i, L_i, T_i, p_i) identify δ in the application equation. Moreover,

repayment responses to variation in (x_i, L_i, T_i) identify α in the repayment equation. Regarding identification of the joint distribution of application and repayment shocks F_ε, the intuition is that consumers observed applying for loans when the model predicts they should not, are likely to have a high ε_{Ai} shock. The conditional correlation between those shocks and observed repayment identifies ρ. In particular, if those borrowers are observed to repay less, then ρ is negative and there is adverse selection.

A relevant concern regarding this strategy is endogeneity of loan prices, which combine policy variation induced by changes in interest rate regulation with variation induced by risk pricing by banks. Under the identification assumption in equation (1.14), (x_i, L_i, T_i) is all the information that enters both banks' pricing and consumers' application choices, and therefore application responses to price variation conditional on such vector identify price sensitivity. However, if the assumption fails and banks observe drivers of applications that are unobservable to the econometrician and exploit them for pricing, then identification of price sensitivity fails. We use coefficient stability and control function approaches in robustness checks that assess this assumption, which provide support to it.

Banks' Costs. The identification of banks' costs follows from standard arguments in the auctions literature. Assuming independence in cost shocks ω_{ij} across banks and borrowers, our model for the supply side of the market corresponds to an independent private values auction. As established by Athey and Haile (2002), the distribution of values in asymmetric independent private values auctions is non-parametrically identified from transaction prices and the identity of the auction winner. In our setting, we observe prices and the identity of the bank for all contracts in the market, and therefore the distribution of banks' costs is identified.

Figure A.18 provides a diagram that connects data to supply side primitives in our model. For unconstrained approvals, observed prices are a function of the cost of the second lowest bank. Moreover, for constrained approvals, observed prices are those implied by the interest rate cap and are bounded from below by the chosen bank cost and from above by the unconstrained price. Finally, for rejections, prices implied by the interest rate cap are bounded from above by the cost of the lowest cost bank. Thus, we learn about the

underlying cost function of banks by combining our model with data on bank choices, loan prices and application outcomes.

We relate this identification argument to our specification of banks' costs. Conditional on observable funding cost f_i, identification of banks' costs relies on variation in contract prices, bank choices, and application outcomes. First, identification of constant cost differences across banks τ_j rely on differences in prices of chosen banks across banks. Second, identification of incumbency advantage γ relies on variation in prices within chosen banks across applicants with and without a previous relationship with the bank. Finally, any remaining variation in loan prices within banks and bank-borrower relationships identifies the scale of idiosyncratic cost shocks, σ_ω.

1.5.3 Estimation

Estimation proceeds in three steps. In the first step, we estimate the parameters of the application equation using data from the pool of potential applicants. In the second step, we estimate the repayment equation using data on loan performance for signed contracts. In the third step, we use estimates from the second step to compute fitted repayment risk and then proceed to estimate banks' cost by exploiting the auction structure of the supply side of the model. All three equations are estimated by maximum likelihood.

Applications and Repayment. The joint estimation of the application and repayment equations in (1.11) and (1.12) proceeds in three steps. The two first steps are related to estimation of components of the application equation in equation (1.11) that are not observed for every consumer in the sample, and that we then use as inputs in estimation of the key parameters in that equation.

The first step deals with the fact that loan terms (L_i, T_i) are not observed for non-applicants. We estimate the conditional distribution of loan amount and term using data from applicants and then draw from that distribution for non-applicants. In order to deal with concerns related to selection into application, we implement a control function approach in this step, similar to Attanasio et al. (2008) and based on Das et al. (2003). In the first stage, we estimate a flexible probit model for applications on a rich vector of borrower

covariates, and application shifters in z_i. In the second stage, we compute fitted propensity scores using estimates from the first stage and add that propensity score as a control function in a regressions of loan amount on the same set of borrower covariates. Finally, we estimate an ordered logit model for loan term monthly bins on the same set of borrower covariates and loan amount. We use estimates from that second stage to draw loan amount and term for non-applicants in the sample. As expected, predicted loan amount differs for applicants and non-applicants: loan amount for applicants is \$1,000 larger on average, equivalent to 0.14 standard deviations. For further detail on this procedure, see Appendix A.2.1.

In the second step, we deal with the fact that the approval probability P_C and the density of loan prices conditional on approval $f_{p|C}$ enter the application equation and are not directly observed in data for each borrower. First, we estimate P_C using a probit model for an indicator of application approval on a vector of borrower covariates, previous relationship variables, as well as application amount and term from the first step. We compute fitted approval probabilities for each consumer in the sample and use them as inputs in the third step below. Second, we estimate $f_{p|C}$ using a kernel density estimator after conditioning on the same vector of variables. We use draws from this estimated conditional density in the third step of estimation below. We let both P_C and $f_{p|C}$ to vary across time, to capture the effects that variation of interest rate caps over time have on them. The strategy of estimating these elements from data in a previous stage and use them as inputs for the last step is similar to that in Kawai et al. (2018). We provide more detail about this procedure in Appendix A.2.1.

In the third step, we estimate the parameters in the application and repayment by maximum likelihood using inputs from the first and second steps above.[42] We exploit the assumed joint normality of $(\varepsilon_{Ai}, \varepsilon_{Si})$ in equation (1.13) to derive the likelihood of the data, which provides closed form expressions for the distribution of repayment shocks ε_{Si} conditional on application shocks ε_{Ai}. For a detailed derivation of the likelihood function,

42. We use 100 Halton draws from the estimated density of prices conditional on approval to compute the expected indirect utility from signing a loan contract. Train (2009) argues that Halton draws have better coverage properties than pseudo-random draws, which in practice implies that 100 Halton draws provide a similar level of efficiency than simulation with 1,000 pseudo-random draws.

see Appendix A.2.2.

Banks' Costs. We exploit the structure of the auction model and the distributional assumption imposed on ω_{ij} to estimate the distribution of banks' costs by maximum likelihood. In a first step, we compute fitted repayment risk $E_\varepsilon[\varphi(S_i)]$ using estimates from the application and repayment equations and 100 Halton draws for $(\varepsilon_{Ai}, \varepsilon_{Si})$.[43] Conditional on that input, we work separately on the corresponding likelihood for each of the three potential outcomes in equation (1.9). For the derivation of the likelihood function, see Appendix A.2.3.[44]

Estimating Dataset. We estimate the model using a sample of potential applicants for 2013 and 2014, which are the years before and after the policy change. This sample includes 316,384 potential applicants, of which 49,883 apply for loans. Given application events are rare at a monthly frequency, we collapse the data at the yearly level by including data for the application month for consumers who apply within a year, and data for a random month in the year for consumers that do not apply within a year. We define the set of banks over which consumers shop as the 9 largest banks in the market, which account for 98% of market share. All consumers in the estimating dataset are located in markets in which all 9 banks offer consumer loans.

We allow for observable heterogeneity on two sets of parameters. First, we let the price sensitivity coefficient differ across low- and high-risk borrowers. Second, we allow for the coefficients in banks' costs to differ by loan term, so as to allow for loans of different terms to have different monthly costs for banks.

43. We employ a annual discount rate of $r = 5\%$ for all banks in the market for both estimation and counterfactuals.

44. We describe useful properties of distributions of order statistics of the T1EV distribution in Appendix A.2.4. The relevance of such properties is that they allow for obtaining closed form expressions for the likelihood of each of the potential outcomes of the model in terms of observables, which greatly simplifies estimation.

1.5.4 Results

Application Behavior. Table 1.6-A displays our estimates for the application equation.[45] We find that borrowers are more likely to apply for loans when facing a higher approval probability, and increasing the approval probability by 5 p.p increases the probability of application by 5 p.p. Riskier borrowers are more likely to apply and, in particular, a 5 p.p increase in borrower predicted default probability increases the application probability by 1.75 p.p. Moreover, female and older consumers are less likely to apply. Loan amount increases the probability of application, and a loan amount \$5,000 larger increases the probability of application 1.1 p.p. Finally, borrowers are price sensitive and higher expected prices reduce the application probability. High-risk borrowers are less price-sensitive than low-risk borrowers, for instance a \$200 increase in expected monthly payment decreases the application probability of the former by 1.5 p.p, and of the latter by 2.4 p.p.

As discussed in Section 1.5.2, a concern for our strategy is the potential for unobservables that drive both application decisions by borrowers and pricing decision by banks, which would be captured by our estimates of price sensitivity, δ_p. We address this concern by implementing two robustness exercises. First, we assess the stability of $\hat{\delta}_p$ when estimated using a cumulative set of covariates in the application equation, in line with Altonji et al. (2005). Figure A.20 shows that $\hat{\delta}_p$ from specifications that do not include borrower risk scores and other borrower covariates differ remarkably from those that include such variables. Moreover, the results show that adding additional borrower covariates after accounting for risk score has only minor effects on $\hat{\delta}_p$. Second, we employ a control function approach to provide additional evidence for the robustness of $\hat{\delta}_p$. We follow Petrin and Train (2010) and implement a two-step procedure. In the first step, we regress loan monthly payments on covariates in x_i and a cost shifter of prices. We use funding cost as a shifter, where the funding rate provides variation across time, and heterogeneity

45. We report standard errors based on the inverse of the hessian of the log-likelihood functions we maximize. This procedure does not account for the fact that estimation proceeds in steps. Therefore, our standard errors are possibly incorrect and likely overestimate the precision of our estimates. Bootstrapped standard errors are work in progress. While we are likely underestimating standard errors, the fact that most of our estimates are statistically significant at very high confidence levels suggests our conclusions are unlikely to change after adjusting standard errors.

in loan amount and term across consumers provides individual level variation. In the second step, we include the residuals from the first stage as an additional covariate in x_i, with the objective of controlling for unobservable drivers of prices. The last estimates in Figure A.20 are the result from this approach, and show that our estimates $\hat{\delta}_p$ do not change substantially.[46] While not conclusive, these results suggest that the set of covariates included in x_i for estimation might be able to deal with the concern about unobservable drivers of applications and loan pricing.

Estimates of application costs point in the expected directions. Both the number of branches in the local market and consumers' previous experience with banks reduce application costs, as expected. In particular, having 100 more branches in a local market increases application probability by 0.9 p.p, whereas holding a previous relationship with an additional bank increases the application probability by 2.9 p.p.

Loan Repayment Behavior. Estimates for the loan repayment equation are displayed in column (3) of Table 1.6-A. As expected, riskier borrowers repay less on their loan contracts. In fact, a 5 p.p increase in borrower risk score decreases repayment share by by 0.6 p.p. Moreover, female and older borrowers display better repayment behavior. In terms of loan terms, borrowers taking larger loans tend to repay less, while borrowers taking longer term loans display the opposite behavior. Finally, our estimate of σ_S implies there is substantial unobservable borrower risk.

Adverse Selection. We find no compelling evidence of adverse or advantageous selection in this market, conditional on borrower risk scores. Our point estimate for ρ is close to 0 in magnitude and is not statistically significant. However, our estimate is not precise and thus we cannot rule out that some degree of adverse selection in the market. This result implies that although there is substantial unobservable repayment risk σ_S, that risk does not drive application behavior, conditional on (x_i, L_i, T_i). This result does not imply there

46. Our estimate for the coefficient on the control function is -18.21 with standard error 0.93, thus statistically significant. This suggests that including it indeed controls for such potential unobservables. However, the fact that estimates $\hat{\delta}_p$ remain similar to those without the control function suggests in turn that the relative importance of those unobservables relative to observables in (x_i, L_i, T_i) is minor.

is no selection on observables. In fact, our results show that riskier borrowers are more likely to apply for loans and are less likely to repay them. However, that is accounted for in x_i and is therefore not reflected in our estimate for ρ.

We address the role of observables in determining our selection estimate. Figure A.21 shows estimates of ρ using different sets of borrower covariates in x_i in *both* the application and repayment equations. We find that not accounting for observables yields estimates that would provide strong evidence of adverse selection ($\hat{\rho} < 0$). However, once we include borrower risk scores and income, point estimates of ρ remain close to 0, in line with our preferred specification. These result suggest that observables in our data—which are the same provided by the regulator to banks for risk assessment—account for most of risk selection into the market.

Banks' Costs. Table 1.6-B displays estimates of banks' costs, which reveal substantial heterogeneity in banks costs. To interpret these estimates, we describe how they relate to the monthly payments associated with a loan of \$2,000, that have median and standard deviation (σ_p) of \$118.62 and \$69.90, respectively. Estimates of bank-specific components of ω_{ij} imply sizable cost differences across banks: on average, the cost difference between the most and least efficient banks for a given loan is of \$25.99 per month, equivalent to $0.37\sigma_p$. Moreover, we estimate that having a previous relationship with a bank provides an incumbency advantage to the bank. In particular, having a previous relationship reduces the monthly cost of providing a \$2,000 loan by \$32.74 per month, around $0.46\sigma_p$. Finally, our estimates show that the standard deviation of bank-borrower idiosyncratic shocks is large: a 1 s.d increase in this shock decreases cost by \$19.17 per month, equivalent to $0.27\sigma_p$. This suggests that unobserved variation in costs across banks is a relevant determinant of residual price dispersion. There is heterogeneity in estimates across cost bins, although without a clear pattern associated with loan term. This suggests that after accounting for funding cost the cost per dollar of loan does not correlate strongly with loan term.

It is useful to understand how these estimates relate to the data. First, estimates of bank cost fixed effects τ_j are aligned with observed bank market shares, as displayed by Figure A.19-a. The model rationalizes high market shares as cost advantages, captured by higher

fixed effects in ω_{ij}. Second, market shares and the share of previously related borrowers are positively correlated, as displayed by Figure A.19-b. The model rationalizes this correlation as that banks hold an incumbency advantage when serving previously related borrowers relative to other banks without such preexisting relationships. This explains our positive estimate for γ.

Model Fit

We examine model fit by using the estimated parameters to simulate equilibrium outcomes and compare simulated to observed outcomes. We run this simulation and all simulations in the next section on the estimating dataset. In particular, we proceed as follows:

1. Draw shocks for applications, repayment and cost. Specifically, (i) draw application and loan repayment shocks for each borrower in the sample from the estimated joint distribution, $\{\varepsilon_{Ai}, \varepsilon_{Si}\}$; and (ii) draw a cost shock for each bank-borrower in the sample, ω_{ij}.

2. Draw shocks for integration steps. Specifically, (i) draw a vector of N_ω bank-borrower cost shocks for integration of prices by borrowers, $\{\omega_{ij}^{(s)}\}_{s=1,j\in\mathcal{J}}^{s=N_\omega}$; and (ii) draw a vector of N_S loan repayment shocks per borrower for integration of repayment risk by banks, $\{\varepsilon_{Si}^{(s)}\}_{s=1}^{s=N_S}$.

3. Simulate optimal prices and approval decisions for each of the N_ω vectors of cost shocks for a given interest rate regulation $\bar{p}_i$, which are required for simulating application decisions. This step requires solving a fixed point problem, because banks take the expectation of repayment risk conditional on application into account, and application in turn depends on expected approval probability and prices. We proceed by: (i) computing simulated unconditional repayment risk as a starting point, (ii) computing simulated application decisions, (iii) computing expected approval probability P_{Ci} and monthly payments conditional on approval $\{p_i^{(s)}\}_{s=1}^{s=N_\omega}$ given simulated repayment risk, (iv) computing simulated conditional repayment risk, and (v) repeating (ii)-(iv) until convergence of simulated monthly payments. The outputs

of this step are simulated approval probability P_{Ci}, monthly payments $\{p_i^{(s)}\}_{s=1}^{s=N_\omega}$, and expected repayment risk $E_\varepsilon[\varphi(S_i)]$.

4. Simulate application decisions for each borrower a_i, by computing application probabilities using simulated approval probabilities and monthly payments from Step 3 along with draws for application shocks from Step 1.i.

5. Simulate approval and pricing decisions by banks (L_i, p_i), using draws for cost shocks from Step 1.ii and simulated repayment risk from Step 3.

6. Simulate repayment outcomes for borrowers s_i, using estimates for the repayment equation along with repayment draws in Step 1.i.

Figure 1.5-a shows that simulated application outcomes are close to observed outcomes, although the model overpredicts constrained approvals and underpredicts unconstrained approvals relative to the data. This suggests applicants may face frictions in their choice sets formation that our model does not account for. Figure 1.5-b shows that predicted market shares track observed market shares closely. Moreover, Figure 1.5-c shows that the model fits the distribution of loan prices well, with a correlation between predicted and observed prices of 0.95. Finally, Figure 1.5-d shows that the estimated model provides a good fit of the density of loan repayment share.

Finally, Figure A.22 shows estimated expected profit margins, which are 29.6% on average, and display substantial dispersion. We use the model and simulated data under actual regulation to decompose prices into three components: cost, risk, and market power. This decomposition follows a rearranged version of equation (1.8).[47] Our results show that, on average, funding and banks' costs jointly account for 71.2% of loan prices. Risk accounts for 9.8% of the spread between loan monthly payment and cost, while market power

47. In particular, we decompose prices in risk, cost and market power as follows:

$$p_i^u = \underbrace{\frac{\varphi(T_i) - E_\varepsilon[\varphi(S_i)]}{\varphi(T_i)} p_i^u}_{\text{Risk}} + \underbrace{f_i - L_i\omega_{i(1)}}_{\text{Mg. Cost}} + \underbrace{L_i\omega_{i(1)} - L_i\omega_{i(2)}}_{\text{Market power}}$$

and then compute the share of each component over the loan monthly payment. In the case of constrained loans, the market power component of this decomposition decreases as the price cap becomes binding.

accounts for the remaining 90.2%. Our estimates thus imply that banks hold substantial market power in this setting.

Simulated Effects of Interest Rate Regulation

We simulate equilibrium outcomes for different regulation levels, corresponding to the level at the moment of the policy change, and those 1 and 2 years after the policy change, which is November 2013, November 2014 and November 2015. We then compare simulated effects to estimated effects to assess model predictions.

Results from these simulations are mostly in line with the evidence presented in Section 1.3 and are summarized in Table 1.7. The model predicts that stronger interest rate regulation decreases the number of loans by 23.7%, which combines a decrease in applications and an increase in rejections by banks. As highlighted in Section 1.4.1, the effect of interest rate regulation on application choices depends upon its relative effects on decreased approval probability and decreased expected loan prices. In this case, we find a decrease in applications that in turn implies that the former effects dominates the latter. Moreover, loan monthly payments on loans approved under stronger regulation decrease by $2.59 and the mark-up on such loans decreases by 2 p.p, reflecting that stronger interest rate regulation is in fact protecting consumers who remain in the market. These simulated effects are in line with our analysis in Section 1.3, where we estimated a 19% decrease in the number of loans and a $3.26 decrease in loan monthly payments.

1.6 Welfare Effects of Interest Rate Regulation

1.6.1 Welfare Analysis

We exploit our estimated model to estimate the welfare effects of interest rate regulation in this setting. In particular, we adopt a revealed preferences approach and exploit observed application choices along with our estimates of willingness to pay to estimate changes in expected consumer surplus; whereas we exploit observed prices and our estimates of

banks' costs to estimate changes in banks' profits.[48]

Expected consumer surplus for consumer i under an interest rate cap $\bar{p}_i$ is given by the following expression:

$$E[CS_i(\bar{p}_i)] = \frac{1}{\hat{\delta}_p} \int \max\{P_{Ci}(\bar{p}_i) \int u_L(x_i, L_i, T_i, p; \hat{\delta}) f_{p|C}(p; \bar{p}_i) dp - z_i'\kappa + \varepsilon_A, 0\} f_{\varepsilon_A}(\varepsilon_A) d\varepsilon_A$$

where interest rate regulation enters through both the approval probability and the density of prices conditional on approval. We use our model to construct all components on the right hand side of this expression, and calculate the effect of a change in interest rate regulation from $\bar{p}_i^0$ to $\bar{p}_i^1$ on expected consumer surplus as $\Delta E[CS_i] = E[CS_i(\bar{p}_i^1)] - E[CS_i(\bar{p}_i^0)]$. This change in expected consumer surplus is measured from an expected utility perspective, and thus it reflects how credit market conditions change for potential applicants in terms of approval probability and expected prices, regardless of whether ex-post those applicants are approved and sign contracts at lower prices or rejected.

We find that expected consumer surplus decreases by an average and median of \$82.47 and \$40.24 per month respectively, which is equivalent to 3.5% and 1.7% of average monthly income. There is substantial heterogeneity in estimated effects on expected consumer surplus, as displayed in Figure 1.6-a. Moreover, the distribution of estimated effects on expected consumer surplus is skewed: 66% of potential borrowers display changes in expected consumer surplus smaller than average, and less than 29% of them display decreases in expected consumer surplus of more than \$100 per month. On the other hand, bank monthly profits decrease by \$2.41 per potential borrower in the market under stronger interest rate regulation, which adds up to 18% of total profits in the market. The combination of decreases in consumer surplus and profits implies that average welfare per consumer in the market decreases.

In principle, stronger interest rate regulation may harm some consumers and benefit others. We find that adverse effects dominate positive effects in this setting. In fact, our simulation implies that expected consumer surplus decreases for 82.3% of consumers,

48. All our calculations related to changes in bank profits focus on variable profits from loan contracts. Therefore, any changes in fixed costs or screening costs associated with stronger interest rate regulation are not accounted for in this analysis.

remains unchanged for 1.5%, and increases for 16.2% However, the average loss for the harmed is \$100.01, whereas the average gain for beneficiaries is only \$0.32. Therefore, the effect of a decreased approval probability dominates that of a decreased expected monthly payment in terms of application incentives. These effects are positively correlated as displayed in Figure 1.6-b, and the lack of borrowers in the upper-left area of the plot explains the small number of borrowers obtaining benefits from stronger regulation. Few borrowers receive large decreases in expected prices without large decreases in approval probability.

Risky borrowers are the most affected by interest rate regulation in terms of expected consumer surplus, as displayed in Figure 1.6-c. The average decrease in expected consumer surplus for low- and high-risk borrowers is \$38.70 and \$130.29 per month respectively. This pattern of heterogeneity is driven by three forces: risky borrowers were charged higher prices at baseline, and therefore were more exposed to stronger interest rate regulation; display a stronger preference for loans; and are less sensitive to expected monthly payments.

We decompose changes in borrower expected consumer surplus to further quantify the trade-off between credit access and consumer protection, as follows:

$$\Delta E[CS_i] = \underbrace{(E[CS_i(P^1_{Ci}, p^1_i)] - E[CS_i(P^0_{Ci}, p^1_i)])}_{\text{Credit access}} - \underbrace{(E[CS_i(P^0_{Ci}, p^1_i)] - E[CS_i(P^0_{Ci}, p^0_i)])}_{\text{Consumer protection}}$$

where the first term isolates the effect of lower approval probabilities, and the second term isolates the effect of lower prices, such that the overall effect combines these two effects. We estimate that the average effects of decreased credit access and increased consumer protection on expected consumer surplus are -\$82.63 and \$0.85, respectively. This pattern reflects that the value borrowers place on expected reduced credit access under stronger regulation is substantially higher that value they place on the expected price decrease they would obtain in the market. Both effects are stronger for riskier borrowers, as shown by Figure 1.6-d.

Finally, we study the welfare effects of a range of levels of interest rate regulation. Figure 1.7-a shows that stronger interest rate regulation beyond that in December 2015 would

only further reduce expected consumer surplus. In particular, setting interest rate caps for loans in \$0-\$8,000 would decrease expected consumer surplus by almost \$150. On the other hand, there would not be gains in terms of expected consumer surplus from setting interest rate caps higher than those in December 2013, when regulation was essentially not binding, which implies that any small benefits from increased approval probabilities would be compensated by increased expected prices. Figure 1.7-b shows that the share of consumers that benefit from changes in interest rate regulation is larger for moderate decreases in interest rate caps relative to those in December 2013, but remain below 20% otherwise. However, average gains in expected consumer surplus remain small across the range of regulations we study, which is consistent with the gains from the consumer protection component of interest rate regulation being low relative to the losses due to decreased credit access.

1.6.2 Survey Evidence for the Effects of Reduced Credit Access

In Section 1.3.2, we estimated that stronger interest rate regulation decreased the number of loans. Moreover, in the previous section we adopted a revealed preferences approach to estimate that stronger interest rate regulation decreased the average consumer surplus in the market. In this section, we exploit our survey to provide suggestive evidence about potential channels for how reduced credit access could decrease consumer surplus in our setting.[49]

We study how the effects of economic hardships for household vary depending on whether they deal with them using bank credit. In particular, we exploit information on whether households experienced economic hardships during the last five years, how they dealt with them, and how it affected consumption and financial outcomes for them.[50]

49. We provide some summary statistics for our household survey in Table A.3. The sample of survey respondents is riskier and of lower income than the average borrower in the market. Households are quite experienced in the credit market and most of them hold checking accounts, credit cards and have held consumer loans.

50. We define economic hardship in the survey as a sustained period of time over which the expenditure of the household was higher than its income. In practice, 58.6% of survey participants identified their households as having been under such situation over the last five years. The questions related to how this shock was dealt are expressively linked to the shock itself, rather than general questions about credit access.

We compare outcomes of households that did not experience any shocks with those that experienced shocks and financed them by either (i) obtaining bank credit, (ii) liquidating savings or assets, or (iii) using some other source, including informal sources of credit or increased labor supply. We estimate the following specification:

$$y_i = \alpha + \beta_c credit_i + \beta_s savings_i + \beta_o other_i + x_i'\gamma + \varepsilon_i$$

where y_i is the outcome of interest, x_i is a vector of control variables that includes household income, vulnerability and age of survey respondent, as well as loan approval probability, estimated using administrative data. The coefficients of interest are β_c, β_s and β_o, which measure the difference between outcomes for households that experienced no negative shock relative to those that experienced a negative shock and financed it with either credit, savings or other, respectively.

First, we study whether credit access in the event of shocks is associated with household consumption. In particular, we collect information on whether households cut expenditure on relevant items (e.g., transportation, education, health, travel, among others) due to economic hardships experienced over the last five years. Figure 1.8-a shows that households that experienced these shocks did cut expenses in several items, but that those effects are smaller for households that obtained bank credit upon those shocks relative to those that employed other means. In particular, households that dealt with shocks using bank credit cut expenses for an average of 7.5 p.p less items than households that dealt with economic hardships using other means. These results suggests that reduced credit access might harm consumption smoothing, similar to findings in Morse (2011).

Additionally, we study whether credit access in the event of shocks affects the ability of households to repay their financial commitments. In particular, we focus on whether households stopped paying bills (e.g., health bills, rent, mortgage payments, credit card and consumer loans payments, among others). Figure 1.8-b shows that households that obtained credit access upon economic hardships do not display any differential behavior relative to households that did not experience economic hardships. However, households that did not access credit are significantly more likely to have unpaid bills than the latter.

In particular, households that dealt with economic hardships using bank credit are 36 p.p less likely to have any unpaid bill than those that dealt with economic hardships using other means. This suggests that credit access might provide liquidity to avoid financial distress episodes associated with debt repayment, as in Zinman (2010).

These results provide suggestive evidence that credit access serves as a means for consumption smoothing and alleviation of financial distress upon economic hardships, although we do not claim that our estimates describe a causal relationship between them. We interpret this evidence as complementary to our estimates of effects on consumer surplus. These results are in contrast with research finding adverse effects of access to payday loans on financial distress (e.g., Melzer 2011; Gathergood et al. 2018; Skiba and Tobacman 2018). This contrast might be driven by the fact that interest rates in the market we study are substantially lower than those charged on payday loans—which are often the setting for those studies—, and therefore access to this type of credit is less likely to lead to financial distress, as in Morse (2011).

1.7 Counterfactual Analysis of Interest Rate Regulation

1.7.1 The Interaction between Market Power and Interest Rate Regulation

The usual motivation for implementing interest rate regulation is to limit usurious behavior, which we define as limiting the exercise of market power by banks. In Section 1.5.4, we showed that stronger interest rate regulation indeed reduced average bank profit margins while simultaneously increasing rejections and reducing the number of loans and overall welfare in the market. In this section, we study how those results vary under alternative competitive environments.

We study the role of the competitive environment by sequentially merging banks in the market, starting from the baseline market structure with 9 banks until all banks are consolidated into a monopoly.[51] For each such market structures, we simulate equilibrium

51. In order to isolate the effect of the number of banks in the market—and to make the ordering of mergers inconsequential for our results—, we remove part of the cost heterogeneity across banks: we set bank fixed effects τ_j and incumbency advantages γr_{ij} to the average across banks.

outcomes for interest rate regulation at November 2013 and November 2015, compute the effects of the policy change, and analyze how those effects change across market structures.

Market power plays a relevant role in determining the equilibrium effects of interest rate regulation. The main results from this analysis are displayed in Figure 1.9, which shows the effect of market concentration on equilibrium outcomes for a given interest rate regulation level.[52] We find that as the number of banks decreases and the credit market becomes more concentrated, the effect of stronger interest rate regulation on expected consumer surplus decreases. This result suggests that when banks have more market power and therefore can charge higher prices conditional on borrower cost, interest rate regulation might be able to play a role in constraining the exercise of such market power by banks, shifting rents from banks to borrowers. However, our results show that even under a market structure with a monopoly in the market, both expected consumer surplus and bank profits decrease under stronger interest rate regulation, such that there would not be any efficiency grounds for such a change in interest rate regulation in this market.[53]

In competitive credit markets where banks do not have substantial market power, the trade-off between exclusion and protection becomes less appealing, as bank profit margins are already low. Thus, interest rate caps in such settings will mainly have credit access rather than consumer protection effects. In contrast, welfare losses introduced by interest

52. For reference, we display equilibrium outcomes for relevant variables under baseline interest rate regulation in Figure A.23 As expected, quantities decrease as the market becomes more concentrated.

53. Market power in our model is due to cost heterogeneity, and thus an alternative way to study the role of market power is therefore to study counterfactuals related to cost heterogeneity. We simulate equilibrium outcomes under different environments in which we remove the different dimensions of cost heterogeneity market power, while keeping the average cost in the market constant. We then compute simulated policy effects for the change between November 2013 and November 2015 and compare those to simulated effects using our baseline estimated model. We find that interest rate regulation has more adverse effects when introduced in more competitive markets. Table A.4 displays results from this exercise. Column (1) displays simulated effects using the baseline model. Column (2) displays simulated policy effects if all banks had a fixed effect τ_j equal to the average across banks, while column (3) shown a case in which incumbency advantages are removed by setting γr_{ij} to be equal to the average of such term across banks. Column (4) displays results of a case in which the variance of idiosyncratic shocks σ_ω is reduced to half of its estimated value. Under all these scenarios, simulated policy effects are larger in magnitude than under the baseline model. Furthermore, column (5) shows results for a scenario where the three sources of market power are limited simultaneously, and the contrast is even stronger. For example, if under the baseline scenario rejections increase by 4.9 p.p and the number of loans decreases by 16.3% as a result of the policy, under the scenario with less market power in column (5), rejections increase by 41.4 p.p and the number of loans decreases by 79.8%.

rate regulation policies will be lower in less competitive environments. In fact, considering that our theoretical predictions regarding effects of interest rate regulation on applications and expected consumer surplus are ambiguous, it might be the case that interest rate regulation can deliver welfare increases in other settings, and that may be more likely whenever banks hold substantial market power.

1.7.2 Risk-Based Interest Rate Caps

Despite the trade-off between consumer protection and credit access, innovation in the design of interest rate regulation has been scant. We argue that the cause of such a trade-off is partly in the mismatch between unsophisticated regulation in the form of constant interest rate caps and sophisticated risk pricing by banks. Risk-based interest rate regulation intends to account for borrower risk heterogeneity and risk pricing.

We consider a counterfactual design that sets interest rate caps differently according to borrowers' attributes. Perfect risk-based interest rate regulation would involve setting interest caps at the cost of each borrower for the most efficient bank. Such regulation would yield efficient market outcomes by fully constraining the exercise of market power by banks. In particular, average welfare would be \$164 higher than under the regulation in place in November 2015, which would stem from the average monthly payment being 17% lower and the number of loans in the market being 65% higher. However, this policy design is hardly feasible, as it would require perfect knowledge of cost by the regulator. Instead, we work on a feasible version of risk-based interest rate regulation, with the broad structure of the current design.

Using the notation of equation (1.1), interest rate caps can be written as $\bar{\iota}_{\ell t} = \bar{\iota}^{r}(\tilde{\iota}_{\ell t-1}; \psi, \alpha_{\ell t})$, i.e. as a function of a reference interest rate, a parameter that operates as a multiplier on that rate (ψ) and a parameter that operates as a mark-up ($\alpha_{\ell t}$). We consider a case in which instead $\bar{\iota}^{r}_{\ell t} = \bar{\iota}(\tilde{\iota}_{\ell t-1}, x_{i\ell t}; \psi, \alpha_{\ell t}, \phi)$. If $x_{i\ell t}$ is some measure of risk and $\frac{\partial \bar{\iota}^{r}}{\partial x_{i\ell t}} > 0$, then this design sets a higher interest rate cap to observably riskier borrowers. We adopt a simple linear example of it and measure its performance relative to the design currently in place.

In particular, let:

$$\bar{\iota}^{r}_{\ell t} = \bar{\iota}_{\ell t} + f(x_{i\ell t}), \quad f(x_{i\ell t}) = \phi \frac{x_{i\ell t} - \bar{x}_{\ell t}}{\bar{x}_{\ell t}}$$

be the risk-based interest rate cap for borrower i with risk score $x_{i\ell t}$, where $\bar{x}_{\ell t}$ is the average risk score and ϕ controls the incidence of risk in interest rate caps. Note that the average level of regulation in the market is the same as under the baseline design for each loan-size bracket p, given $E[f(x_{i\ell t})] = 0$, but the interest rate cap is higher (lower) for riskier (safer) borrowers.

Risk-based interest rate caps recover part of the losses in credit access and welfare imposed by constant interest rate caps. We set November 2015 as the reference level of regulation, once the policy change is fully in place. We simulate outcomes for a range of values for ϕ between 0 and 14. Figure 1.10-a shows that there is a range of values of ϕ for which risk-based interest rate caps increase the number of loans in the market relative to constant interest rate caps. Figure 1.10-b shows a similar pattern for expected consumer surplus and welfare. At its best specification, risk-based interest rate caps increase the number of loans and average expected consumer surplus in the market by almost 2% and \$22 respectively, while average profits per consumer remain constant.[54]

From a welfare perspective, these results suggest that risk-based interest rate caps may be able to manage the trade-off between consumer protection and credit access better than usual constant interest rate caps. This result stems from the fact that banks implement risk pricing in the credit market. In absence of risk pricing, adverse effects of risk-based caps on safe borrowers may actually be larger than under constant caps. The case we analyze here is, of course, an example. Other versions of risk-based interest rate regulation might

54. To further illustrate the effects of risk-based interest rate caps, we study patterns of heterogeneity across borrower risk induced by it. We compare the case of $\phi = 4$ with the baseline case of $\phi = 0$. Figure 1.10-c shows that it affects application outcomes by strongly increasing approval rates for risky borrowers, while slightly decreasing approval rates for safe borrowers. On the other hand, Figure 1.10-d shows that average monthly payments increase (decrease) for risky (safe) borrowers as they face relatively weaker (stronger) interest rate regulation. How this heterogeneity across the distribution of borrower risk aggregates depends on the joint distribution of borrower demand, risk and cost. The fact that our estimates imply that risky borrowers value loans more than safe borrowers explains that the relative benefits from risk-based interest rates are stronger in terms of expected consumer surplus than in terms of loans: as interest rate caps become more aggressive, the benefits in terms of limiting losses in credit access diminish, but the fact that the policy increases the share of risky borrowers in the market implies that it still increases average expected consumer surplus.

further improve market outcomes relative to designs that do not take risk into account.

1.8 Conclusion

In this paper, we study the implications of interest rate regulation in consumer credit markets. Despite the fact that this regulation is widespread and has been used for a long time in credit markets, there is disagreement about its effects. Moreover, its design often lacks sophistication, which may lead to unintended consequences. In this paper, we provide extensive evidence of the effects of interest rate caps on market outcomes and welfare, using the Chilean credit market as a setting. We find that the trade-off between consumer protection and credit access exists, but that adverse effects on credit access dominate consumer protection effects. Thus, while the objective of interest rate regulation is often to protect borrowers from bank market power, we find it ends up mostly harming borrowers' access to credit.

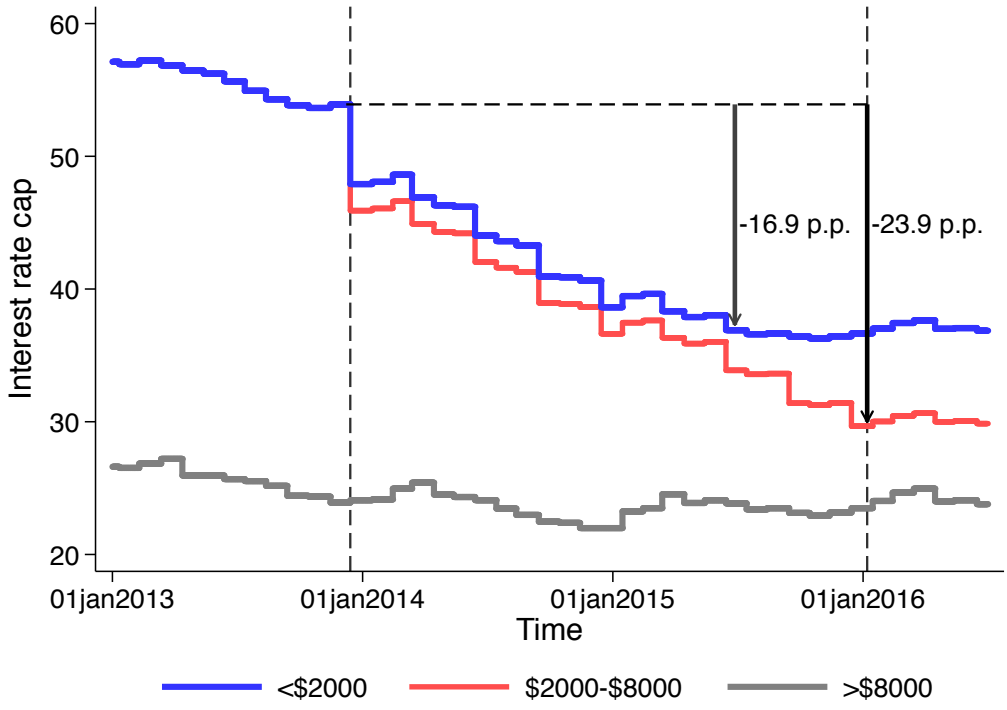
We develop and estimate a model of demand and supply for consumer loans, which we exploit in a variety of ways. First, we use it to estimate welfare effects of interest rate regulation and find that welfare mostly decreased in our setting. Second, we use the model to show that the adverse effects of interest rate regulation are smaller in more concentrated markets as the consumer protection motive becomes more relevant, but that welfare decreases even under a monopoly. Finally, we explore how equilibrium outcomes differ under risk-based interest rate caps, and find that such design reduces adverse effects of interest rate regulation and recovers at least part of the losses in terms of credit access and consumer welfare, without increasing bank profits. This result suggests that this design may perform better in terms of providing consumer protection without harming credit access.

Our welfare analysis follows a revealed preferences approach, and does not account for any behavioral biases that might take place in consumer credit markets (Zinman, 2015; Beshears et al., 2018). In our approach, we exploit consumer application and repayment behavior to estimate our model and estimate welfare effects. We acknowledge that such behavioral biases might affect our conclusions regarding borrower behavior and the welfare

implications of interest rate regulation, and consider it a relevant line for future research. However, evidence from our survey suggests that households that access bank credit upon economic hardships display a higher degree of consumption smoothing and a lower degree of financial distress. This complementary evidence provides support to our findings that does not rely on revealed preferences.

Our analysis shows how a combination of a theoretical framework and data can inform the design of regulation for consumer credit markets, by identifying relevant economic forces at work, and by measuring its implications and their relationship to relevant features of credit markets. Importantly, while our findings show mostly adverse effects of interest rate regulation in our setting, the theoretical predictions of our model regarding its welfare effects are ambiguous. This implies that interest rate regulation might improve market outcomes in other settings with different underlying market and demand structures. However, the fact that most of the related literature points towards adverse or non-existent effects of interest rate regulation on market outcomes suggests such a setting might be uncommon.

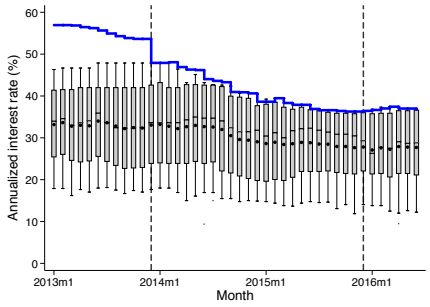
Figure 1.1: Evolution of interest rate caps



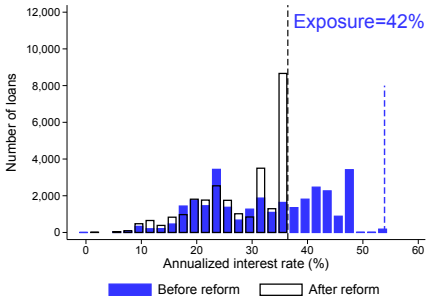
(a) Evolution of interest rate caps

Notes: These figure displays the evolution of the level of interest rate caps for different loan size brackets. The first dashed black line indicates the implementation of Law 20,715, after which interest rate caps for all loans under $8,000 were reduced, in December 2013. The second dashed line indicates the date in which the policy was fully implemented, in December 2015.

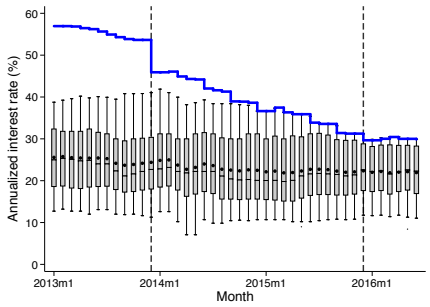
Figure 1.2: Evolution of the distribution of interest rates



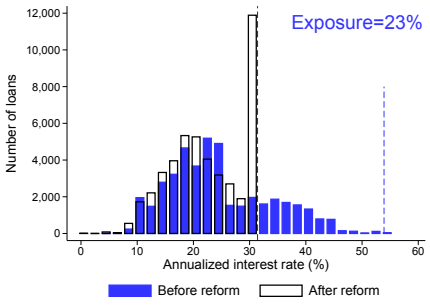
(a) Under $2,000



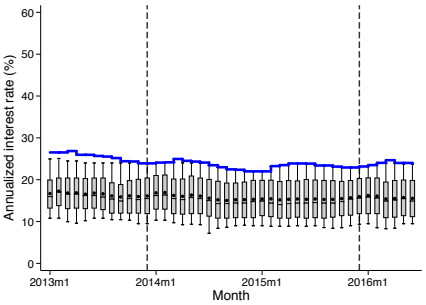
(b) Under $2,000



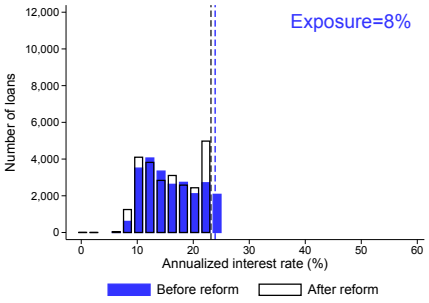
(c) Between $2,000 and $8,000



(d) Between $2,000 and $8,000



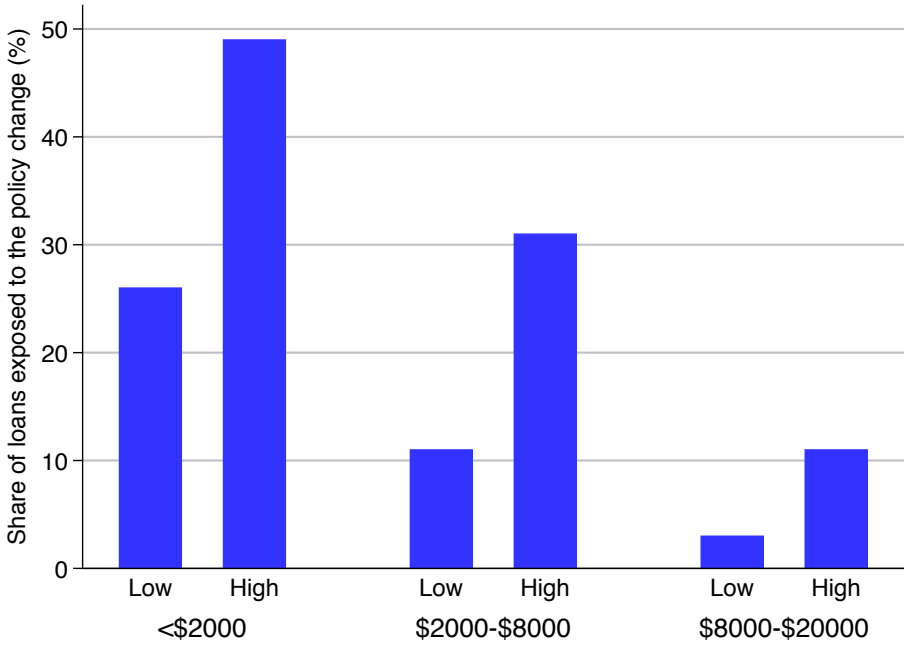
(e) Between $8,000 and $20,000



(f) Between $8,000 and $20,000

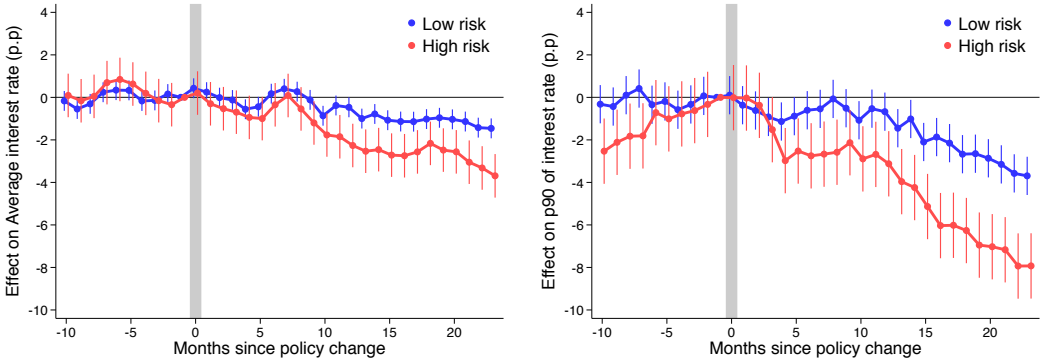
Notes: Panels (a), (c) and (e) in this figure display the evolution of the distribution of interest rates by loan size within each month. Each box displays the 25th, 50th and 75th percentiles of such distribution. Spikes display the 5th and 95th percentiles of it. Black dots indicate the mean of it. In each plot, the blue line displays the current interest rate cap relevant for the corresponding loan size interval. Panels (b), (d) and (f) in this figure display frequency histograms of interest rates for the month before the reform started, December 2013 (blue), and for the month in which it was fully in place, December 2015 (white). The blue dashed line indicates the level of the interest rate cap for each size bracket before the reform was implemented, while the black dashed line does so for the month when the reform was fully in place. Exposure to the policy is calculated as the share of loans that were signed before the policy was implemented at interest rates higher than the interest rate cap once the policy was fully in place.

Figure 1.3: Exposure to interest rate regulation by loan size and borrower risk



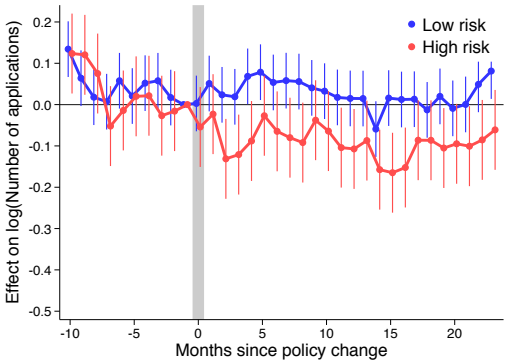
Notes: This figure displays a measure of exposure to interest rate regulation across loan size and borrower risk. Exposure to the policy is calculated as the share of loans that were signed before the policy was implemented in December 2013, at interest rates higher than the interest rate cap once the policy was fully in place in December 2015.

Figure 1.4: Differences-in-differences effects through time

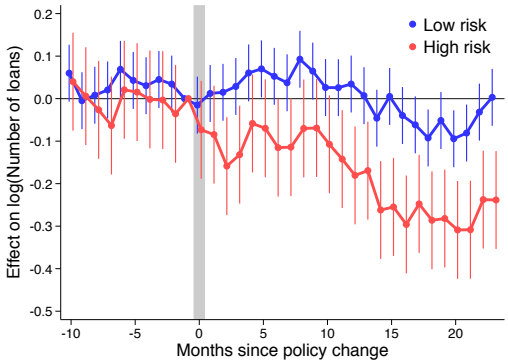


(a) Average interest rate

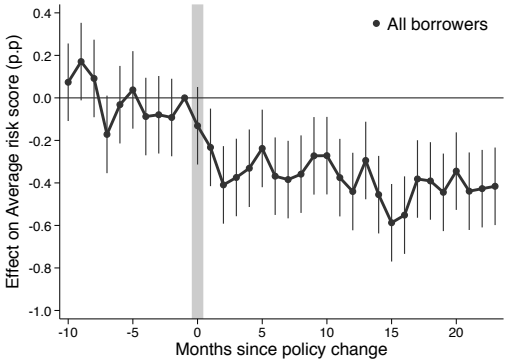
(b) p90 of interest rates



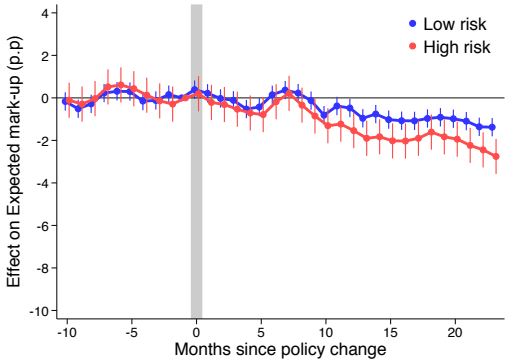
(c) Number of applications



(d) Number of loans



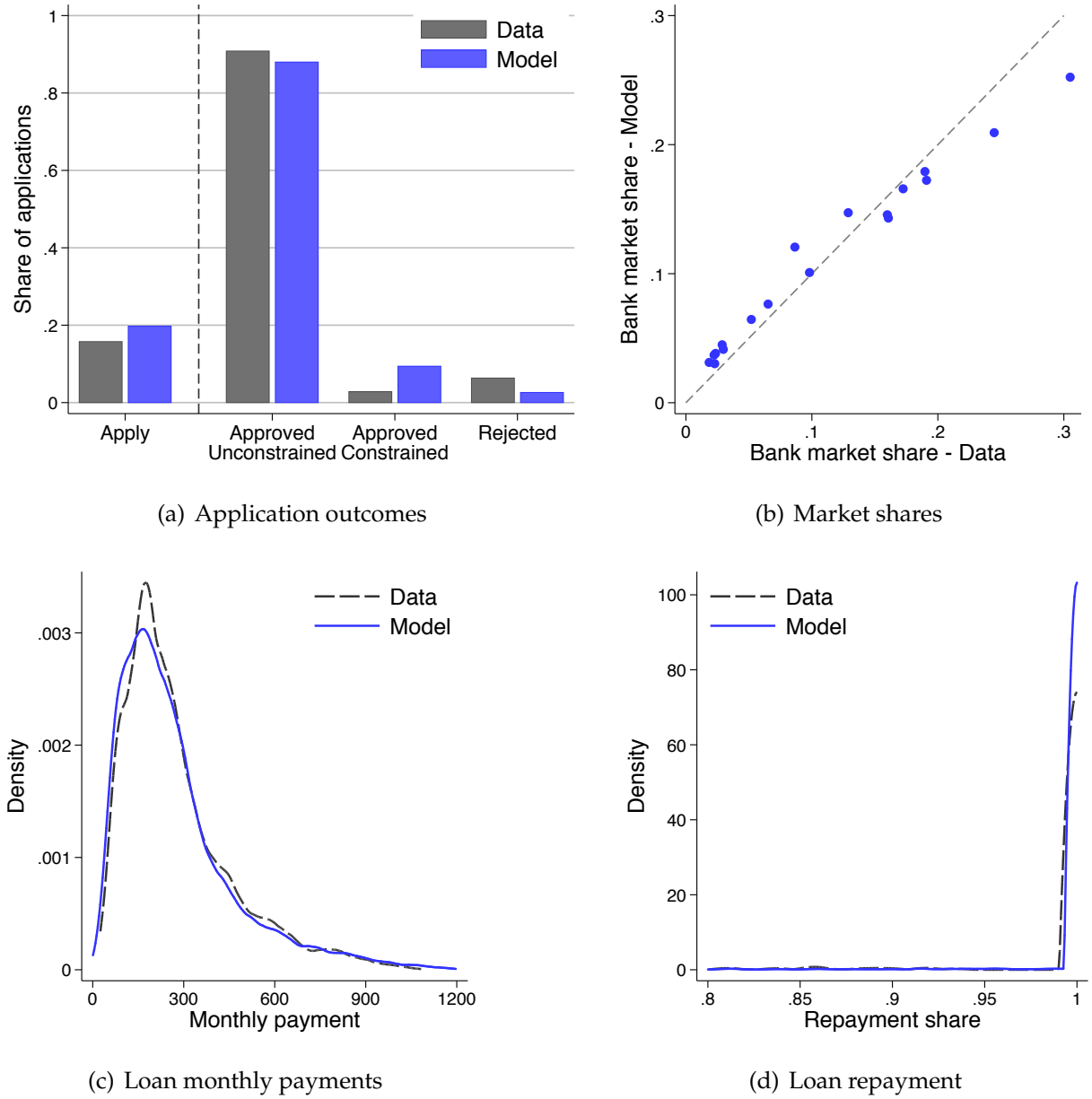
(e) Average risk score



(f) Average expected mark-up

Notes: These figures display results from estimating equation (1.2). Each figure displays results for a different outcome outcome. Within each plot, dots indicate estimated effects for a given month while dashed lines indicate standard errors. Effects for low- (high-) risk borrowers are displayed in blue (red). All regressions are weighted by the number of loans in the product-risk bin before the policy was implemented.

Figure 1.5: Model fit



(a) Application outcomes

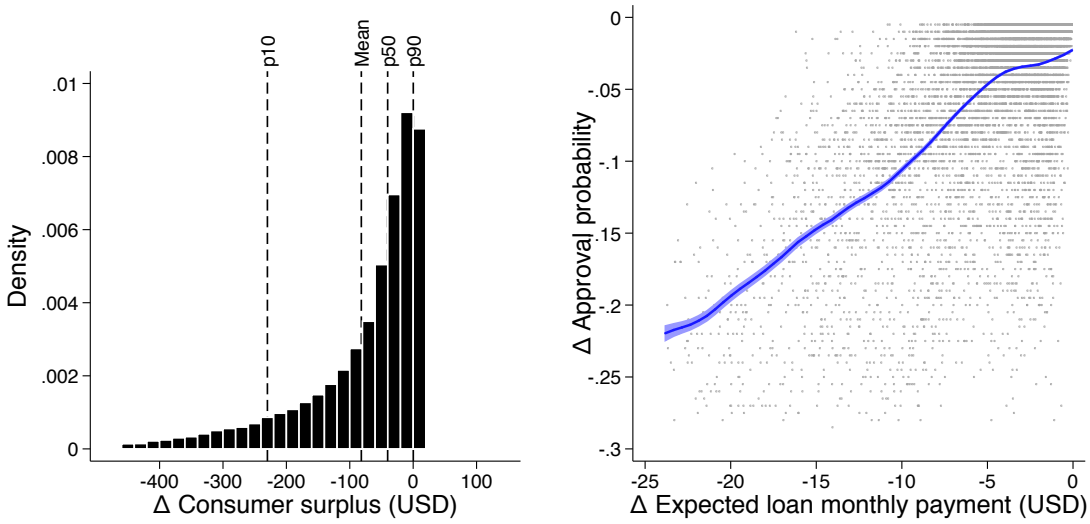
(b) Market shares

(c) Loan monthly payments

(d) Loan repayment

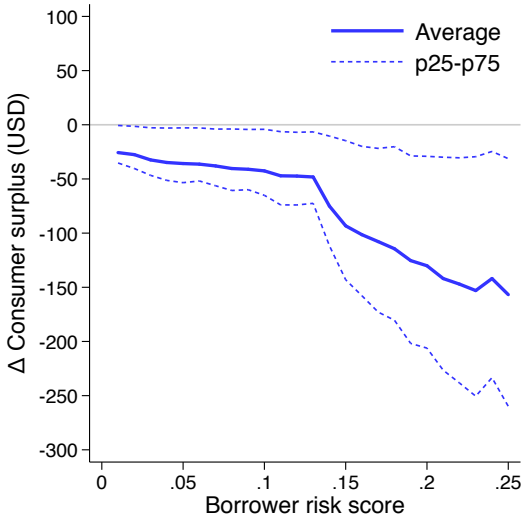
Notes: This figure displays results for model fit. Predictions are implemented as detailed in Section 1.5.4, using estimates from the model described in such section. Panel (a) displays observed and predicted shares of approved, constrained and rejected applications. Panel (b) displays observed and predicted bank market shares. Panel (c) displays the observed and predicted distribution of loan monthly payments. Panel (d) displays the observed and predicted distribution of loan repayment.

Figure 1.6: Heterogeneity in welfare effects of interest rate regulation

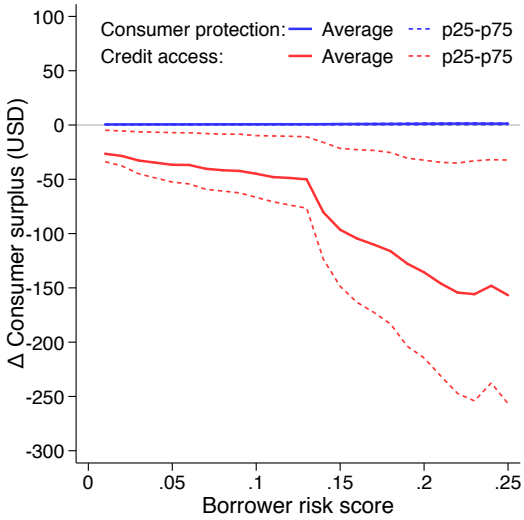


(a) Effects on consumer surplus

(b) Effects on drivers of applications



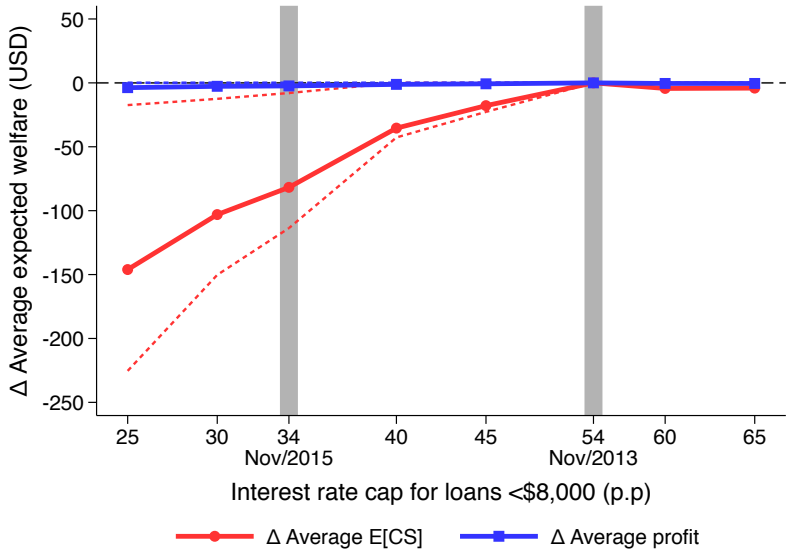
(c) Consumer surplus and risk



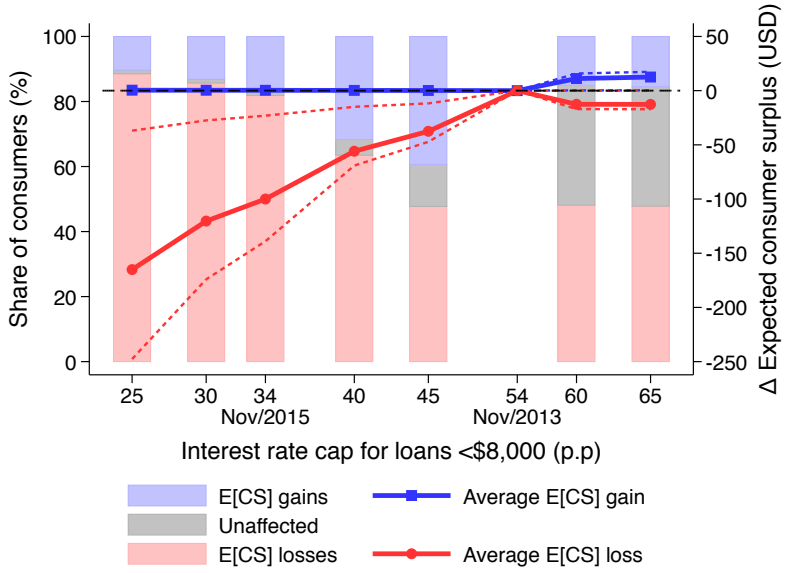
(d) Credit access vs Consumer protection

Notes: These figures display heterogeneity in effects of interest rate regulation across borrowers. All figures compare outcomes under full regulation by November 2015 with outcomes under baseline regulation by November 2013. Panel (a) displays the correlation between effects o expected approval probability and expected loan monthly payment. Panel (b) displays changes in consumer surplus across borrowers. Panel (c) displays the average, and 25th and 75th percentiles of changes in consumer surplus across borrower risk. Panel (d) displays the average, and 25th and 75th percentiles of changes in consumer surplus, decomposed decomposed between decreased credit access and increased consumer protection.

Figure 1.7: Welfare effects of interest rate regulation



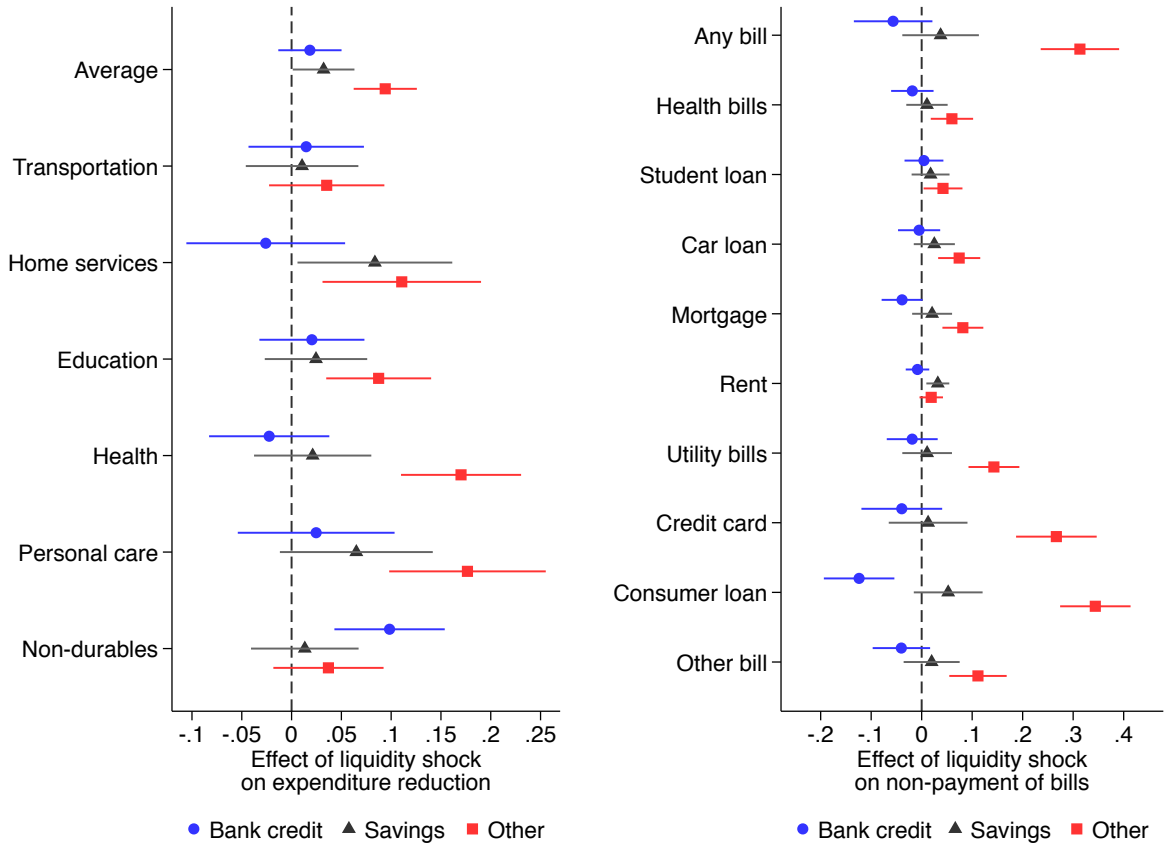
(a) Welfare effects



(b) Expected consumer surplus gains and losses

Notes: These figures display welfare effects of interest rate regulation across borrowers. Both figures compare outcomes for a range of regulation scenarios around that in November 2013 and December 2015. Panel (a) displays the change in average expected consumer surplus and average profit per consumer. Panel (b) displays average changes in expected consumer surplus for consumers that increase and decrease their expected consumer surplus relative to baseline, along with the share of consumers that experience experience consumer surplus gains, losses or none of them. Solid lines indicate averages and dotted lines indicate the 25th and 75th percentiles.

Figure 1.8: Survey evidence for effects of reduced credit access on household outcomes

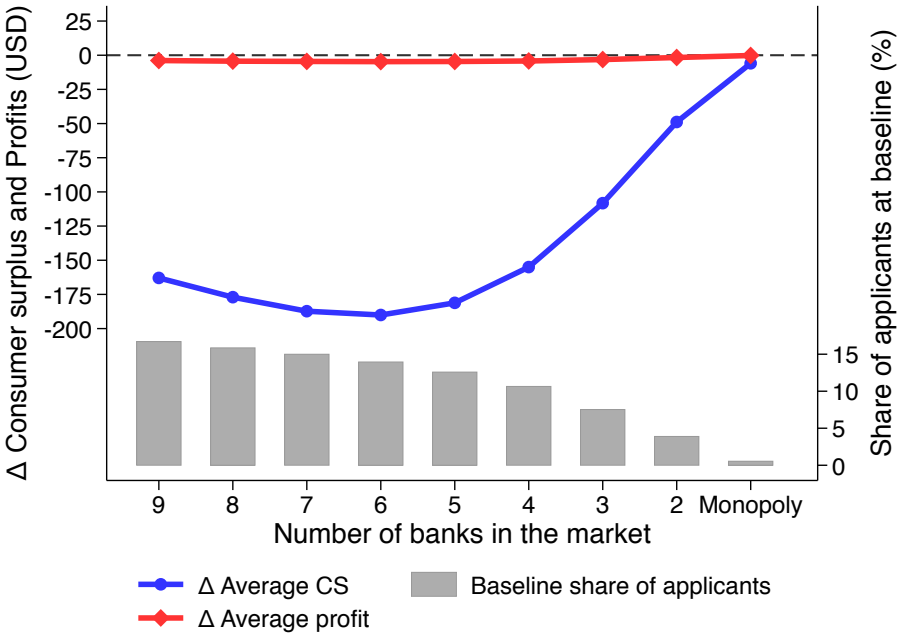


(a) Credit access and household expenses

(b) Credit access and unpaid bills

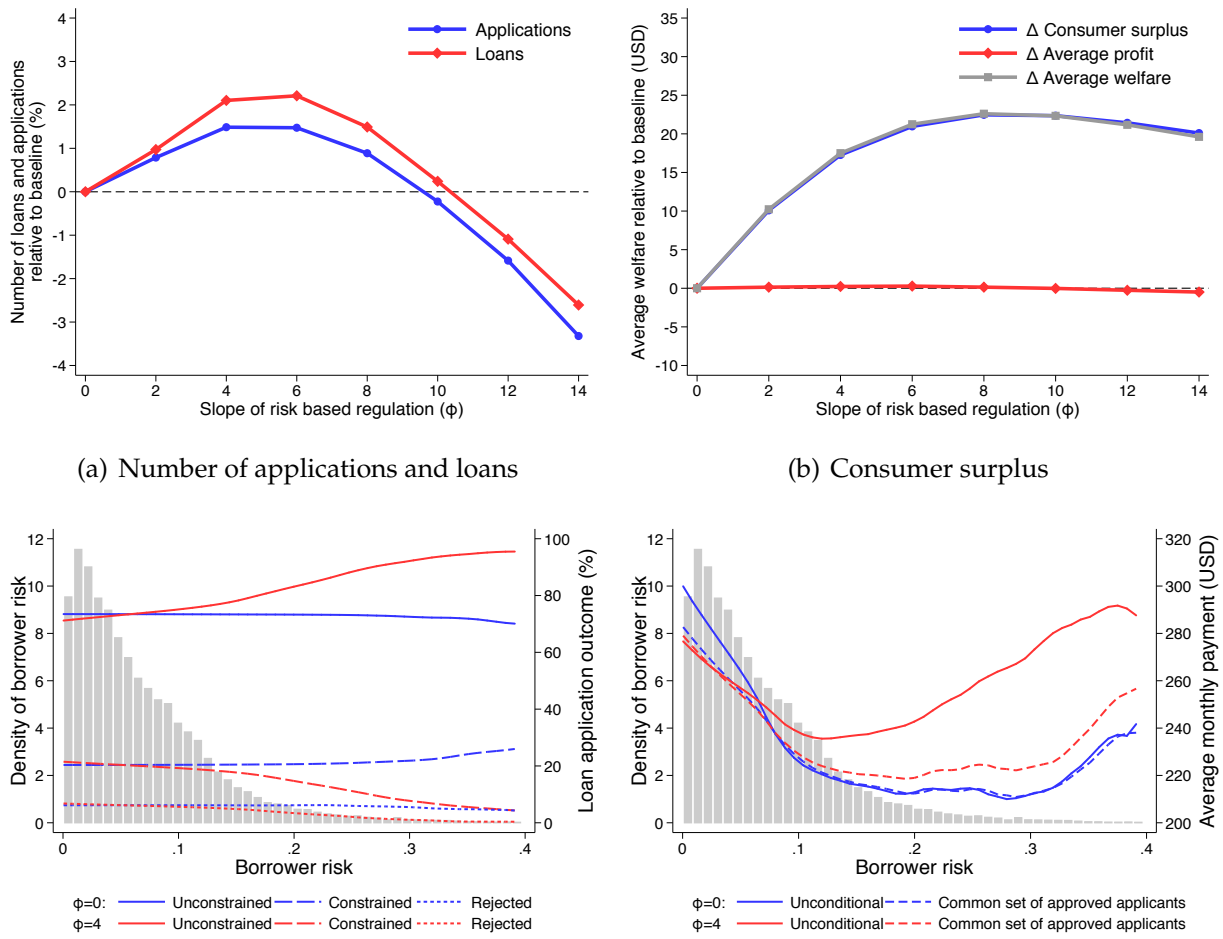
Notes: This figure displays results from regressions of outcomes related to household consumption and financial distress on indicators for whether a households suffered economic hardships and dealt with the using bank credit (blue), liquidating savings or assets (gray), or in some other way, including informal credit and increased labor supply (red). For more detail regarding the specification, see Section 1.6.2. Panel (a) display results for indicators of reduced household expenditure on a variety of items. Panel (b) display results for unpaid bills on a variety of categories. Markers indicate coefficients. Lines indicate 95% confidence intervals.

Figure 1.9: The effects of interest rate regulation under different market structures



Notes: This figure display the effects of interest rate regulation on average consumer surplus (blue) and average profits (red) under different market structures, as measured by the left y-axis. We start with the baseline market structure of 9 banks, and sequentially merge banks until a scenario in which the market is served by a monopoly, as indicated by the x-axis. Each line displays the effect of the full policy on the outcome, for each market structure. Additionally, gray bars display the share of applicants under each market structure, as measured by the right y-axis.

Figure 1.10: Risk-based interest rate caps



(a) Number of applications and loans

(b) Consumer surplus

(c) Application outcomes and risk

(d) Loan prices and risk

Notes: These figures display simulated market outcomes under different levels of risk-based interest rate regulation, as characterized by the parameter ϕ. Baseline outcomes for $\phi = 0$ correspond to simulated equilibrium outcomes for November 2015 under the baseline regulation design. See Section 1.7.2 for details. Panel (a) displays changes in number of loans relative to the baseline level, while Panel (b) displays changes in consumer surplus. Panel (c) displays a local polynomial fit of application outcomes over borrower risk, along with a histogram of borrower risk in the background. Panel (d) displays a local polynomial fit of loan monthly payments over borrower risk, along with a histogram of borrower risk in the background. Solid lines provide results for approved loans under each regulation, whereas dashed lines provide results for the common set of approved loans under both regulation regimes.

Table 1.1: Summary statistics

Variable	N	Mean	SD	p10	p50	p90
A - Loan attributes						
Interest rate	3,362,384	23.17	10.16	10.99	21.24	38.40
Amount	3,362,384	6,705.25	7,007.14	952.14	4,350.17	16,241.59
Term	3,362,384	33.02	16.19	12.17	36.17	50.87
Monthly payment	3,362,384	266.37	323.77	65.95	189.52	522.21
B - Loan performance						
Default during loan first year	3,362,384	0.05	0.21	0.00	0.00	0.00
Default during loan term	3,362,384	0.11	0.31	0.00	0.00	1.00
Amount of charge-off	3,362,384	291.71	1,793.79	0.00	0.00	0.00
Predicted default probability - Income	3,362,384	0.11	0.06	0.04	0.11	0.18
Predicted default probability - History	3,358,842	0.11	0.10	0.02	0.09	0.24
C - Borrower attributes						
Annual income	3,362,384	18,684.65	17,059.19	5,639.61	13,081.43	37,215.05
Age	3,358,842	43.80	13.30	28.00	42.00	63.00
Female	3,362,384	0.40	0.49	0.00	0.00	1.00
Consumer debt	3,362,384	7,021.96	10,514.91	70.72	3,149.40	18,285.16
Consumer debt to income ratio	3,362,384	4.58	5.29	0.07	2.92	10.97
Consumer debt under default	3,362,384	41.00	592.30	0.00	0.00	0.00
Mortgage debt	3,362,384	12,447.09	31,309.96	0.00	0.00	48,179.93
Mortgage debt to income ratio	3,362,384	5.87	13.59	0.00	0.00	24.20
Mortgage debt under default	3,362,384	11.67	664.78	0.00	0.00	0.00
Previously related to bank	3,362,384	0.76	0.43	0.00	1.00	1.00
Previously related to any bank	3,362,384	0.94	0.24	1.00	1.00	1.00
D - Borrowers through the dataset						
Number of loans	1,909,393	1.76	1.22	1.00	1.00	3.00
Amount in loans	1,909,393	11,807.75	14,451.15	1,518.89	6,878.77	28,224.96
Number of banks with loan contracts	1,909,393	1.21	0.48	1.00	1.00	2.00
Previously related banks	1,909,393	3.04	1.55	1.00	3.00	5.00
E - Application events						
Loan amount	3,014,213	7,099.37	7,252.63	1,036.93	4,827.14	16,960.44
Loan term	2,706,289	34.52	15.60	12.63	36.50	53.87
Approved application	3,014,322	0.83	0.38	0.00	1.00	1.00
Rejected application	3,014,322	0.17	0.38	0.00	0.00	1.00
F - Local Market Structure						
Number of banks	1,944	8.00	4.03	2.00	8.00	13.00
Number of branches	1,944	43.11	133.06	1.00	19.00	58.00
Top-1 market share	1,944	0.31	0.13	0.22	0.27	0.46
Top-3 market share	1,944	0.66	0.13	0.52	0.62	0.84
Top-5 market share	1,944	0.83	0.09	0.72	0.81	0.96
HHI	1,944	1,959.38	945.64	1,327.35	1,643.30	2,877.59

Notes: This table displays summary statistics for our datasets. All monetary variables are expressed in U.S. dollars for June 2016. Credit history variables are computed as average over the year previous to each loan.

Table 1.2: Borrower risk, behavior and outcomes

	(1)	(2)	(3)	(4)
	1(Application)	1(Approval)	log(Interest rate)	1(Default)
Risk score	0.0003***	-0.011***	0.037***	0.035***
	(0.0000)	(0.000)	(0.000)	(0.000)
log(Loan size)		0.050***	-0.361***	-0.007***
		(0.000)	(0.000)	(0.000)
log(Loan term)		-0.098***	0.173***	0.071***
		(0.000))	(0.001)	(0.000)
Related to bank		0.099***	-0.008***	-0.042***
		(0.000)	(0.001)	(0.000)
Related to any bank	0.0144***	-0.042***	0.019***	0.022***
	(0.0001)	(0.001)	(0.001)	(0.001)
Mean of dep. var.	0.02	0.82	19.92	0.08
County-Month FE	Y	N	N	N
Bank-County-Month FE	N	Y	Y	Y
Observations	10,696,213	845,046	611,273	611,275
R-squared	0.002	0.138	0.609	0.080
Sample	Population	Applications	Contracts	Contracts

Notes: This Table displays regressions of relevant behaviors and outcomes on borrower risk scores, contract covariates, previous relationships and fixed effects, for the period between January 2013 and November 2013, before the policy change. Column (1) in this table displays results from a regression of an indicator for loan application on loan and borrower covariates. The sample includes a random sample of 10% of potential borrowers in the market. Column (2) does so for an indicator for approval conditional on application, for a sample of all applications in the market. Column (3) does so using interest rates as outcomes, for a sample of all loan contracts for which we observe applications and are approved. Finally, column (4) does so using an indicator for loan default as an outcome, using the same sample an in column (3). Standard errors are displayed in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.3: Effects on interest rates

	(1)	(2)	(3)	(4)	(5)	(6)
	Panel A: Maximum interest rate			Panel B: Average interest rate		
	All	Low-risk	High-risk	All	Low-risk	High-risk
Loans in $0-$2000						
Marginal effect (β)	-1.001***	-0.961***	-0.996***	-0.231***	-0.126***	-0.262***
	(0.010)	(0.024)	(0.012)	(0.032)	(0.021)	(0.041)
Full effect ($\beta \times \Delta\bar{\imath}$)	-16.369***	-15.720***	-16.298***	-3.771***	-2.060***	-4.286***
	(0.157)	(0.390)	(0.200)	(0.526)	(0.345)	(0.666)
Baseline mean	55.145	54.675	55.384	33.023	28.630	35.256
Loans in $2000-$8000						
Marginal effect (β)	-0.785***	-0.660***	-0.803***	-0.073***	-0.033**	-0.107***
	(0.030)	(0.042)	(0.032)	(0.020)	(0.013)	(0.025)
Full effect ($\beta \times \Delta\bar{\imath}$)	-18.307***	-15.405***	-18.744***	-1.714***	-0.776**	-2.496***
	(0.701)	(0.972)	(0.745)	(0.478)	(0.295)	(0.590)
Baseline mean	50.401	48.920	51.634	24.912	21.426	27.813
Observations	2,880	2,880	2,829	2,880	2,880	2,829
R-squared	0.986	0.976	0.984	0.984	0.985	0.975
Product bin FE	Y	Y	Y	Y	Y	Y
Product bin-month of year FE	Y	Y	Y	Y	Y	Y
Month FE	Y	Y	Y	Y	Y	Y

Notes: This table displays results from estimating equation (1.4). For each outcome, the regression is estimated across borrower risk bins and separately by borrower risk bin. All regressions include risk bin-product bin fixed effects and risk bin-month fixed effects. Marginal effects measure the effect of reducing interest rate caps by 1 p.p. Full effects are calculated as the product of the marginal effect of the policy and the magnitude of the policy change once fully implemented for each policy loan-size bracket. All regressions are weighted by the number of loans in the product bin-risk bin before the policy was implemented. Clustered standard errors at the product bin-risk bin level are displayed in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.4: Effects on quantity outcomes

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Panel A: log(Applications)			Panel B: log(Number of loans)			Panel C: log(Credit volume)		
	All	Low-risk	High-risk	All	Low-risk	High-risk	All	Low-risk	High-risk
Loans in $0-$2000									
Marginal effect (β)	-0.003	-0.002	-0.010**	-0.020***	-0.008***	-0.025***	-0.018***	-0.008***	-0.022***
	(0.004)	(0.003)	(0.005)	(0.003)	(0.003)	(0.004)	(0.004)	(0.003)	(0.004)
Full effect ($\beta \times \Delta\bar{t}$)	-0.053	-0.030	-0.164**	-0.323***	-0.129***	-0.414***	-0.291***	-0.135***	-0.362***
	(0.058)	(0.046)	(0.082)	(0.053)	(0.044)	(0.058)	(0.060)	(0.048)	(0.065)
Baseline mean	20,401.500	5,594.100	14,807.400	28,792.909	9,701.909	19,091.000	35,108.197	12,374.705	22,733.492
Loans in $2000-$8000									
Marginal effect (β)	0.000	-0.001	-0.004	-0.005*	-0.002	-0.007**	-0.006*	-0.003	-0.007*
	(0.003)	(0.003)	(0.004)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Full effect ($\beta \times \Delta\bar{t}$)	0.004	-0.017	-0.094	-0.127*	-0.046	-0.171**	-0.133*	-0.066	-0.159*
	(0.071)	(0.068)	(0.099)	(0.073)	(0.061)	(0.082)	(0.073)	(0.063)	(0.080)
Baseline mean	33,312.200	13,582.300	19,729.900	38,822.636	17,633.000	21,189.636	170,025.54	81,855.398	88,170.140
Observations	2,800	2,800	2,713	2,880	2,880	2,880	2,880	2,880	2,880
R-squared	0.994	0.989	0.993	0.990	0.988	0.989	0.985	0.986	0.979
Product bin FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Product bin-month of year FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Month FE	Y	Y	Y	Y	Y	Y	Y	Y	Y

Notes: This table displays results from estimating equation (1.4). For each outcome, the regression is estimated across borrower risk bins and separately by borrower risk bin. All regressions include risk bin-product bin fixed effects and risk bin-month fixed effects. Marginal effects measure the effect of reducing interest rate caps by 1 p.p. Full effects are calculated as the product of the marginal effect of the policy and the magnitude of the policy change once fully implemented for each policy loan-size bracket. Baseline mean for credit volume is reported in thousands. All regressions are weighted by the number of loans in the product bin-risk bin before the policy was implemented. Clustered standard errors at the product bin-risk bin level are displayed in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.5: Effects on risk selection, loan performance and profitability

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Panel A: Risk selection			Panel B: Loan performance			Panel C: Profitability		
	Risk-I	Risk-H	log(Income)	Share of loans under 1Y default			Average expected mark-up		
	All	All	All	All	Low-risk	High-risk	All	Low-risk	High-risk
Loans in $0-$2000									
Marginal effect (β)	-0.067***	-0.043***	0.004***	-0.093***	-0.040***	-0.057**	-0.161***	-0.116***	-0.187***
	(0.003)	(0.007)	(0.001)	(0.018)	(0.010)	(0.027)	(0.027)	(0.020)	(0.032)
Full effect ($\beta \times \Delta\bar{\imath}$)	-1.136***	-0.700***	0.071***	-1.519***	-0.652***	-0.938**	-2.608***	-1.899***	-3.064***
	(0.052)	(0.115)	(0.014)	(0.287)	(0.161)	(0.450)	(0.447)	(0.320)	(0.530)
Baseline mean	6.925	6.616	13.013	6.747	3.353	8.472	22.491	20.773	23.364
Loans in $2000-$8000									
Marginal effect (β)	-0.021***	-0.015***	0.000	-0.038***	-0.004	-0.031*	-0.052***	-0.032***	-0.078***
	(0.004)	(0.005)	(0.001)	(0.011)	(0.006)	(0.018)	(0.016)	(0.012)	(0.020)
Full effect ($\beta \times \Delta\bar{\imath}$)	-0.486***	-0.348***	0.006	-0.879***	-0.101	-0.732*	-1.193***	-0.742***	-1.827***
	(0.101)	(0.113)	(0.014)	(0.259)	(0.143)	(0.411)	(0.380)	(0.271)	(0.471)
Baseline mean	5.782	6.011	16.641	6.026	2.873	8.649	16.013	14.285	17.452
Observations	2,880	2,880	2,880	2,880	2,880	2,829	2,880	2,880	2,829
R-squared	0.982	0.969	0.989	0.915	0.832	0.766	0.983	0.984	0.975
Product bin FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Product bin-month of year FE	Y	Y	Y	Y	Y	Y	Y	Y	Y
Month FE	Y	Y	Y	Y	Y	Y	Y	Y	Y

Notes: This table displays results from estimating equation (1.4). For each outcome, the regression is estimated across borrower risk bins and separately by borrower risk bin. All regressions include risk bin-product bin fixed effects and risk bin-month fixed effects. Marginal effects measure the effect of reducing interest rate caps by 1 p.p. Full effects are calculated as the product of the marginal effect of the policy and the magnitude of the policy change once fully implemented for each policy loan-size bracket. Predicted risk is computed as described in Section 1.2.2 and measured in a 0-100 scale. Income is measured in thousands of U.S. dollars. Share of loans under default in first year is computed in a 0-100 scale. All regressions are weighted by the number of loans in the product bin-risk bin before the policy was implemented. Clustered standard errors at the product bin-risk bin level are displayed in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.6: Model estimates

	(1)	(2)	(3)	(4)
Panel A - Demand side	Application		Repayment	
	Estimate	S.E.	Estimate	S.E.
Drivers of application (δ) and repayment (α)				
Constant	3.500***	(0.076)	0.049	(0.126)
Risk score	1.390***	(0.053)	-2.008***	(0.098)
Female	-0.124***	(0.006)	0.026**	(0.010)
Age $\in$ [33,55)	-0.032***	(0.006)	0.031***	(0.010)
Age $\in$ [55,+)	-0.309***	(0.009)	0.079***	(0.016)
log(Annual income)	0.016***	(0.005)	0.110***	(0.011)
Debt to income ratio	0.442***	(0.040)	0.024	(0.065)
Default to debt ratio	-0.838***	(0.039)	-0.006	(0.019)
Loan term	0.045***	(0.003)	-0.082***	(0.005)
Loan amount	0.009***	(0.001)	0.003***	(0.001)
Monthly payment, low-risk	0.479***	(0.016)		
Monthly payment, high-risk	0.304***	(0.017)		
Application cost (κ)				
Constant	4.607***	(0.099)		
Number of branches	-0.000***	(0.000)		
Previously related banks	-0.196***	(0.003)		
Application and repayment shocks				
Standard deviation (σ_A, σ_S)	1.000	—	0.525***	(0.008)
Correlation (ρ)	-0.010	(0.046)		
Month FEs	Y		Y	
Market FEs	Y		Y	
Panel B - Banks' costs	Short term		Long term	
	Estimate	S.E.	Estimate	S.E.
Range of bank fixed effects (τ)	[-0.038***,-0.024***]		[-0.038***,-0.025***]	
Previously related to bank (γ)	0.016***	(0.000)	0.016***	(0.000)
Bank-borrower shock (σ_ω)	0.009***	(0.000)	0.010***	(0.000)

Notes: Panel A in this table displays estimates from the demand side of the model. Columns (1) and (2) display estimates and standard errors for parameters in the application equation. Columns (3) and (4) display estimates and standard errors for parameters in the repayment equation. The specifications of both the application and repayment equations include month and market fixed effects. Panel B displays estimates from the supply side of the model. Standard errors are displayed in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table 1.7: Simulated effects of interest rate regulation

	(1)	(2)	(3)
Outcome	Baseline Nov/2013	Mid effect Nov/2014	Full effect Nov/2015
Apply for loans (p.p)	19.92	-2.20	-4.11
Unconstrained|Apply (p.p)	90.07	-9.50	-17.35
Constrained|Apply (p.p)	7.58	7.74	13.57
Rejected|Apply (p.p)	2.34	1.75	3.78
Number of loans (%)	-	-12.69	-23.73
Monthly payment ($)	251.73	-5.29	3.98
Monthly payment on approved under full policy ($)	258.30	-2.00	-2.59
Mark-up (p.p)	29.11	0.73	2.00
Mark-up on approved under full policy (p.p)	31.80	-0.37	-0.67
Default probability (p.p)	7.26	-0.10	-0.27
Consumer surplus ($)	-	-44.20	-82.47
Monthly profit ($)	73.46	-0.19	6.13
Monthly profit on approved under full policy ($)	82.15	-1.97	-2.55
Average monthly profit ($)	13.26	-1.77	-2.40
Average welfare ($)	-	-46.15	-84.84

Notes: This Table displays results for simulated policy effects of moving from the baseline interest rate regulation in November 2013 to interest rate regulation in November 2014 and November 2015, when the policy change was fully in place. Mid and full effects are measured relative to baseline levels. Column (1) displays simulated equilibrium outcomes for regulation at November 2013. Column (2) displays simulated changes in equilibrium outcomes under regulation present in November 2014 and baseline regulation, while column (3) does the same for regulation by the end of the policy change in November 2015 and base line regulation.

CHAPTER 2
QUALITY REGULATION AND COMPETITION: EVIDENCE FROM PHARMACEUTICAL MARKETS

2.1 Introduction

Increased penetration of generic drugs has been one of the major sources of cost savings in the U.S. health care in recent decades (Grabowski et al., 2006). A variety of policies incentivizing generic adoption, together with the expiration of several patents, led the retail market share of generics in the U.S. to rise from 34% in 1994 to 87% in 2015 (Berndt et al., 2017). However, generic penetration remains a first-order policy concern in low- and middle-income countries as a means to increase the access to affordable medicines (UN, 2010; Pinto et al., 2018).

Quality regulation is considered a key precondition for the success of policies to foster penetration of generic drugs and increase price competition (WHO, 2000). Weak quality regulation undermines physician and patient trust in generics, and may limit price competition due to differences in perceived quality. Governments introducing quality regulation in pharmaceutical markets expect to ensure drug quality and improve the perception of generic alternatives, which increases the propensity to prescribe and choose generics, leading to increased competition. However, these regulations may also induce the exit of affordable and yet high-quality drugs due to costly compliance. Drug exit might in turn reduce price competition, overturning positive effects of reduced (perceived) quality differences between innovators and generics brought on by the regulation. Therefore, the equilibrium market outcomes of quality regulation policies are the result of an interplay between reduced vertical differentiation and changes in market structure due to costly compliance.[1]

1. In models of vertical differentiation, *differences* in quality are a source of market power (see, e.g., Gabszewicz and Thisse, 1979), such that a smaller difference is expected to lead to more intense price competition (conditional on market structure). Price differences between innovator and generic drugs are typically attributed to market segmentation (see, e.g., Frank and Salkever, 1992), consistent with vertical differentiation models where consumers with high willingness-to-pay for perceived quality choose a higher priced innovator drug.

In this paper, we study the equilibrium effects of quality regulation policies in pharmaceutical markets by exploiting the roll-out of a requirement to certify bioequivalence for generics in Chile. To the best of our knowledge, this is the first paper to measure the overall market effects of introducing bioequivalence requirements; which is the most common policy instrument for generic drugs quality assurance. At the onset of this policy, unbranded generics accounted for less than 30% of total retail sales on average, even though they were on average 6 and 10 times cheaper than branded generics and innovator drugs respectively.[2][3] The primary objectives of the reform were to increase the perceived quality of generics and enhance price competition.[4]. Bioequivalence is a central requirement in the process of approving generics in developed countries and, increasingly so, in developing countries. An innovator drug can be substituted by a bioequivalent generic with the full expectation that the generic has the same clinical effect and safety profile.[5] After the reform, generics without bioequivalence certification were no longer allowed to be sold in Chile.

We estimate the effects of quality regulation on market structure, drug prices, market shares and drug sales. For this purpose, we combine administrative data on entry and exit from the national drug registry of Chile with price and sales data from IMS Health for 2010–2017. Our empirical strategy exploits the staggered implementation of the reform along with features of its enforcement, to compare outcomes across and within markets (molecules)

2. *Innovator drugs* are the first ones containing its specific active ingredient to receive approval for use, and are often referred to as originator drugs. *Generics* are drugs with the same active ingredient as an innovator drug and can be marketed after the expiration of the patent of the innovator drug. *Unbranded generics* are marketed by molecule name and compete on prices, whereas *branded generics* are marketed under a trade name, typically advertise, and compete on brand (see, e.g., Danzon and Furukawa, 2008). In the U.S. and Europe, branded generics are predominantly marketed by (subsidiaries of) innovating pharmaceutical firms (see Grabowski and Vernon, 1992, p. 346), whereas in many Latin American and developing countries, branded generics are produced and marketed by generic manufacturers.

3. Reported market shares for generics and price premiums are based on our own calculations from IMS Health data using the sample employed in the main analysis of the paper. See Section 2.4 for further details.

4. These objectives were explicitly stated by government officials, as discussed in Section 2.2.2. On the other hand, to the best of our knowledge, there was no public discussion justifying this regulation on the grounds of concerns regarding the poor quality of generics

5. More precisely, a generic drug is bioequivalent to its reference innovator counterpart when its rate and extent of absorption are not significantly different from those of its reference drug when administered under the same conditions (Davit et al., 2013). Bioequivalence became the primary means for generic drugs approval in the U.S. after the passage of the Hatch-Waxman Act in 1984, which allowed generics seeking marketing approval to submit proof of bioequivalence with the reference drugs in lieu of preclinical (animal) and clinical (human) testing on safety and efficacy.

differently exposed to the regulation. This strategy delivers reduced form estimates of the effects of the policy on equilibrium market outcomes. We interpret our results using a model where innovator and generic drugs compete in prices in an environment where consumers only imperfectly observe the quality of generic drugs.

We start by showing that stronger quality regulation induced laboratories to obtain bioequivalence certification for their drugs. We find that drugs were 12 times more likely to have bioequivalence certification after requirements were implemented. Moreover, we show that certification was more frequent in more profitable and less competitive markets.

Stronger quality regulation had large effects on market structure, prices, market shares and sales. First, we estimate that stronger quality regulation affected market structure by decreasing the number of drugs by 25%. Second, we estimate a 10% increase in average paid drug prices, most of which was due to drug-specific price increases rather than changes in market shares or changes in the composition of drugs driven by entry and exit. Third, we show that the policy shifted sales from branded generics to innovator drugs, whereas total sales volume decreased by 20%. Most of these effects are concentrated in small markets.In small markets, the number of drugs decreased by 36%, and average paid prices increased by 26%. Furthermore, the market share of innovator drugs in small markets increased by 8 percentage points (p.p.) at the expense of generics, whereas total sales volume decreased by 30%. In contrast, for large markets we estimate a 15% decrease in the number of drugs, but no significant effect on average paid prices or the market share of generics.

In principle, these adverse effects on market outcomes could have been compensated by improvements in drug quality. However, we find no evidence suggestive of such improvements. We leverage administrative data on hospital admissions associated to drug adverse effects and drug recalls as measures of quality. We do not find evidence of a significance decrease in these outcomes following the reform, neither overall nor in small markets. The lack of effects on drug quality suggests that the negative welfare effects from changes in market structure and higher prices were not compensated by higher underlying drug quality.

Overall, our results suggest that any direct effect of increased price competition due to decreased scope for quality differentiation was overturned by indirect adverse effects

to competition due to drug exit. The heterogeneity of these effects across market size reinforces this interpretation, and suggests that fixed compliance costs played a significant role in driving these outcomes.

We complement our main analysis with a survey of a sample of pharmacy customers in Chile. Our survey suggests that a variety of demand-side frictions may continue to undermine the ability of the regulation to generate its intended effects. In particular, we find that our interviewees: (i) lack an appropriate understanding of what bioequivalence entails and continue to place substantial perceived quality premiums on innovator drugs, even several years after the policy change; (ii) underestimate price differences between innovators, branded generics and unbranded generics; and (iii) frequently declare that their physicians prescribe by the brand name. Although these results come from a small sample of consumers, they are suggestive of barriers that may reduce incentives for laboratories manufacturing generics to enter or remain in the market in the presence of fixed costs of complying with the regulation. The lessons from our survey suggest that policies complementary to quality regulation may be necessary to increase generic penetration and competition in this context, such as consumer information policies or the regulation of prescription behavior.

This paper is related to a large literature analyzing the effect of regulatory policies on pharmaceutical markets. Much of this research focuses on the equilibrium implications of price regulation for pharmaceutical markets in developed countries (see, e.g., Danzon and Chao, 2000; Dubois and Lasio, 2018; Dubois and Sæthre, 2018; Lakdawalla, 2018), whereas the equilibrium effects of quality regulation have yet to be studied. We contribute to this literature by analyzing the equilibrium effects of one of the most common forms of quality regulation in pharmaceutical markets.Directly related to our setting, Balmaceda et al. (2015) provide an early exploration of the reform in Chile, estimating its short-term effects on drug prices. We implement a broader analysis by evaluating effects on market structure, sales and quality outcomes after the full implementation of the policy.[6]

6. This paper differs from Balmaceda et al. (2015) along several other dimensions. First, their sample covers until March 2014, when 75% of all bioequivalence approvals to date and several relevant policy events had not yet come into effect. Second, our empirical strategy relies on exploiting variation in the roll-out of the policy across and within markets, instead of assuming parallel-trends between markets affected and

Moreover, we contribute to a literature that studies the participation of generics in pharmaceutical markets. First, our study is related to previous research on the entry of generics after patent expiration in the U.S., which has highlighted the importance of market variables for entry decisions (Scott Morton, 1999, 2000). We contribute to this literature by studying a different regulatory context where generic drugs that are already in the market face the decision of whether to stay in the market under stronger quality regulation, and by focusing on a middle-income market. Our results highlight that quality regulation indeed affect drug exit decisions. Second, we build on a large empirical literature analyzing competition between innovator and generic drugs, which has primarily focused on analyzing the market responses to the entry of generics when innovator drugs go off-patent (see Caves et al. 1991; Grabowski and Vernon 1992; Frank and Salkever 1997; Grabowski et al. 2006; Knittel and Huckfeldt 2012; Branstetter et al. 2016, among others). Our paper relates to this literature by providing evidence from a regulatory change that induces generic exit, coupled with potential changes in perceived generic quality. Finally, we also contribute to a better understanding of the sources of aversion to generics that sustain brand premiums (Colgan et al., 2015; Bairoliya et al., 2017), by studying the effects of minimum quality standards that attempt to reduce information asymmetries which could bias consumers against generics.

The remainder of the paper is organized as follows: Section 2.2 describes the Chilean pharmaceutical market and bioequivalence regulation; Section 2.3 proposes a model that guides our analysis of the effects of quality regulation; Section 2.4 describes the data used in our analysis; Section 2.5 analyzes the extent of bioequivalence certification, and entry and exit choices at the drug level; Section 2.6 provides our main estimates of the effects on market structure and market outcomes; Section 2.7 provides evidence from survey data that sheds light on potential mechanisms behind our findings; and Section 3.6 concludes with a discussion of our findings and policy implications.

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unaffected by the policy in a simpler differences-in-differences analysis. Third, we develop a conceptual framework that guides the interpretation of our results in the context of a model of competition with vertical differentiation across drugs.

2.2 Pharmaceutical Market and Quality Regulation in Chile

2.2.1 Institutional Framework

Spending and Coverage. Chileans spend 0.9% of their GDP on pharmaceuticals, which is lower than the OECD average of 1.5% (OECD, 2013). However, expenditure on both overall health care and pharmaceuticals has grown steadily over recent years and pharmaceutical spending accounts for around 40% of all out-of-pocket health expenditures in the country (Benítez et al., 2018).

One third of Chileans pay for their prescription drugs fully out-of-pocket (Minsal, 2013). The level of financial coverage for prescription drugs depends both on whether the individual opts to enroll in the public insurance system (*Fondo Nacional de Salud*, FONASA) or in a private insurance plan, and on the specific disease to be treated.[7] FONASA enrollees who opt to receive health care within the network of public providers face copayment rates that depend on socioeconomic variables, although outpatient claims are free of charge, including prescription drugs.[8] FONASA enrollees who instead opt for receiving care in private hospitals pay procedure-specific prices negotiated between FONASA and each provider.[9] Insurance plans in the private system do not generally include coverage for prescription drugs.

Pharmaceutical Market. The institution in charge of oversight of this market is the Public Health Institute (*Instituto de Salud Pública*, ISP). Laboratories present applications to ISP to obtain marketing licenses for distribution in Chile. These marketing licenses must be renewed every five years. ISP is also responsible for drug quality assurance and has overseen the roll-out of the bioequivalence reform.

Two additional features of the retail pharmaceutical market in Chile may influence the workings of the bioequivalence reform. First, as opposed to the U.S., direct-to-consumer

7. FONASA covers around 80% of the population. Most of the remaining 20% is covered by the private market. For a more detailed description of the health insurance market in Chile, see Duarte (2012b).

8. The total level of copayment is capped for a set of 80 prioritized diseases.

9. Enrollees receive partial coverage of claims in these cases, with the exception of the pharmacological treatment of a list of 11 high-cost diseases that are fully covered.

advertisement of prescription drugs is forbidden, which could, in principle, make consumers more price sensitive because expensive branded drugs cannot use advertising to signal quality and boost demand. Second, the retail pharmacy sector in Chile is highly concentrated, which might affect the degree of supply-side reaction to the bioequivalence requirements. Three large pharmacy chains account for more than 90% of the market, with a fraction of their sales corresponding to private-label drugs. The remainder of the market is comprised of several small chains without national presence.[10]

Prescriptions and Generic Substitution. Prescription behavior of physicians and the ability of pharmacists to offer alternative versions of prescribed drugs to consumers are important mediators of consumer choice in the pharmaceutical market. In Chile, pharmacists may only offer generic substitution for prescriptions that specify the generic name and when a bioequivalent substitute is available. Despite recent policy efforts towards constraining discretion in prescriptions, physicians still often prescribe by brand name only, which limits substitution towards generics in practice.[11]

2.2.2 Bioequivalence in the Chilean Pharmaceutical Market

Bioequivalence is established to demonstrate therapeutic equivalence between a generic drug and the corresponding reference drug, that is often the innovator drug. In particular, two drugs are bioequivalent when the rate and extent of absorption of the tested drug and the reference drug do not show significant differences, when administered at the same molar dose of the therapeutic ingredient under similar experimental conditions (Davit et al., 2013). Bioequivalent drugs can be substituted with the full expectation that the generic drug

10. The three large chains were involved in a collusion scandal in early 2008, almost two years before our study period. See Alé (2017) for a detail discussion of the collusion case and for a more detailed description of the retail pharmacy market in Chile.

11. In February 2014, Law 20,724 was passed with the objective of requiring physicians to include the generic name in the prescription and allow for substitution towards bioequivalent generics if requested by the patient. However, different industry actors concede that the requirement has not been enforced in practice, and that physicians have continued to prescribe branded drugs. Our survey evidence in Section 2.7 is consistent with this view. The lack of enforcement of the original requirement is well known, and has motivated a new pharmaceutical law that is currently under discussion in the Congress. See, e.g., La Tercera (2015).

yields the same clinical effect and safety profile as the reference drug (FDA, 2017). Therefore, bioequivalence allows bridging pre-clinical and clinical data associated with the reference drug to the generic drug. Bioequivalence is a standard requirement for commercialization of generic drugs in most high-income countries (Balmaceda et al., 2015). Moreover, many OECD countries either allow, encourage or require substitution of innovators for cheaper bioequivalent drugs (OECD, 2000). Although bioequivalence requirements were originally implemented in the developed world to foster generic entry, they have been recently adopted by developing countries as the primary tool for testing the effectiveness of the drugs allowed in their markets (Balmaceda et al., 2015). Prior to bioequivalence, quality standards in Chile required generic manufacturers to follow guidelines of the International Pharmacopeia books (WHO, 2017), which ensured minimum production standards and safety but did not ensure therapeutic efficiency. The bioequivalence requirement was introduced as an addition to the previous quality standards.

The stated goals of the bioequivalence regulation in Chile were to increase competition in the pharmaceutical market and reduce prices.[12][13] For instance, in the early years of the reform, the Head of the National Drug Agency (*Agencia Nacional de Medicamentos*, ANAMED) stated in an article published in La Tercera (2012):

> "We have no doubts that drug prices will decrease, because the population will have access to a wider and more competitive drug market"
>
> *Elizabeth Armstrong, Head of National Drug Agency*
> *May, 2012*

12. To the best of our knowledge, there was no public discussion justifying this regulation on the grounds of concerns regarding the poor quality of generics. Arguably, the bioequivalence regulation was also meant as the first step in a series of reforms intended to increase substitution towards generics, as evidenced by the current discussions in Congress discussed in footnote 11.

13. In a context where there is underlying heterogeneity in quality that is unobservable to consumers, it could be argued that voluntary quality disclosure might take place and lead to unravelling, by which consumers would become aware of quality differences and low quality drugs might exit the market (Dranove and Jin, 2010). However, this prediction does not hold in a setting in which disclosure is too costly (Jovanovic, 1982). In the setting we study, generic drugs were not aware of whether they were bioequivalent prior to the costly verification. Moreover, consumers were likely not familiar with the concept of bioequivalence before this regulation was implemented, which would limit the returns to disclosure. These two factors may jointly explain the lack of private quality disclosure.

The first list of active ingredients subject to bioequivalence was published in 2005 by the Chilean Ministry of Health (*Ministerio de Salud*, MINSAL). This list consisted of active ingredients deemed to be potentially prescribed for chronic conditions included in a major reform to the public health insurance system called AUGE (Bitrán et al., 2010a). However, it was not until 2009 that the regulator established the technical norms for bioequivalence testing (Balmaceda et al., 2015). Bioequivalence requirements were phased in since then, with 167 molecules covered by this regulation as of March 2018. All new drugs containing the molecule listed in each law decree were mandated to certify bioequivalence before obtaining a marketing license.[14] Each decree specified the deadline for bioequivalence testing among incumbent drugs already registered. In practice, however, enforcement of the requirements occurred mostly by the time of license renewal, when ISP often denied renewal to drugs without bioequivalence approval (Vasallo, 2010). Drugs with bioequivalence certification carry a distinctive label intended to serve an as indication of bioequivalence status for the consumer.[15] We show an example of this label in Figure B.1.

The costs of bioequivalence testing are in the range of $50,000 to $240,000 U.S dollars per drug, and are covered by the manufacturer.[16] To put this number into context, the median drug in our data had a yearly revenue of $103,600 at the onset of the reform in 2010. Moreover, 35% and 71% of drugs had yearly revenues lower than $50,000 and $250,000 respectively. Although these figures only account for revenue in the retail market, they suggest that the financial burden imposed by bioequivalence compliance costs was not negligible for several drugs.[17]

In most cases, the original deadlines to provide proof of bioequivalence were extended—through a series of subsequent decrees—due to the slow uptake and capacity constraints in

14. Bioequivalence requirements were only imposed for orally administered drugs, i.e. the requirements do not apply to topical medications, vaccines, or any other type of drugs that are not orally administered.

15. In practice, one could argue that the label in itself has an effect on demand through quality disclosure (see Dranove and Jin (2010) for a review of the theoretical and empirical literature on quality disclosure). However, drugs without bioequivalence approval must exit the market, so that, if consumers are aware of the policy, the label does not carry any additional informational content in our setting.

16. This range for certification costs is based on reports that include statements from market participants about certification costs (La Tercera, 2012; CIPER, 2015).

17. All monetary values in the paper are inflation-adjusted to December 2013. For reference, the exchange rate at that point was of $529 CLP per U.S. dollar.

the laboratories performing the tests. Among the molecules with bioequivalence requirement, there are nine unique combinations of the relevant policy dates, namely the date of the first decree, date of extensions (if applicable), and corresponding deadlines established in the first decree and the extensions. Table 2.1-A shows the dates of the first decree (the first date when a bioequivalence requirement was announced), the last decree (the last date when an extension to the original deadline was announced) and the corresponding deadlines for each of these nine groups, as well as the number of molecules included in each group.[18] For example, Group 1 includes four molecules that had their first decree announced in January 2011, which established a deadline for February 2012. However, the original deadline was extended, and its final decree was announced in June 2013, with a deadline for December 2013. Variation in the timing of bioequivalence regulation is summarized in Figure A.17-a. We exploit this variation for estimation of policy effects later in the paper.

In practice, bioequivalence certification is provided after the manufacturer presents satisfactory studies. Generally, bioequivalence is determined through *in-vivo* clinical studies for one specific presentation of a given drug, though (under certain conditions) only *in vitro* studies are required for different dosages of the same drug. Bioequivalence certification of imported drugs is normally validated in Chile if the drug has already obtained it in countries considered to have high certification standards (e.g., Canada, USA, the European Union, New Zealand, among others). Although the certification is awarded *ad eternum* for a given formula and production technology, any change in one of these dimensions requires a new certification.

2.3 Conceptual Framework

We introduce an equilibrium model of pharmaceutical markets to shed light on the mechanisms through which quality regulation affects market outcomes. Our model considers

18. We exclude from this classification all molecules that received their first decree before 2010, because they are excluded from the sample we use in our main analysis due to data limitations (our sample from IMS Health, covering sales and revenues, starts in 2010). Similarly, we exclude molecules that were not affected at all by any bioequivalence requirement.

several important features of the market, including: (i) vertical differentiation, where generics and innovator drugs can be perceived to be of different quality either due to fundamental quality issues (e.g., lack of bioequivalence or presence of side-effects), or due to product valuation; (ii) heterogeneity in consumers' willingness-to-pay for (perceived) quality; (iii) asymmetric information on quality of generics, where consumers (and physicians) cannot observe the quality of generics; and (iv) fixed costs of operating in the market and of bioequivalence certification, which leads to entry and exit considerations among producers.

The importance of vertical differentiation follows from the general observation that innovator and generic drug prices often differ substantially (see e.g., Frank and Salkever 1997; Danzon and Furukawa 2008), which is consistent with the type of segmentation that arises in this class of models. Asymmetric information on generic quality is introduced to allow for the possibility that the perceived quality of generics is inefficiently low, such that quality regulation potentially increases both perceived quality and competition. Fixed costs allow market structure to be endogenously determined in the model. In particular, when quality regulation imposes substantial compliance costs, as in the case we study, it may lead to an unintended decrease in the number of generic drugs by deterring entry or inducing exit.

The way we model asymmetric information in this market is similar to Leland (1979), from which we differ by including vertical differentiation. Pure vertical differentiation, as introduced by Gabszewicz and Thisse (1979), has been considered by previous theoretical work on minimum quality standards,[19] though mostly in settings with perfect information on quality and exogenous market structure.[20] The novelty of our model comes from combining asymmetric information and vertical differentiation in a setting where market structure is endogenously determined.

19. See, e.g., Ronnen (1991); Crampes and Hollander (1995); Scarpa (1998).

20. An exception is Garella and Petrakis (2008), who consider imperfect information in strategic games with endogenous quality, allowing for both horizontal and vertical differentiation. Our model differs from theirs on how we model asymmetric information on quality, on which we are closer to Leland (1979), and by allowing for endogenous market structure.

2.3.1 Model

Environment. The supply side of the market consists of an innovator drug I and N_G generic drugs indexed by g that may or may not participate in the market. Each drug has an exogenous quality level ψ. The quality of the innovator drug I is known to consumers and given by ψ_I and the unobservable quality of generic drug g is $\psi_g \leq \psi_I$. Generic quality has a (known) cumulative distribution F_ψ, so that if all generics with quality between ψ_a and ψ_b participate, the number of generic firms is given by $n_G = N_G \left(F_\psi(\psi_b) - F_\psi(\psi_a)\right)$. Drugs decide to participate in the market or not and compete in prices in a Bertrand game in which all drugs set prices simultaneously.

There is a continuum of consumers in the market, with preferences over drug quality and prices, but unable to distinguish the quality of each generic drug.[21] Instead, they treat all generic drugs as being of the average quality among market participants, denoted by $\overline{\psi}$.[22] The indirect utility that consumer i obtains from purchasing either the innovator drug I or a generic drugs g is:

$$\begin{aligned} u_{iI} &= \tau_i \psi_I - p_I + \varepsilon_{iI} \\ u_{ig} &= \tau_i \overline{\psi} - p_g + \varepsilon_{ig} \quad \forall g, \end{aligned}$$

where τ_i is the preference for quality of consumer i, and ε_{iI} and ε_{ig} are idiosyncratic preference shocks. The idiosyncratic utility terms can be interpreted as an additional, symmetric differentiation between producers, allowing prices above marginal cost among generics to be sustained in a Bertrand-Nash equilibrium.[23] Heterogeneity in preference for

21. We assume that quality is not revealed by consumption. Lack of learning about quality may be reasonable in markets where differences in medical effects or side-effects are hard to detect or realized over a longer horizon, such that experience with any given generic can be assumed to reveal no information, neither for consumers nor physicians.

22. This is similar to Leland (1979) and follows, e.g., from an assumption that any credible quality signal is too costly for generic producers. We note that the decision to market drugs under brand names (branded generics) may be a strategy to reduce information asymmetry in the market we study, although we do not consider this aspect in our model.

23. Our formulation—with price entering linearly with a coefficient of one—implies that indirect utility is measured in terms of willingness-to-pay. Allowing for a utility scaling of price (αp_j) does not change the qualitative implications of the model (results available from the authors upon request).

quality, τ, provides a role for vertical differentiation: whenever $\overline{\psi} < \psi_I$, a consumer with high τ would be more likely to purchase the innovator drug at a higher price, whereas a consumer with low τ would be more likely to buy a lower priced generic. With such sorting, quality differences reduce price competition (Shaked and Sutton, 1982). Finally, a consumer may decide not to purchase any of the drugs in the market, and instead choose an outside option that yields indirect utility $u_{i0} = \varepsilon_{i0}$.

Profits of innovator and generic drugs are given by

$$\begin{aligned}\pi_I &= Ms_I p_I - C_I \\ \pi_g &= Ms_g p_g - C_G(\psi_g) - \kappa_{QC} \quad \forall g,\end{aligned}$$

where M is market size, C_I is the fixed cost of the innovator drug, $C_G(\cdot)$ is a quality-dependent fixed cost faced by generic drugs; and κ_{QC} is an additional sunk cost associated with quality certification. For simplicity, we set marginal cost to zero for all producers.[24] We assume that fixed manufacturing costs are continuous and increasing in quality ($C'_G(\cdot) > 0$). Due to asymmetric information on generic quality, this leads to adverse selection, because incentives to enter the market are higher for lower quality drugs.

Equilibrium with quality certification. Given that generic drugs are symmetric up to a quality-specific fixed cost, we focus on a symmetric equilibrium in which all generic producers set the same price p_G and obtain the same market share, denoted by s_G. In this equilibrium, generic producers choose to participate in the market as long as:

$$\pi_g \geq 0 \iff Ms_G p_G \geq C_G(\psi_g) + \kappa_{QC}$$

which determines the set of active generic producers. Since all generics obtain the same variable profits and quality-dependent fixed costs are increasing, it follows that the marginal generic entrant is of (weakly) higher quality than inframarginal entrants.

Quality certification takes the form of a minimum quality standard denoted by $\underline{\psi}$.

24. For most oral solids (tablets), this is likely a good approximation (see, e.g., Berndt and Newhouse, 2012). Otherwise, allowing for positive and asymmetric marginal costs is straightforward in our model.

Conditional on $\underline{\psi}$, there is a one-to-one relation between the number of generics in the market and the quality of the marginal entrant, $\hat{\psi}$, given by $n_G = N_G\left(F_\psi(\hat{\psi}) - F_\psi(\underline{\psi})\right)$. Thus, n_G is the number of generics with quality between the minimum allowable to the highest that still achieves a non-negative profit. Then, the average generic quality $\overline{\psi}$ is equal to the expected quality among the n_G active generic producers, that is, the ones with quality between $\underline{\psi}$ and $\hat{\psi}$.[25]

The market equilibrium will be determined by the conditions for a Bertrand Nash equilibrium in the prices of the generics and innovator, together with the zero-profit entry condition for the highest quality generic entrant. That is, the equilibrium is imperfectly competitive, with positive variable profits which covers the fixed costs for the marginal (i.e., highest quality active) generic entrant. The difference from standard entry models is selection: additional entry by generics will have a positive effect on the expected quality of all generics, possibly leading to higher generic prices and/or market shares. This could happen in cases where the perceived quality of generics is very low.[26]

2.3.2 Comparative Statics: The Equilibrium Effects of Quality Regulation

In this section, we discuss the equilibrium effects of stronger quality regulation implied by our model. Consider an increase in the minimum quality standard from $\underline{\psi}_0$ to $\underline{\psi}_1$, with $\underline{\psi}_0 < \underline{\psi}_1$. Stronger quality regulation has a direct effect on the willingness-to-pay for generics. Keeping the set of active producers fixed, perceived quality of generics increases because consumers know that these producers have quality $\psi_g \geq \underline{\psi}_1$. Decreased vertical differentiation resulting from this increase in perceived quality leads to more intense price competition with the innovator. Thus, keeping the set of generics fixed, the price of the innovator decreases. Prices of generics might increase or decrease, because the increased willingness-to-pay for the higher perceived quality is counteracted by the higher intensity

25. If generic quality was uniformly distributed, the expected quality would simply be the midpoint $(\hat{\psi} + \underline{\psi})/2$.

26. Note that there is an incentive for generics to keep quality lower than the innovator to soften price competition, such that we have in mind a situation where perceived quality is lower than a quality level that would be optimal from the generic firms' view (i.e., trading off higher willingness to pay of consumers and less differentiation from the innovator).

of price competition with the innovator.

However, stronger quality regulation also has effects on market structure. First, there is a direct effect through the exit of all $N_G(F_\psi(\underline{\psi}_1) - F_\psi(\underline{\psi}_0))$ producers with quality $\psi_g < \underline{\psi}_1$ that were previously in the market. The exit of these drugs decreases the intensity of price competition, particularly among generics. In addition, fewer generic competitors leads to higher demand for the remaining generic drugs and for the innovator. Second, an increase in perceived quality—together with higher demand for any single generic drug—may induce $N_G(F_\psi(\hat{\psi}_1) - F_\psi(\hat{\psi}_0))$ higher quality generics to enter the market at the margin, further increasing the perceived quality of generics and the intensity of price competition with the innovator. Overall, stronger quality regulation increases the quality of generics in the market and has an uncertain effect on prices that depends on the changes in vertical differentiation and price competition.[27]

Our model provides a framework to analyze the effects of quality regulation and shows that a variety of outcomes are possible. Depending on the primitives of the market, stronger quality regulation may lead to higher perceived quality and lower prices of all drugs, thus increasing access; but it could also lead to substantial exit of generics and higher prices due to reduced price competition. It is even theoretically possible that the equilibrium with higher quality standards entails lower perceived quality and reduced access, say, if certification costs are large enough to induce substantial exit among high-quality generics. The ambiguity of theoretical predictions partly motivates the empirical analysis we develop in the remainder of the paper.

Although it is not possible to determine a priori what the equilibrium effects of stronger quality regulation are in our framework, higher fixed costs of quality certification are generally associated with worse equilibrium outcomes. In particular, large certification costs decrease generic entry; therefore, they harm price competition. We discuss the role of certification costs in detail in the next section.

27. Note that, to the extent that the stronger quality regulation results in both higher generic quality and higher prices, consumers with a sufficiently low willingness-to-pay for quality are worse off, and some reduce their consumption of the drug. This happens for consumers with $\tau_i \leq \Delta p_G / \Delta\bar{\psi}$, where Δp_G is the change in prices and $\Delta\bar{\psi}$ is the change in perceived generic quality.

2.3.3 *The Importance of Fixed Compliance Costs and Market Size*

In this section, we simulate our model to illustrate the equilibrium effects of stronger quality regulation. In particular, we study the effects of stronger quality regulation and their relationship with the cost of quality certification κ_{QC}. The effect of κ_{QC} is of particular interest, because it is a reform-specific cost that is fully covered by generics and acts as a sunk cost to participate in the market, with the potential for affecting market structure.

Our simulation consists of solving for market equilibrium across a range of minimum quality standards, separately for the cases with free and costly compliance, $\kappa_{QC} = 0$ and $\kappa_{QC} > 0$ respectively. In particular, we highlight three regulatory environments in which: (a) there is a baseline level of quality regulation in the form of a minimum quality standard; (b) there is stronger quality regulation but it does not impose any certification costs κ_{QC} on generic producers; and (c) there is stronger quality regulation and quality certification is costly for generic producers. Details on the model specification and parametrization used for this exercise and formulas for all calculations are provided in Appendix B.1.1.

Figure 2.1 displays the simulation results, where we highlight the three environments, labelled by **a**, **b** and **c** respectively. Compared with the baseline scenario (**a**), quality regulation with costless certification (**b**) increases consumer surplus and welfare. These effects are driven by increased perceived generic quality without large decreases in generic competition, which limits the extent to which generic prices increase; and decreased innovator price due to decreased vertical differentiation with generics. Moreover, generic prices increase slightly and the market share of generics increases at the expense of the innovator. For the case with costly certification (**c**), consumer surplus and welfare fall, driven by higher prices of all drugs due to reduced competition caused by substantial generic exit. In this case, the market share of generics decreases, and that of the outside good increases. Overall, our illustration suggests that stronger quality regulation may be able to decrease vertical differentiation and increase the intensity of price competition, but that fixed compliance costs may counteract such forces and lead to adverse effects.

Higher market size M reduces the importance of fixed costs, and is a source of heterogeneity in the effects of the reform. As we illustrate in Appendix B.1.2, the detrimental

competitive effects of fixed compliance costs are stronger in smaller markets than in large markets, everything else constant. In particular, the model predicts that fixed compliance costs induce more exit and larger price increases in small markets. We exploit this theoretical result in our empirical analysis to test the model predictions related to κ_{QC} by contrasting results for small and large markets.

2.4 Data and Descriptive Statistics

2.4.1 Data Sources

We employ three sources of data for our empirical analysis. First, we use the drug registry maintained by ISP for the Chilean pharmaceutical market, which provides licensing data for the universe of drugs marketed in the country. The registry provides information on manufacturer (laboratory), the date when the drug was first licensed in Chile, the date of the last license approval, and the due date of the next license renewal. It also includes information on the drug dosage (e.g., number of milligrams of the active ingredient contained in each tablet), presentation (i.e. tablet, capsule, injectable, or other), and marketing status (prescription, over-the-counter, or discontinued). We restrict our analysis to molecules under a bioequivalence requirement within the sample period, which includes all molecules with bioequivalence requirements initiated after 2010. Our data cover all licensed drugs up to December 2017. Second, we combine the drug registry data with data on bioequivalence certification by drugs in the market, which are also available from ISP. These data contain a list of all drugs with bioequivalence certification, including certification date and the corresponding reference drug.

Finally, we use data from IMS Health Chile, which contain detailed information on monthly prices and sales of drugs sold across the market for the period between January 2010 and December 2017. IMS Health collects data from two sources. The four largest pharmacy chains in the country, accounting for more than 90% of drugs sold in Chile, report retail prices and sales directly to IMS Health. Sales from other pharmacies are supplied by wholesalers, which report wholesale prices and sales to IMS Health. Wholesale prices

are transformed to retail prices using a standard methodology.[28][29] We employ monthly sales and prices from all 83 local markets included in the IMS Health data, which cover most of the urban areas of the country. We aggregate prices and sales for each drug across local markets. In particular, we compute total monthly sales by aggregating monthly sales across local markets and calculate monthly drug prices as sales-weighted averages of prices across local markets.[30]

The IMS Health dataset provides price and sales at the product level for branded drugs, identifying the laboratory, dosage and presentation of each drug. For unbranded drugs, it provides price and sales at the dosage and presentation level, aggregated across laboratories.[31] We focus prescription drugs, which account for more than 90% of drugs in the molecules we study.

2.4.2 Descriptive Statistics for Quality Certification

The number of bioequivalent drugs in the Chilean market increased substantially throughout our study period. Figure 2.2-a shows the number of bioequivalent drugs between January 2010 and December 2017. Bioequivalence certification started at a low pace in early 2010, but increased steadily since then, with a rapid uptake by mid-2012. By December 2017, there were 1,433 drugs with bioequivalence certification in our sample, among which 909 were branded generics.

The growth in the number of bioequivalent drugs relates to the regulation roll-out, which was announced and implemented at different dates through the decrees and deadlines described in Section 2.2.2. Figures 2.2-b through 2.2-e display the number of bioequivalence

28. This methodology consists of adding a VAT of 19% and a retail margin of 30%.

29. We adjust retail prices in two ways. First, we transform nominal prices to real prices in 2013 using the health CPI from the National Institute of Statistics (*Instituto Nacional de Estadística*, INE). Second, we normalize drug prices across drug presentations by their drug content by calculating prices per gram of the active ingredient.

30. There is little variation in drug prices across local markets, and no geographic variation in any of the sources of identifying variation we use in the main analysis of the paper.

31. This limitation of the IMS Health data imposes some restrictions on our analysis, because all unbranded generics of a given molecule, presentation, and dosage are coded together as if they were manufactured by a single laboratory. In particular, it limits the extent to which we can track the composition of sales of a given unbranded generic across laboratories over time.

approvals around four policy events of each market: (1) the first decree, (2) the last decree, (3) the first deadline, and (4) the last deadline. We highlight three facts from these figures. First, bioequivalence approval was uncommon before the first decree, which shows that bioequivalence incidence was rare before it was mandated by law. Second, bioequivalence approval increased markedly after the first decree, which suggests that bioequivalence regulation had an impact on bioequivalence incidence. Third, several bioequivalence approvals occurred after the first and last deadlines, which shows that deadlines were only imperfectly enforced, a point to which we return in our empirical strategy.

2.4.3 Descriptive Statistics for Market Outcomes

We merged the price and sales data from IMS health with the drug registry from ISP, to construct a monthly panel dataset for the period between January 2010 and December 2017. After some data cleaning, the resulting dataset covers 131 molecules. The data contain 2,292 unique drugs, defined as a unique combination of drug name, dosage, and presentation. These drugs are manufactured by 80 different laboratories.[32] Importantly, not all drugs in the panel are sold in every period. In fact, only 65.5% of the drug-month observations in our panel dataset display positive sales. Drug prices are not observed for months in which a drug registers no sales.

Table 2.2 displays basic descriptive statistics. On average, innovator drug prices are around twice as high as those of the average drug in the market, whereas branded (unbranded) generic prices are around two thirds (one fifth) of the average drug in the market. We go beyond these raw averages and estimate price premiums within markets for innovator and branded generics below. The highest market share is captured by branded generics, with an average market share of 43%, followed by innovator and unbranded generics with market shares of 30% and 27%, respectively. On average, bioequivalent drugs hold a market share of only 7%. However, the average market share of bioequivalent drugs increased substantially during our study period, from only 0.06% in 2010, to 22.8% by the

32. As stated above, for this calculation, all unbranded generics within a given molecule, dosage, and presentation, are counted as being produced by the same laboratory due to limitations in the IMS Health data.

end of 2017. This shift in market shares is also displayed by Figure 2.4. The average market has around 13 drugs and five laboratories in a given month. As expected, the numbers of drugs and laboratories are remarkably larger in the segment of branded generics than in the innovator and the bioequivalent segment.[33]

Figure 2.5 shows pre-reform price premiums per drug type, using 2010 and 2011 prices.[34] Four facts become apparent: First, price premiums are on average positive across all molecules in the sample. Second, price premiums are large overall: innovators and branded generics are substantially more expensive than unbranded generics, with average relative premiums of 10 and 6 times, respectively. Third, relative price premiums are much larger for innovator drugs than for branded generics. Fourth, there is substantial heterogeneity in price premiums across molecules. Whereas several molecules display relative price premiums on the order of 3 to 5 times, several other molecules display relative price premiums higher than 10 times, particularly for innovator drugs.

2.5 Effects of Quality Regulation on Certification, Entry and Exit

We start our analysis by studying quality certification and exit by drugs in the market. First, we study whether drugs that became exposed to bioequivalence requirements obtained bioequivalence approval. Second, we study whether drugs were more likely to exit the market once bioequivalence requirements were imposed. For this analysis, and for the remainder of the paper, we follow Duggan et al. (2016) and treat each molecule as a separate market, because there is generally little to no substitution across molecules for the treatment of health conditions.

33. This partly comes from our inability to identify different producers of unbranded drugs in IMS Health, as explained in Section 2.4.1.

34. We calculate these premiums by estimating regressions of logged (real) prices per gram in 2010 and 2011 on indicators for innovator and branded generics separately for each market. The exponentiated coefficients on the indicators for drug type provide a measure of average price premiums of each type relative to unbranded generics (the omitted category). We restrict the estimation sample to molecules with price information for at least one innovator drug, one branded drug and one unbranded drug during the period, which limits the sample to 56 molecules.

2.5.1 *Evidence for Bioequivalence Approval*

In section 2.4.2, we provided suggestive evidence that bioequivalence certification increased substantially after the reform. We now turn to survival analysis to study its determinants. Survival analysis is a convenient method to describe bioequivalence approval, because it flexibly accommodates the absorbing nature of bioequivalence, right-censoring, and time-varying covariates.

The hazard function $h(s)$ measures the probability of becoming bioequivalent in period s. We parameterize $h(s)$ using a proportional hazard model for drug i in market m and calendar month t that takes the following functional form:

$$h(s|X_{imt}, t) = \lambda_s \times \exp(X'_{imt}\beta + \psi_t). \tag{2.1}$$

where λ_s is a *baseline* hazard that depends on drug tenure s (measured in months since entry to the market) and is estimated non-parametrically. Coefficients in β measure the proportional increase in the hazard following a one-unit increase in the corresponding covariate. The vector X_{imt} includes indicators for branded and imported drugs, logged average market revenue in the past 12 months, and logged counts of branded and unbranded drugs in the market, as well as indicator variables for time periods after policy decrees and deadlines. We consider the same four market-specific events analyzed in section 2.4.2: date of first deadline, date of first decree, date of last deadline, and date of last decree. We quantify the changes in the probability of becoming bioequivalent after each event date t^d_m with indicators $\mathbb{1}(t > t^d_m)$. Finally, ψ_t are calendar month fixed effects.

Table 2.3-A displays estimates from equation (2.1). Column (1) through (4) include each policy event separately, whereas column (5) includes all of them jointly. The most relevant policy events are the first decree and the first deadline, which increase the probability of becoming bioequivalent by exp(2.52) = 12.4 and exp(1.78) = 5.9 times, respectively, whereas posterior policy events do not significantly increase the hazard of quality certification. These results reinforce the graphical evidence of Figure 2.2: periods after the first decree and first deadline are stronger predictors of bioequivalence certification than periods after the last decree and last deadline. Also, drugs are more likely to become bioequivalent after

the first deadline than after the last deadline, showing that the first deadline triggered a higher rate of bioequivalence certification than subsequent extensions.

We then turn to analyze the relationship between bioequivalence approval rates and drug characteristics as well as market variables. Branded and imported drugs are estimated to be more likely to obtain bioequivalence approval, although the coefficients are not statistically significant. Market variables are strong predictors of bioequivalence approval: A 10% increase in market revenue is associated with a 5.8% increase in the hazard of becoming bioequivalent. Moreover, the number of competing drugs in a market is negatively associated with bioequivalence approval. A 10% increase in the number of branded drugs is associated with a 2.9% lower hazard rate, whereas a 10% increase in the number of unbranded drugs is associated with a 3% lower hazard rate.

Heterogeneity. We study how baseline drug attributes affect quality certification choices. Table B.3-A displays results from a version of equation (2.1) in which policy events are interacted with indicators for drug covariates at baseline.[35] We focus on the first deadline of bioequivalence requirements for a market, which showed to be the most relevant in our baseline analysis. The most relevant pattern of heterogeneity we find is that drugs with higher baseline revenue are differentially more likely to engage in quality certification after bioequivalence requirements are imposed, as predicted by the model in Section 2.3. In particular, a 10% increase in revenue is associated with a differential increase in the hazard rate of 1.3%.

2.5.2 Evidence for Entry and Exit of Drugs

We turn to analyze the relationship between bioequivalence regulation and the dynamics of entry and exit. We construct measures of entry and exit using the ISP registry data on licensing and renewals. For each registered drug, we record an entry as the event of

35. Baseline drug characteristics are measured as indicators for whether a drug was, on average, above or below the median drug in their market during 2010. These characteristics are constructed using the IMS Health data. The number of observations decreases relative to that in Table 2.3-A because several drugs were not in the market in 2010. The comparison between column (2) in Table 2.3 and column (1) in Table B.3 shows that both samples deliver similar results for the baseline specification in equation (2.1).

obtaining a license for the first time, and an exit as the event of not renewing a license upon expiration.[36] Figure 2.3-a shows the total number of drugs that entered and exited the market during our sample period. We find that drug exit was relatively stable up to late 2014, and that there was a large increase in the number of exiting drugs afterwards. On the other hand, we do not find a large change in entry during the period. Figures 2.3-b through 2.3-e display the number of drugs that entered and exited the market at each point in time relative to relevant policy events. These figures show that the marked increase in exit of drugs occurred after the enactment of the bioequivalence policy.

We estimate a hazard model for drug exit to quantify these patterns, analogous to that in equation (2.1). Our results are shown in Table 2.3-B. We focus on Column (10), which displays estimates from a specification that includes all policy variables jointly. The results imply that the first deadline is the policy variable that most strongly influences drug exit. In particular, the probability of exiting increases by $\exp(0.42) = 1.52$ times after the first deadline. Branded drugs have a lower propensity to exit compared with unbranded, and innovator drugs display a lower exit hazard rate than generics. Interestingly, imported drugs are more likely to exit. We do not find significant effects of market variables on exit, which display similar effects across specifications.

Heterogeneity. We implement a heterogeneity analysis of exit rates. Table B.3-B displays results for heterogeneity in the effect of the first deadline of bioequivalence requirements on drug exit. We do not find any strong patterns of heterogeneity. However, we find suggestive evidence of the overall determinant of exit: conditional on market size and the number of competing drugs, drugs with higher sales and revenues at baseline are less likely to exit the market, as expected.

36. Thus, for the purpose of this exercise, we assume that exit happened exactly at the due date of the failed renewal (i.e. five years after the last renewal) although the decision to exit was likely taken some time before the due date.

2.6 Effects of Quality Regulation on Market Outcomes

We now turn to the main analysis of the paper, where we estimate the effects of quality regulation on market outcomes. We employ an empirical strategy that exploits variation in the roll-out timing of bioequivalence requirements within and across markets. We explore potentially heterogeneous effects of the policy in line with the model in Section 2.3, focusing on the differences in the effects of quality regulation across small and large markets.

2.6.1 Empirical Strategy

Our empirical strategy exploits policy variation across and within markets. The first source of identifying variation is the staggered roll-out of the reform, as already discussed in Section 2.2.2. This variation is displayed in Figure A.17-a. In practice, the differences in the timing of the regulation generate a series of comparison groups comprised of markets that faced bioequivalence requirements at different dates throughout our period of study. The second source of identifying variation comes from a particular feature of the institutional setting. In practice, deadlines for incumbent drugs become binding every time a drug must renew its marketing license with ISP, i.e. every five years. As stated by ISP officials, regulation enforcement occurs mostly at the time of license renewal, when ISP is likely to deny renewal to drugs without bioequivalence approval (Vasallo, 2010). Thus, for each drug, the first license renewal after the policy deadline marks the effective deadline to comply. License-renewal dates vary across drugs within each market, reflecting the date at which the drug was first licensed, and are arguably exogenous for drugs that were in the registry before the deadline was known. Differences in renewal dates across drugs generate variation in the share of drugs for which the policy is effectively binding, both across markets sharing the same deadline, as well as within markets over time.

We combine these two sources of variation by constructing a variable that measures the evolution of the policy roll-out within each market. This variable captures three main features of the regulation. First, the policy becomes relevant for a market only after its first corresponding decree. Second, the policy becomes increasingly relevant for each drug in the market as its respective license renewal date approaches. Finally, the policy is fully

in place for a market when the license renewal date has been reached for all drugs in it. Formally, denote the policy date for market m by t_m^d and renewal date of drug i in m by t_{im}^r. For a given drug i, the share of time between the decree and next renewal date that has elapsed by time any time t is given by:

$$T_{imt} = \begin{cases} 0 & \text{if } t \leq t_m^d \\ \frac{t - t_m^d}{t_{im}^r - t_m^d} & \text{if } t_m^d < t \leq t_{im}^r \\ 1 & \text{if } t_{im}^r < t \end{cases}$$

For each market m, we then define the *share of market under regulation* by month t as the average of T_{imt} across the set of generic drugs (branded and unbranded) present in market m in period t_m^d, $\mathcal{G}_m$:

$$T_{mt} = \frac{1}{|\mathcal{G}_m|} \sum_{i \in \mathcal{G}_m} T_{imt} \tag{2.2}$$

where $|\mathcal{G}_m|$ is the number of generic drugs present in market m in month t_m^d.

We employ T_{mt} as a treatment variable for our analysis of the effect of the regulation on market outcomes. T_{mt} is a weakly increasing function of time relative to the policy date t_m^d: it is equal to 0 before t_m^d and is equal to 1 after the latest renewal date across drugs in $\mathcal{G}_m$ is reached. Figure A.17-b displays the evolution of T_{mt} over time for all markets in the sample, showing substantial variation across markets at any given point in time, as well as variation within market across time.[37] Finally, Figure A.17-c shows that this variable is correlated with the share of bioequivalent drugs in the market, even after accounting for market and month fixed effects.

Our main specification for measuring policy effects on market-level outcomes y_{mt} is given by:

$$y_{mt} = \beta T_{mt} + \theta_m + \delta_t + \varepsilon_{mt} \tag{2.3}$$

37. For further illustration, Figure B.2 shows particular examples for the evolution of T_{mt} over time for four markets, along with the evolution in the number of bioequivalent drugs in each of them. These examples are highlighted in Figure A.17-b. These plots show how bioequivalence certification increases as bioequivalence requirements become relevant for a market.

where the coefficient of interest, β, is interpreted as the effect of the fully implemented bioequivalence policy on outcome y_{mt}. We include two sets of fixed effects: θ_m are market fixed effects that control for permanent differences across markets that may be correlated with T_{mt}, and δ_t are time (year-month) fixed effects that control for shocks common to all markets in a given period of time. To interpret our results, we discuss the effect of an increase in T_{mt} from zero to one, corresponding to the estimated effect of moving from not having bioequivalence regulation to having the regulation fully in place for a given market.

The key identifying assumption in (2.3) is that there are no unobserved market-specific trends that drive both the timing of the policy roll-out and the outcomes of interest. The main assumption behind this strategy is that decree deadlines and renewal dates were not set as a function of unobserved shocks not captured by market and time fixed effects. A violation to this assumption would happen if, for example, decrees and deadlines were set earlier for markets that were expected to have earlier price increases. Although we cannot directly test this, the fact that decrees were set and modified mostly based on capacity constraints of laboratories testing bioequivalence makes it unlikely that they were driven by unobserved future demand or supply shocks.

Moreover, market-level observable characteristics do not show a clear correlation with the timing of the policy. Table 2.1-B shows descriptive statistics for market outcomes in 2010 for markets affected differently by the policy. Overall, these statistics display substantial heterogeneity across different groups in terms of number of drugs, market size, and market outcomes, but do not display a clear pattern related to the timing of bioequivalence requirements roll-out. Furthermore, Table B.2 displays results from an ordered logit model for the timing of the policy on baseline market attributes in 2010, including variables related to market structure, market size, price and treatment. We find that most of these attributes are unrelated to the timing of the policy, and the only significant predictor of policy timing is having a low branded generic market share. Moreover, the pseudo-R^2 of the is not larger than 3.6% across specifications. The timing of the policy roll-out is thus mostly unrelated to baseline market characteristics.

Event-Study Evidence. As a complement to estimating equation (2.3), we implement an event-study analysis. The event study serves two purposes: (i) assessing the plausibility of the assumption of parallel trends across groups of molecules treated by the policy at different dates; and (ii) providing transparent visual evidence of the effects of bioequivalence on relevant market outcomes. A disadvantage of the event study compared to our main specification based on equation (2.3) is that it does not exploit the within-market identifying variation coming from the pattern of drug license renewal dates. We describe this event study analysis in Appendix B.2 and provide results in Figure B.6. Overall, trends in outcomes before the first deadline of bioequivalence requirements are well behaved, as most of the estimated coefficients are close to zero. This fact is reassuring in terms of exploiting the differential timing of decrees across markets as exogenous variation to estimate the effects of quality regulation in our setting. Moreover, the results obtained from this event-study analysis are consistent with those from our main analysis in this section.

Heterogeneity. The model in Section 2.3 suggests that whenever compliance is costly, quality regulation should have stronger effects in smaller markets because it would induce more drug exit. To test this prediction, we estimate differential effects of the policy according to market size, measured as the average sales in the pre-reform period. Specifically, we divide markets according to whether the average monthly market revenue in 2010 was above or below the median and identify them as large and small markets respectively.

2.6.2 Effects of Quality Regulation on Market Structure

We start by discussing the estimated effects of bioequivalence regulation on market structure. We focus on two key features of market structure, the number of drugs of different types that are in the market and the number of laboratories offering drugs in each segment of the market.

Results for Number of Drugs

We start by estimating equation (2.3) using the number of drugs as dependent variable.[38] Column (1) in Table 2.4-A shows the results across drug types. We find that the policy decreased the number of drugs in the market by 25%. Columns (2)–(8) split these results across different drug types. The overall reduction is driven by exit of branded and unbranded generics. We estimate a 26% decrease in the number of branded generics and a 25% decrease in the number of unbranded generics. However, we estimate a negative effect for innovator drugs, although it is not statistically significant. Even though there is an increase in the number of bioequivalent generics, it is not enough to compensate for the exit of non-bioequivalents. The fact that the number of drugs in the market decreased as a result of stronger quality regulation is consistent with the model in Section 2.3, which suggests that the intensity of price competition in the market may have decreased.

Consistent with our model predictions, the negative effects on the number of drugs are particularly pronounced in small markets, driven by a significant amount of exit by both innovator drugs and generics. We estimate that the number of drugs decreased by 35% in small markets and 15% in large markets, as shown by Table 2.4-B. Conversely, bioequivalence certification is significantly larger in high-revenue markets. This is also consistent with our model, because a larger market makes bioequivalence certification relatively less costly.

Results for Number of Laboratories

Since most manufacturers are multiproduct firms, we turn to study whether drug exit is driven by exit of manufacturers or changes in their drug portfolios. Evidence of laboratory exit as a result of stronger quality regulation would imply unintended competitive effects.

The number of laboratories competing in a given market decreased by 14% on average

38. We use $\ln(1 + N_{mt})$ as dependent variable, where N_{mt} is the number of drugs (i.e., the number of presentations), to accommodate observations where there are no drugs of a certain category, e.g., no bioequivalent unbranded generics. As a robustness check, we show that the results are virtually unchanged when using $\sinh^{-1}(N_{mt})$ as the dependent variable in Table B.4. This transformation also reduces skew, yields coefficients approximating percentage changes, and allows for zeros, all of which are desirable statistical properties with this type of data (see, e.g., Kline et al. 2017).

as a result of the reform, as shown by Table 2.5-A.[39] This reduction in the number of firms comes mostly from a decrease in the number of laboratories offering generics, whereas we find no significant effect on the number of laboratories offering innovator drugs. However, we find a large increase in the number of laboratories offering bioequivalent generics. Table 2.5-B displays heterogeneous effects across small and large markets. Our results are consistent with our findings for the number of drugs and with the model predictions. We find that stronger quality regulation reduced the number of competing firms in small markets: the number of laboratories in small markets decreased by 23%, whereas it did not change significantly in large markets. The decrease in the number of laboratories in small markets is mostly driven by exit of laboratories offering unbranded drugs. Conversely, entry of laboratories to the segments of branded and unbranded bioequivalents was stronger in large markets.[40]

Combining the estimates of policy effects on the number of drugs and the number of laboratories, we can measure the effect on the number of drugs per laboratory. Our estimates imply that, across markets, 40% of the decrease in the number of drugs is driven by a reduction in the number of drugs offered by laboratories rather than by the exit of laboratories. Consistent with our previous findings, this result is heterogeneous across market sizes. As much as 68% of the effect on the number of drugs comes from laboratory exit in small markets, whereas 43% of the effect on the number of drugs comes from laboratory exit in large markets.[41]

The finding that a large share of drug exit is due to a reduction in the size of the portfolio of laboratories gives some support to the notion that laboratories selectively test for bioequivalence. It is reasonable to believe that (the underlying) bioequivalence status of drugs is highly homogeneous within laboratories, such that variation in bioequivalence

39. For this analysis, we treat different laboratories owned by a same conglomerate as the same laboratory. We thank Gastón Palmucci and Thomas Krussig at the National Economic Prosecutor of Chile (*Fiscalía Nacional Económica*, FNE) for help in constructing this dataset.

40. As a robustness check, we estimate the same regressions using $\sinh^{-1}(N_{mt})$ as the dependent variable. See footnote 38 for details. Table B.4 displays results for these specifications. Overall, the results are remarkably similar to those using $\ln(1 + N_{mt})$ as the dependent variable.

41. For completeness, we report results of regressions using the average number of drugs per laboratory as the dependent variable. Table B.6 displays results for those specifications.

certification within laboratories reflects heterogeneity in drug profitability. Selective testing based on drug profitability is consistent with regulation compliance costs being a driver for our results.

2.6.3 Effects of Quality Regulation on Drug Prices

We turn to studying the effects of quality regulation on drug prices. Having documented large changes in market structure, we interpret these price effects as the combination of different forces at play. A reduction in the number of competitors—particularly a large exit of branded generics—may lead to price changes due to reduced competition. As described in Section 2.3, the sign of the price change of incumbent competitors is ambiguous. Innovators are expected to increase their prices to exploit their increased market power.[42] Moreover, changes in market structure are coupled with potential changes in consumer perceived quality, changing the scope for vertical differentiation and the intensity of price competition.

We estimate the effects of quality regulation on a market price index constructed as the share-weighted average of log prices (see, e.g., Chevalier et al., 2003; Nevo and Hatzitaskos, 2006):

$$\hat{P}_{mt} = \sum_{i \in \mathcal{I}_{mt}} w_{it} P_{it} \tag{2.4}$$

where $\mathcal{I}_{mt}$, is the set of drugs in the market in period t, P_{it} is the logarithm of price per gram of product i in period t and w_{it} denotes the share of sales of drug i in market m in period t.

Average prices across all drugs increased by 10% as a result of the regulation, as shown by Table 2.6-A. We estimate price effects by drug type and find that most of the increase in average paid prices comes from increases among unbranded generics, whereas innovators and branded generics display no statistically significant changes.[43] As shown in Section

42. Another theoretical possibility is that innovators decrease their prices to cater a more elastic part of the demand (see, e.g., Frank and Salkever (1992), which we illustrate using our model in Appendix B.1.2).

43. We construct the same price index for each drug type, but define the weights as shares within the corresponding type. The effect of the regulation for the type-specific price indices are computed for the subset of markets for which there is at least one drug of that type in the baseline period.

2.6.2, the decrease in the number of drugs is concentrated in small markets; therefore, these are the markets where we expect to find the strongest competitive effects, which is largely confirmed by our heterogeneity analysis in Table 2.6-B. The increase in prices across all drugs is driven by an increase of 26% in small markets. Our estimates show that stronger quality regulation induced price increases of 7% and 18% among innovator drugs and unbranded generics respectively in small markets. On the other hand, our estimates for price effects in large markets are close to zero and not statistically significant.

Decomposition of Price Effects

The effects on average prices at the market level combine drug-specific price changes (P_{it}), changes in shares (w_{it}), and changes in the composition of drugs offered in each market. To better understand the drivers of price effects, we decompose the evolution of average market prices into such components.

Consider the change in the share-weighted average of log prices between a baseline period $t = 0$ and any period $t > 0$. Denote the set of drugs in the market in t that were also in the market in the baseline period as $\mathcal{S}_{m,t} \equiv \mathcal{I}_{mt} \cap \mathcal{I}_{m0}$; the set of drugs that entered market m after the baseline period and remain in the market in period t as $\mathcal{E}_{mt} \equiv \mathcal{I}_{mt} \setminus \mathcal{I}_{m0}$; and the set of drugs that exited between the baseline period and t as $\mathcal{X}_{mt} \equiv \mathcal{I}_{m0} \setminus \mathcal{I}_{mt}$. We decompose the change in the share-weighted average of log prices as:

$$\begin{aligned}\sum_{i\in\mathcal{I}_{mt}} w_{it}P_{it} - \sum_{i\in\mathcal{I}_{m0}} w_{i0}P_{i0} \;=\; & \underbrace{\sum_{i\in\mathcal{S}_{mt}} w_{i0}(P_{it}-P_{i0})}_{\Delta P_{mt,C}} + \underbrace{\sum_{i\in\mathcal{S}_{mt}} (P_{it}-P_{m0})(w_{it}-w_{i0})}_{\Delta P_{mt,RW}} \\ & + \underbrace{\sum_{i\in\mathcal{S}_{mt}} (w_{it}-w_{i0})(P_{it}-P_{i0})}_{\Delta P_{mt,CS}} + \underbrace{\sum_{i\in\mathcal{E}_{mt}} w_{it}(P_{it}-P_{m0})}_{\Delta P_{mt,E}} \\ & - \underbrace{\sum_{i\in\mathcal{X}_{mt}} w_{i0}(P_{i0}-P_{m0})}_{\Delta P_{mt,X}}\end{aligned}$$

The first term, $\Delta P_{mt,C}$, measures the change in the share-weighted average price due

to price changes among incumbent drugs, holding weights fixed at their baseline level. The second term, $\Delta P_{mt,RW}$, measures the change in the share-weighted average due to changes in relative market shares, holding prices fixed. This term is positive (negative) when relatively expensive (cheap) incumbent drugs increase their market share. The third term, $\Delta P_{mt,CS}$, measures the change in sales-weighted prices due to the correlation between price changes and changes in market shares. This term is positive (negative) when drugs that increase their prices are also those that increase (decrease) their market shares. The fourth term $\Delta P_{mt,E}$, captures price changes due to the entry of drugs in the market. This component is positive (negative) whenever drugs that enter the marker are more (less) expensive that the average drug in the baseline period. Finally, the fifth term, $\Delta P_{mt,X}$, measures the change in the share-weighted average due to the exit of drugs. This component is positive (negative) whenever drugs that exit the market are less (more) expensive than the average drug in the baseline period. It follows that the price index can be decomposed as:

$$\hat{P}_{mt} = \hat{P}_{m0} + \Delta P_{mt,C} + \Delta P_{mt,RW} + \Delta P_{mt,CS} + \Delta P_{mt,E} + \Delta P_{mt,X} \tag{2.5}$$

To evaluate the effect of the policy on these components of price changes, we estimate equation (2.3) for the following dependent variables: $\hat{P}_{mt,C} \equiv \hat{P}_{m0} + \Delta P_{mt,C}$, $\hat{P}_{mt,RW} \equiv \hat{P}_{m0} + \Delta P_{mt,RW}$, $\hat{P}_{mt,CS} \equiv \hat{P}_{m0} + \Delta P_{mt,CS}$, $\hat{P}_{mt,E} \equiv \hat{P}_{m0} + \Delta P_{mt,E}$ and $\hat{P}_{mt,X} \equiv \Delta\hat{P}_{m0} + P_{mt,X}$. By construction, the sum of the OLS coefficients on T_{mt} from these regressions is equal to the coefficient on T_{mt} in equation (2.3). Each coefficient measures the policy effect on the corresponding component of the price index.

Most of the increase in overall prices is driven by within-drug price changes. Table 2.6-C, shows estimates for policy effects on each of the components, both for the overall market price and for the price of each drug type. Of the 10% increase in average prices, 7 p.p come from price changes within drugs ($\hat{P}_{PC}$) and 2 p.p from the entry of relatively expensive drugs ($\hat{P}_{E}$). Similarly, we also find that most of price increases among unbranded generics are due to within-drug price changes ($\hat{P}_{PC}$). As noted above, unbranded generics are aggregated across laboratories; therefore, the decomposition for this segment should be

interpreted with caution. Overall, the finding that the estimated increase in average drug prices is due mostly to price increases of products already in the market before the policy supports our interpretation that the exit of drugs documented in Section 2.6.2 reduced the intensity of price competition in the market.

2.6.4 Effects of Quality Regulation on Market Shares and Sales

We now estimate the effect of quality regulation on quantity outcomes. We are mostly interested in exploring whether quality regulation significantly affected generic penetration. Given our model, we expect changes in market shares due to changes in the market structure and changes in demand.

Table 2.7-I-A displays estimates of equation (2.3) for market shares. Overall, we do not find significant changes in the market shares of innovator drugs and generics. If anything, we find a non statistically significant increase of 4 p.p in the market share of innovator drugs and a decrease of the same magnitude in the market share of generics. The decrease in the market share of generics is concentrated among branded generics, whereas the market share of unbranded generics remains unchanged. As expected, we find a significant increase of 10 p.p. in the market share of bioequivalent generics and a decrease of 14 p.p. in non-bioequivalent generics.[44] Considering the decrease in the number of branded generics found in Table 2.4, these results are consistent with consumers mostly substituting towards innovator drugs as generics exit the market.

Most of the increase in the innovator market share comes from a 8 p.p increase in small markets, as shown by Table 2.7-I-B. In contrast, we find no significant change in the innovator market share in large markets. Moreover, in large markets—where we find smaller exit among generics—we find a shift from branded to unbranded generics: we estimate a decrease of 6 p.p in the market share of branded generics and a 4 p.p increase in the market share of unbranded generics.

We now focus on the effects of the policy on total sales. Estimating the effect on sales allows us to disentangle changes in market shares of different types of drugs from changes

44. As previously explained, we are unable to separate unbranded generics between bioequivalents and non-bioequivalents due to limitations of the IMS Health data.

in the size of the outside option. We are particularly interested in evaluating whether the substantial drug exit induced substitution towards stayers, or if it increased the share of the outside option. In theory, stronger quality regulation can either increase or decrease the share of the outside option. However, an increase in the perceived quality of generics could induce individuals choosing the outside option to purchase generics. Moreover, there are endogenous price effects caused by changes in the market structure and the extent of vertical differentiation.

Drug sales decreased as a result of the regulation. Table 2.7-II-A displays estimates of equation (2.3) using sales volume as dependent variable. Point estimates are negative and large in magnitude, but we find no statistically significant effect on sales of innovator drugs and unbranded generics across all markets. However, we estimate a decrease of 37% in sales of branded generics. These results indicate that stronger quality regulation generated substitution towards the outside option.

Finally, we find that decreases in sales are concentrated in small markets, as shown by Table 2.7-II-B. We estimate that sales decreased by 29% across all drugs as opposed to a smaller and non-statistically significant decrease in sales in large markets of 9%. The overall decrease in sales in small markets is driven by decreases in sales of both branded and unbranded generics. This result is consistent with our results showing substantial exit and reduced competition in small markets. In contrast, we estimate that in large markets there is a large but not statistically significant decrease in sales of branded generics, whereas there is an increase in sales of unbranded generics of 60%.

2.6.5 Effects of Quality Regulation on Drug Quality

Imposing bioequivalence requirements as a minimum quality standard was successful in inducing generics willing to stay or enter the market to obtain bioequivalence certification. However, we documented that stronger quality regulation affected market structure, particularly by inducing exit in small markets.

Theoretically, we expect the rate of bioequivalence certification to be higher in larger markets even if the underlying drug quality is constant across markets of different size, as

shown in our model of Section 2.3. The compliance cost associated with the regulation acts as a fixed entry cost that only firms expecting to earn profits large enough are willing to incur, as predicted by standard entry models (e.g., Bresnahan and Reiss 1991). Therefore, the compliance cost induces the exit of drugs of high quality but low revenue, with potentially adverse welfare consequences. Alternatively, the underlying drug quality prevailing before the policy change could have varied across markets of different size. When product quality is endogenous and produced with fixed costs, larger markets can sustain higher quality levels (Berry and Waldfogel, 2010). In that context, market revenue and underlying product quality are positively correlated; therefore, a higher exit in low-revenue markets may imply that the average quality in the market increased after the reform.

To inform this margin, we study whether the bioequivalence regulation had any measurable effects on improving the quality of drugs in the market. Finding no quality effects would be consistent with higher exit in small markets being associated with negative welfare consequences. Direct measures of quality (e.g., results from laboratory drug testing) are not available in our setting, which motivates using drug adverse effects and recalls as indirect measures of drug quality.

Let the quality outcome for market j in time t be $y_{jt} = \mu_{jt} sales_{jt}$, where μ_{jt} is the probability of an adverse effect associated with drugs in market j, and $sales_{jt}$ is the amount sold of drug t. Similar to Jin and Leslie (2003), we model the probability of adverse events as $\mu_{jt} = \mu_{j0} + \gamma_t + \theta T_{jt} + \varepsilon_{jt}$, which combines a baseline probability μ_{j0}, with time shocks common to all markets γ_t, a shifter related to quality regulation θT_{jt}, and a random shock ε_{jt}. This simple framework motivates the equation we estimate, which is:

$$\frac{y_{jt}}{sales_{jt}} = \mu_{j0} + \gamma_t + \theta T_{jt} + \varepsilon_{jt} \tag{2.6}$$

where θ is the parameter of interest and measures true quality improvements as the effect of stronger quality regulation on the number of adverse outcomes per unit of sales. μ_{j0} and γ_t are captured by market and time fixed effects.

Evidence from Adverse Health Events

A first set of outcomes related to drug quality are the adverse health events associated with their consumption. We collect data on yearly clinical outcomes between 2010 and 2017 for ICD-10 diagnosis codes associated with active ingredients in our sample, including admissions and days of hospitalization, and number of surgeries.[45] Adverse effects of drugs are relatively rare in our setting. In 2010, there were on average 7.3 admissions, 13.2 hospital days and 0.002 surgeries per 100,000 daily doses sold across all markets.

We estimate equation (2.6) using these outcomes as dependent variable. Columns (1) through (3) in Table 2.8-A display results from this analysis across all markets. We find no evidence suggesting that stronger quality regulation decreased the number of discharges and the number of days associated with them. Moreover, we find no evidence of effects on these outcomes when estimating separately for small and large markets in Table 2.8-B. These results suggest that stronger quality regulation was not able to affect health outcomes associated with adverse effects from drug utilization.

Evidence from Drug Recalls

Additionally, we estimate effects of stronger quality regulation on drug recalls. We collect data on the 209 recalls for prescription drugs that occurred between January 2010 and December 2017. Recalls are implemented by ISP upon notice of adverse events associated with a licensed drug that justify recall as a preventative sanitary measure.[46] In the period we study, there is an average of 1.9 recalls per month, of which 1.4 (0.5) relate to active

45. We construct this dataset using public records collected by DEIS (2019), which cover every hospital in Chile. We allocate diagnosis to active ingredients using a crosswalk between American Hospital Formulary Service (AHFS) and ICD-9 codes by WHO (2007). When several ICD-10 codes capture adverse effects associated with the same active ingredient, we assign outcomes to active ingredients using weights for sales volume across active ingredients within each ICD-10 code. As an example of the resulting dataset, admissions coded under "T455 - Poisoning by, adverse effect of and underdosing of anticoagulants and antithrombotic drugs" are attributed to the consumption of Acenocoumarol (an anticoagulant). We restricted the analysis to the subsample of active ingredients with at least one adverse effect, which are 52 out of 131.

46. The reasons for these recalls can be categorized broadly into (i) manufacturing defects including chemical defects and contamination (71%); (ii) efficacy concerns or side effects (19 %); or (iii) others, which mostly correspond to counterfeit drugs or mislabeling (20 %). Due to the small number of recall events, we use all data irrespective of the specific reason.

ingredients without (with) bioequivalence requirement. Figure B.7 shows the monthly recall frequency, split into drugs subject to bioequivalence requirements (and included in our sample), and drugs without bioequivalence requirement. As a first test for quality effects, we cannot reject the null hypothesis of a same trend in recalls over time across these two groups.[47]

Our estimates of equation (2.6) in the sample of active ingredients with bioequivalence requirements provide no evidence suggesting that stronger quality regulation improved drug quality as measured by recalls. Columns (4) and (5) in Table 2.8-A display results across all markets, while Table 2.8-B does so by market size. Our point estimates are close to zero across specifications.

2.6.6 Discussion

We provide evidence for the equilibrium effects of quality regulation and interpret it using our model in Section 2.3. Our estimates imply that stronger quality regulation had sizable effects. We start by showing that stronger quality regulation induced drugs to exit the market. Drug exit combines reductions in the portfolio of drugs offered by laboratories within a market with a reduction in the number of laboratories. Whereas one could have expected stronger quality regulation to reduce vertical differentiation and increase the intensity of price competition, our estimates suggest that the negative effect through market structure overturned those positive competitive effects. We find that drug prices increased as a result of the policy. Furthermore, we find no evidence of an increase in the market share of generics, which was one of the main motives behind the policy. Finally, we provide suggestive evidence that drug quality did not improve, at least as measured by adverse health events and recalls.

Most of the adverse effects from stronger quality regulation are concentrated among small markets. This pattern of heterogeneity suggests that laboratories decide to exit the market whenever the fixed cost of regulation compliance is large enough relative to the

47. We test the null hypothesis of no differential trends by estimating an OLS for the recall rates on an indicator of having a requirement, and its interaction with a time trend. We find that the interaction term is not significantly different than zero.

profitability of the market, as predicted by our model. In particular, our estimates for small markets follow the model predictions for changes in equilibrium under costly compliance (a shift from **a** to **c** in Figure 2.1), whereas our estimates for large markets are consistent with the model predictions under free compliance (a shift from **a** to **b** in Figure 2.1).

It is important to stress that the overall welfare effects of quality regulations are theoretically ambiguous and, in particular, that lower compliance costs make the policy more likely to yield increases in welfare. On the demand side, a higher willingness-to-pay for quality tends to both increase the likelihood of high-quality generics to enter the market and increase the impact on consumer surplus from higher average quality in the market. We formalize these arguments and provide an illustration of them in Appendix B.1.2.

2.7 Complementary Evidence from Consumer Surveys

Our findings show that quality regulation had unexpected adverse effects. Whereas its goal was to increase price competition by reducing quality differentiation, we find that drug exit due to compliance costs reduced competition and led to price increases. There are several potential explanations for why stronger quality regulation had these adverse effects. For instance, consumers may not update their perceived quality of generics accordingly. Large biases against generics reduce incentives for bioequivalence certification and, in turn, reduce the scope for the intended competitive effects of the policy. Part of those biases could be related to a lack of understanding of what bioequivalence means. Moreover, consumers may understate the (often large) price differences between innovators and generics, reducing search. Finally, physicians may limit the extent to which bioequivalence affects consumer choices through prescribing innovators or branded generics.

We collect survey data on consumers to assess different aspects of their purchase behavior, including attitudes towards generics, their understanding and familiarity with bioequivalence, as well as the role of physicians in influencing their purchase decisions. We conducted in-person surveys to frequent consumers who were recruited outside pharmacies after a drug purchase. To collect perceptions, we focus on Atorvastatin, a common anti-cholesterol drug with a large market presence in Chile. We asked consumers

for their quality and price perceptions for different drugs representing the different drug types, namely the innovator drug (Lipitor, by Pfizer), a bioequivalent branded generic (Lipoten, by Pharmavita) and bioequivalent and non-bioquivalent unbranded generics (Atorvastatina, by Mintlab). For more details about the survey design and methodology, see Appendix B.3. We collected surveys from $N = 401$ consumers, of which 58% reported having a household member with a chronic disease, and 34% reported purchasing Atorvastatin for a household member. Table B.7 provides summary statistics for the main variables in the survey.

2.7.1 Main Results

Knowledge About Bioequivalence. There is substantial heterogeneity in knowledge about bioequivalence among consumers in our sample, despite the fact that 84% of consumers are familiar with the label attached to bioequivalent drugs. Figure 2.7-a shows that almost 30% of consumers are not familiar at all with bioequivalence and 55% is not able to provide a good definition for it.Limited knowledge about bioequivalence might reduce the extent to which bioequivalence effectively signals drug quality and induce consumers to switch from innovator or branded generic drugs to cheaper bioequivalent unbranded generics.

Perceived Quality Differences. Consumers display substantial variation in their perceived quality of drugs in the market. We collect data on the perceived quality for each drug on a 1-7 scale. We define the perceived quality premium as the difference between the perceived quality of the innovator drug and that of another drug type. Figure 2.7-b displays the distribution of perceived quality premiums relative to the innovator. As expected, consumers perceive that the innovator drug is of higher quality than branded and unbranded generics. Branded generics are perceived to have a slightly better quality than unbranded generics. Additionally, consumers perceive that bioequivalent drugs are of higher quality than non-bioequivalent drugs. Therefore, consumers attribute a quality premium to bioequivalence, although not large enough as to close the quality premium attributed to innovators. This might be partly due to a poor understanding of what bioe-

quivalence means. We explore this possibility in Figure 2.7-c, which shows that for all drug types, the quality premiums attached to innovators are weakly lower for consumers with high knowledge about bioequivalence than for consumers with low knowledge about it, which is consistent with Bronnenberg et al. (2015).[48] This pattern is particularly strong for bioequivalent unbranded generics.

Perceived Price Premiums. To complement these facts about perceived quality, we collect data on perceived price differences. An additional explanation for our findings is that consumers underestimate the price differences between drug types. This demand-side friction would decrease substitution towards generics and limit incentives for laboratories to stay or enter the market under stronger quality regulation. Figure 2.7-d displays perceived price premiums of the innovator drug relative to other drug types.[49] Consumers perceive that prices of generics are substantially lower than those of innovator drugs. On average, consumers perceive that branded generics, bioequivalent unbranded generics and non-bioequivalent unbranded generics have discounts of 49%, 68%, and 75% relative to the innovator, respectively. Moreover, a large share of the consumers identify discounts of unbranded generics between 90% and 100%. Whereas perceived price differences are lower than actual price differences, these patterns suggest that consumers are to a large extent aware of differences in market prices across drug types.

The Role of Physicians. Prescription behavior by physicians plays a key role in drug purchase behavior and generic penetration (Dickstein, 2015). This has motivated policies of *generic substitution* in different countries, so as to limit the extent to which physicians prescribing expensive named drugs may limit generic penetration. We gather information regarding consumer experiences with prescription behavior of physicians. We find that 65% of consumers answer that physicians often prescribe drugs by the name instead of the active ingredient. However, consumers display some degree of willingness to deviate

48. We classify consumers with none or low knowledge about bioequivalence as uninformed and those with medium, high or excellent knowledge about bioequivalence as informed consumers.

49. The actual price of the innovator drug we consider is around $50,000 CLP, whereas the prices of the branded and unbranded generics are around $10,000 CLP and $2,500, respectively ($77.5, $15.5 and $7.8 U.S. dollars, respectively). Actual discounts are therefore in the order of 80% and 95%, respectively.

from physicians' recommendations. Conditional on a physician prescription, only 15% of consumers state they purchase the prescribed named drug *always and regardless of drug prices*, whereas 52% state that they deviate from the brand prescribed by the physician whenever there is a large enough price difference. Finally, 34% of respondents state that they shop only on price, disregarding the brand recommended by their physician.

2.7.2 Discussion

We employ a consumer survey to explore potential explanations for the unintended consequences of stronger quality regulation that we document in our main analysis. We show that, after almost 10 years since the beginning of the reform to quality regulation, a large share of consumers has none or an imprecise understanding of what bioequivalence means. In terms of the model in Section 2.3, this evidence implies that $\overline{\psi} < \psi_I$.[50] Additionally, we find that perceived quality premiums are lower for consumers with a higher understanding of bioequivalence. This evidence is related to research on how biases against generics limit generic penetration (Bronnenberg et al., 2015; Colgan et al., 2015; Bairoliya et al., 2017). Moreover, it suggests that information policies might be complementary to quality regulation by inducing consumers to update their perception about perceived generic quality in accordance with the regulation.

Additionally, our survey highlights two additional barriers for generic penetration. On the one hand, whereas consumers are aware about the existence of price differences across different drug types, they underestimate them. On the other hand, consumers argue that physicians most often prescribe brand-named drugs, which limits the extent to which consumers choose generics. The fact that consumers mention they are willing to disregard physicians' recommendations whenever price differences are large enough limits, but do not eliminate, the effect of physician behavior on generic penetration. These are two

50. This survey does not allow the direct measurement of the perceived quality of generics before the reform, and thus the estimation of changes in the perceived quality of generics due to it. Making a strong assumption on the evolution of perceived quality, one could argue that the policy influenced the perceived quality by comparing the perceived quality of bioequivalent and non-bioequivalent unbranded generics: the perceived quality premium of bioequivalent unbranded generics is 60% lower than that of non-bioequivalent unbranded generics, which suggests the policy did affect perceived quality.

additional barriers for generic penetration.

Overall, the results of the survey point towards the existence of barriers to generic penetration in our setting. These frictions undermine the ability of the regulation to effectively shift consumers towards generics that have proven to be bioequivalent. These barriers, in turn, reduce the profitability of generic manufacturers to entering or remaining in the market, relative to the fixed regulation compliance cost. This is consistent with the finding in our main analysis, where we documented a reduction in the number of drugs in the market and an increase in drug prices as a result of stronger quality regulation, particularly for small markets.

2.8 Conclusion

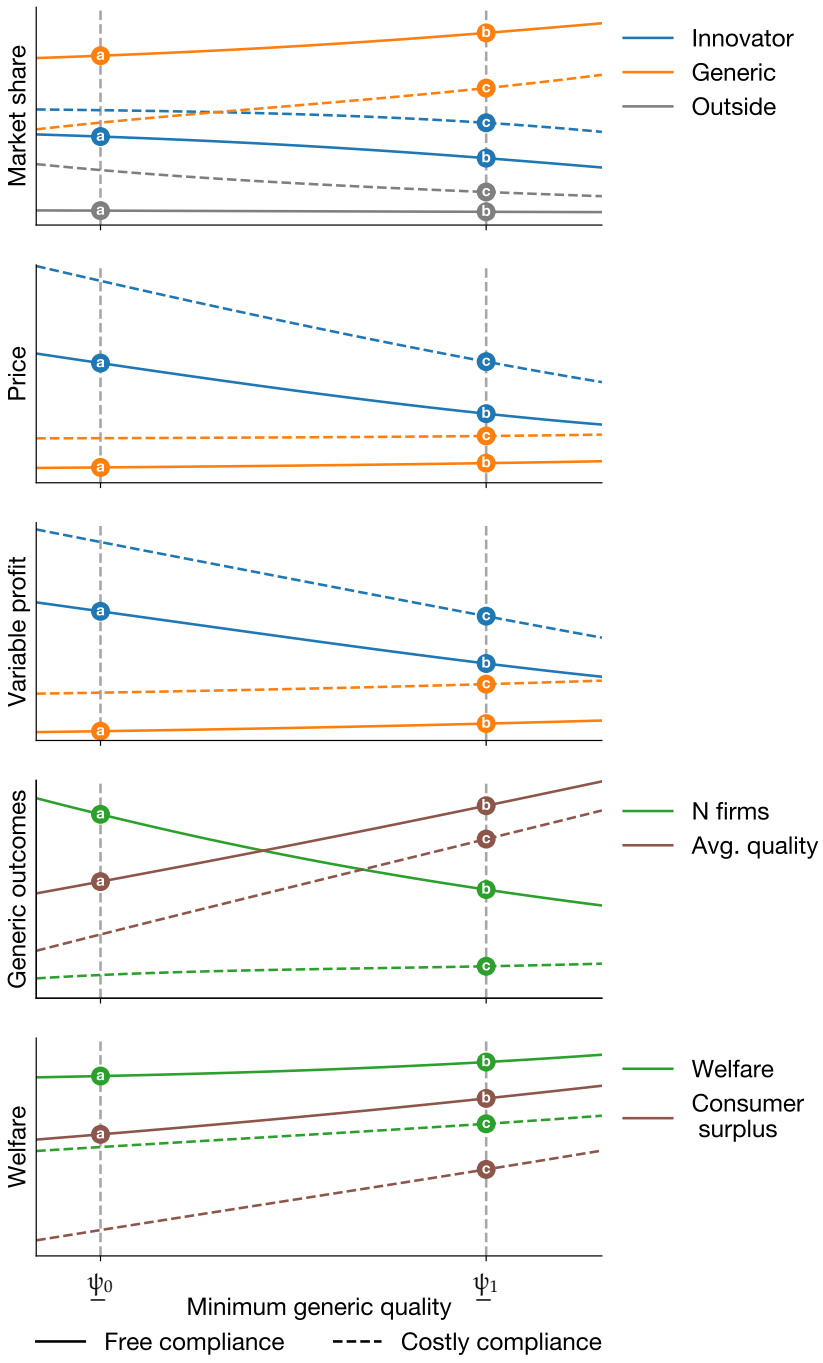
Quality regulation in markets with asymmetric information may ensure product quality, change consumer perceptions of product quality and foster price competition by reducing vertical differentiation. However, costly compliance with these regulations may also have unintended adverse consequences on market structure by inducing product exit and, thereby, harm price competition.

We study a reform to bioequivalence requirements in the Chilean pharmaceutical market. Our findings suggest that quality regulation may have unintended competitive effects. Contrary to the motivation of reducing prices through increased competition, we find that average paid prices increased, and that the market share of generics did not increase. These effects are concentrated among low-revenue markets, where we also find sizable exit. We employ an equilibrium model of competition in pharmaceutical markets to interpret these findings. We argue that fixed compliance costs imposed by stronger quality regulation induced exit, which, in turn, decreased the intensity of price competition.

Stronger quality regulation can generate desirable competitive effects, and our analysis provides lessons for the design of a quality regulation to achieve them. Through the lens of our model, we find that the main driver of the unintended consequences we find are regulation compliance costs. Inefficiencies caused by compliance costs point towards the desirability of subsidizing certification costs, which may limit drug exit from the market

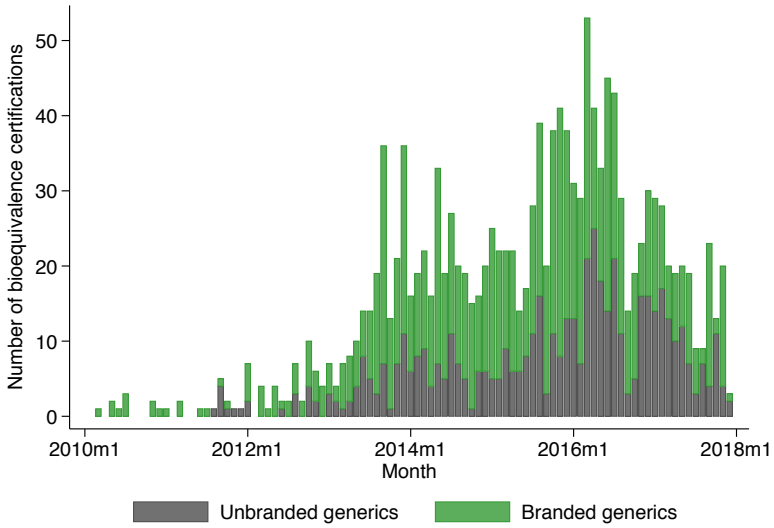
and, therefore, prevent decreases in the intensity of price competition. Additionally, the competitive effects of quality regulations depend crucially on how they affect demand, and pharmaceutical markets impose particular challenges in this regard. First, demand reactions are limited by prescribing behavior of physicians, whose incentives may differ from those of their patients (Dickstein, 2015). Second, attitudes towards generics may only change slowly over time as consumers learn about their quality (Bairoliya et al., 2017). Unexperienced consumers may have long-lasting biases against generics; therefore, quality regulation may not achieve its desired effects in the short run. Consumer survey data we gathered from the Chilean market confirms the presence of these lasting biases and frictions, and points towards the need of complementary policies to achieve the desired competitive effects of minimum quality standards.

Figure 2.1: Effects of Quality Regulation: With and without Costly Compliance/Certification

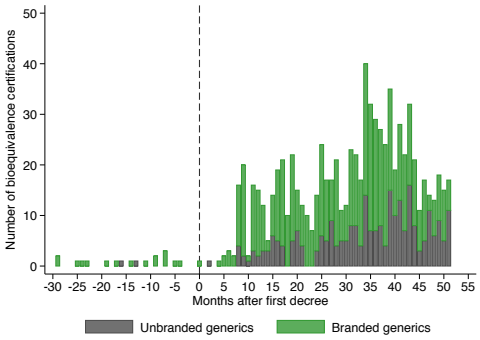


Notes: Market outcomes for different levels of minimum quality in the market. The dashed (solid) lines represent a situation with (no) compliance costs. Example minimum qualities before and after regulation are indicated by $\underline{\psi}_0$ and $\underline{\psi}_1$, where points **a** indicate pre-reform outcomes, **b** indicates post-reform outcomes if compliance was free, while **c** indicates post-reform outcomes with costly compliance. Simulation details are provided in Appendix B.1.

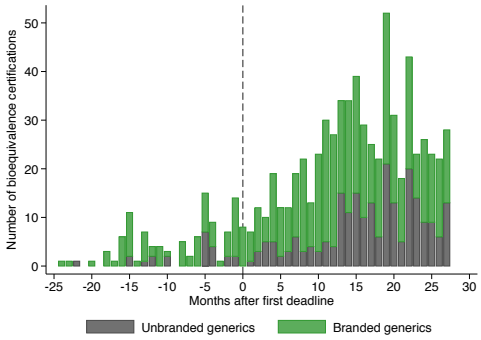
Figure 2.2: Bioequivalence Approvals around Policy Events



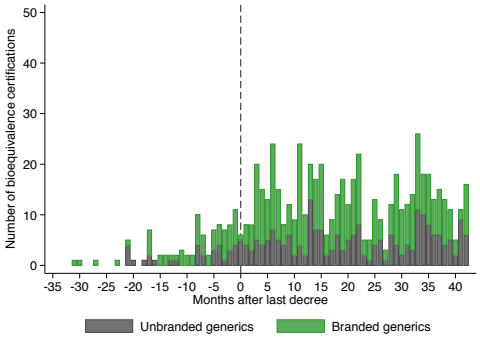
(a) Approvals over time



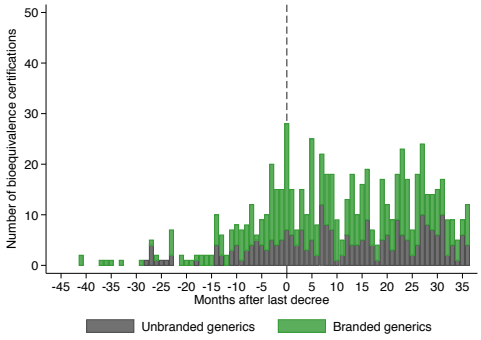
(b) Approvals around first decree



(c) Approvals around first deadline



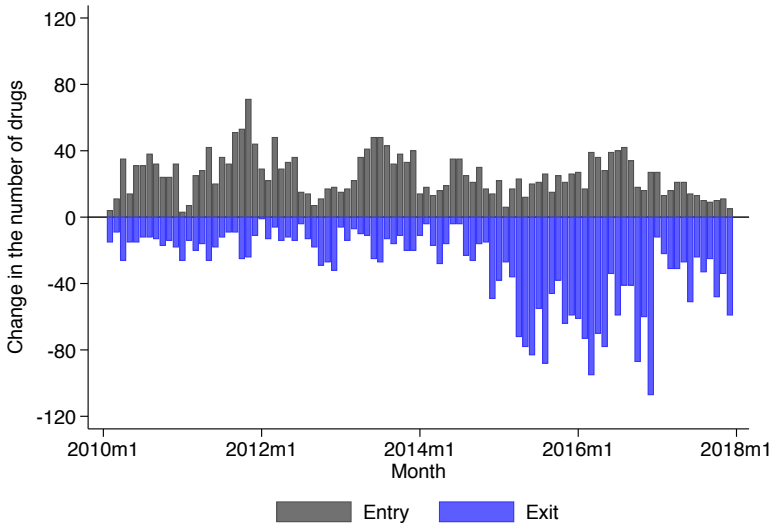
(d) Approvals around to last decree



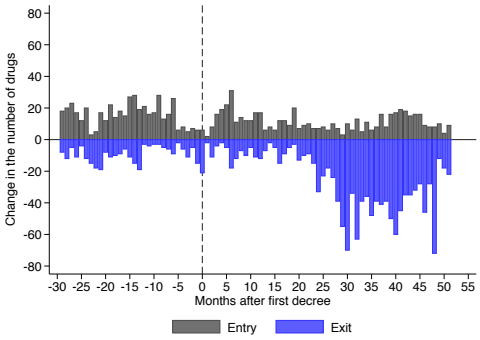
(e) Approvals around to last deadline

Notes: Panel (a) in this figure displays the evolution of the number of drugs with bioequivalence approval over time, split by unbranded generics (gray) and branded generics (green). Panels (b) through (e) display the number of bioequivalence approvals around bioequivalence decrees and deadlines.

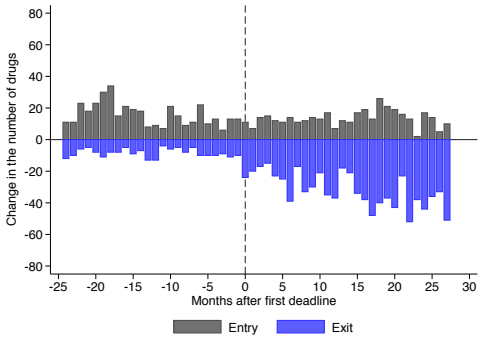
Figure 2.3: Number of Entry and Exit of Drugs around Policy Events



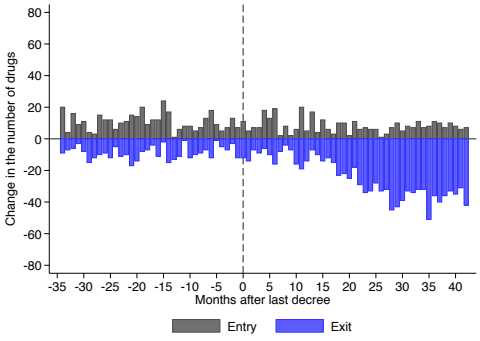
(a) Entry and exit over time



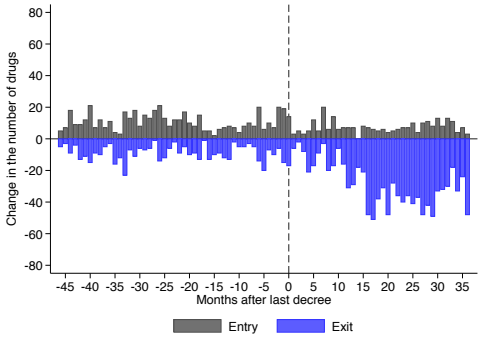
(b) Relative to first decree



(c) Relative to first deadline



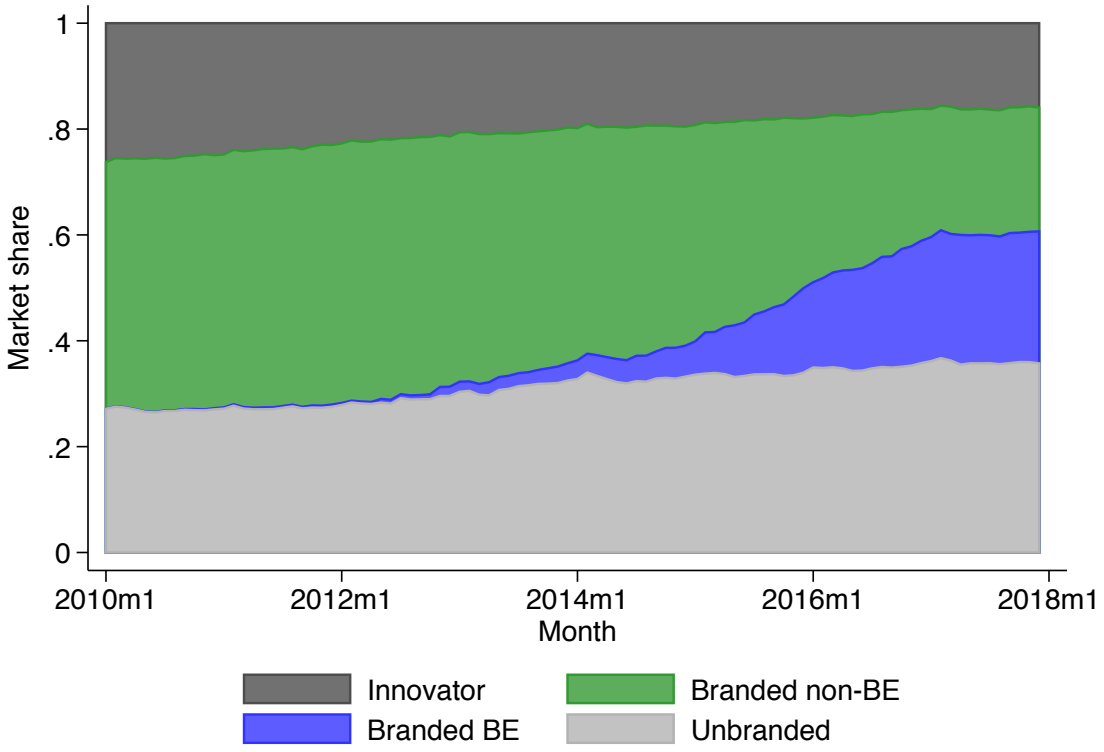
(d) Relative to last decree



(e) Relative to last deadline

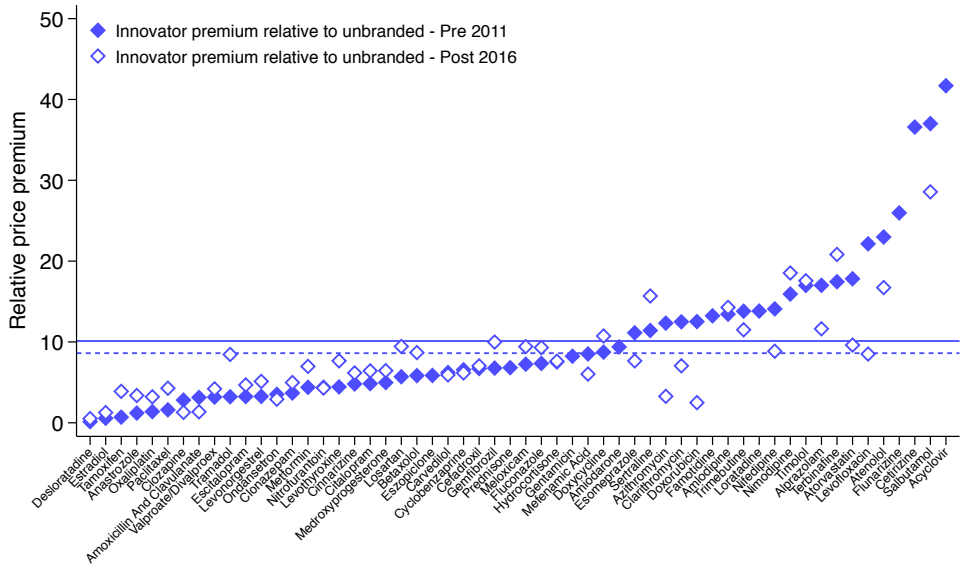
Notes: This figure displays the number of entering (gray) and exiting (blue) drugs around bioequivalence decrees and deadlines. The vertical axis displays the count of such events. Panel (a) display the evolution of entry and exit of drugs over time, while panels (b) through (e) display the evolution of entry and exit relative to bioequivalence decrees and deadline.

Figure 2.4: Market Shares by Drug Type

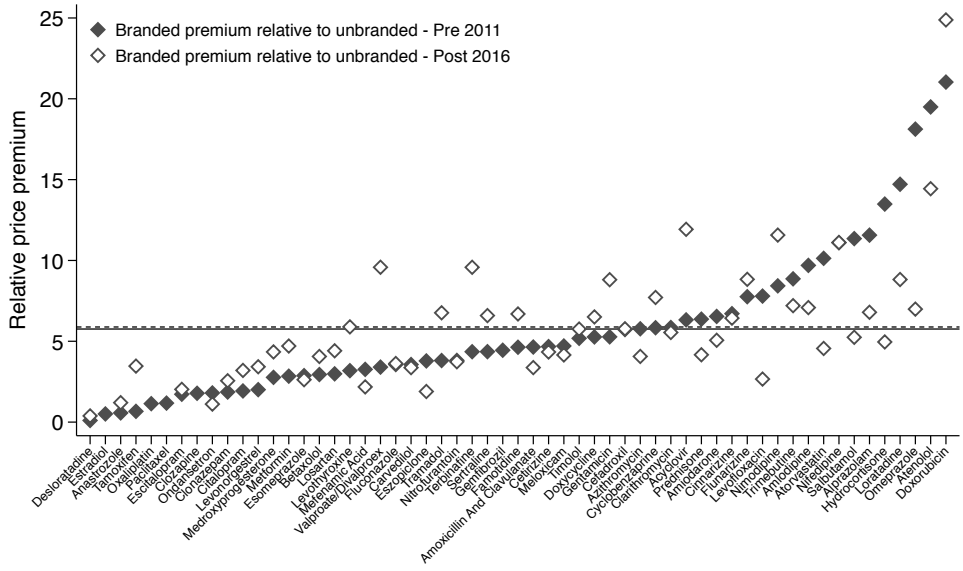


Notes: This figure displays the evolution of market shares of different drug types over time. For each type, we plot the average market share across markets for each month in our sample.

Figure 2.5: Innovator and Branded Drugs Price Premiums by Market



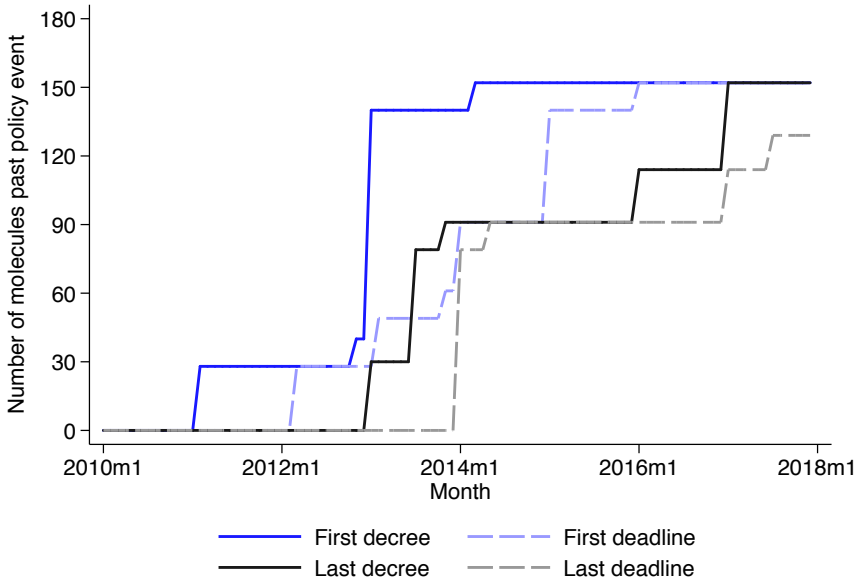
(a) Innovator drugs price premiums relative to unbranded generics



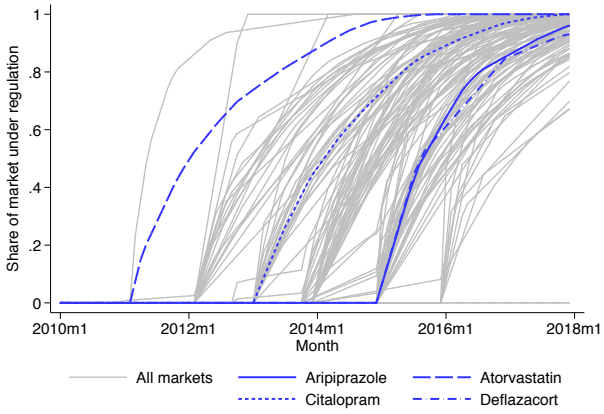
(b) Branded drugs price premiums relative to unbranded generics

Notes: This figure displays estimated price premium for innovator and branded generic drugs relative to unbranded generic drugs. Each dot in the figure corresponds to an exponentiated coefficient from a regression of log prices on innovator and branded drug dummies, estimated separately for each molecule using data for 2010-2011 and 2016-2017 for the pre and post periods respectively. The sample of markets is that with price information for at least one innovator, one branded and one unbranded drug during that period. Solid and dashed lines indicate the average price premium across this set of molecules for the pre and post period respectively.

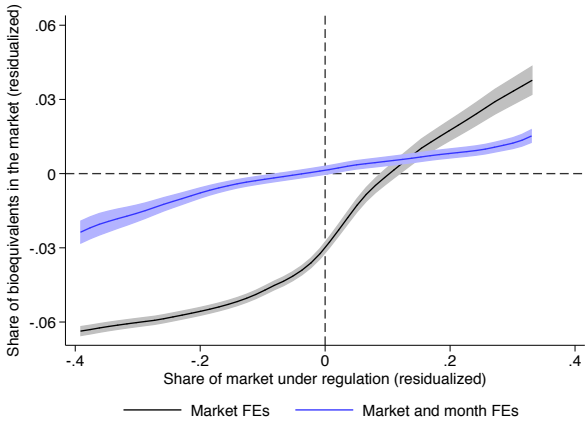
Figure 2.6: Evolution of Quality Regulation



(a) Timing of bioequivalence decrees and deadlines



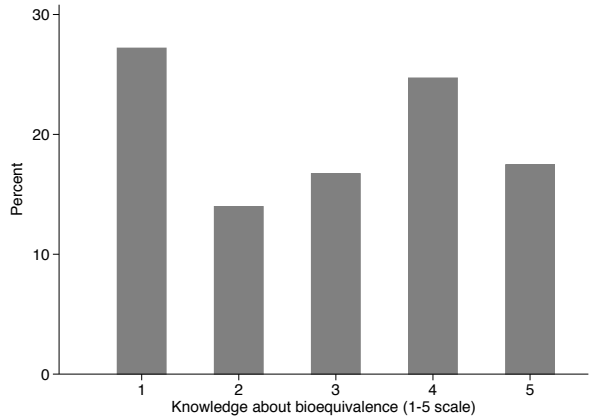
(b) Evolution of quality regulation by market



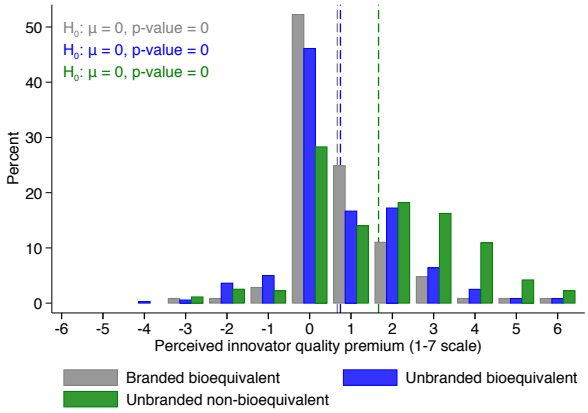
(c) Quality regulation and share of bioequivalent drugs

Notes: Panel (a) in this figure displays the number of markets affected by different policy events associated to bioequivalence regulation, from the first decree to the last deadline. Panel (b) displays the evolution over time of the treatment variable defined in equation (2.2) for each market in the sample. This version of the treatment variable uses the first deadline as the relevant date. We highlight some particular examples in blue, which are displayed in more detail in Figure B.2. Panel (c) displays the non-parametric relationship between the residualized policy intensity variable and share of bioequivalent drugs in the market, controlling for market fixed effects (gray) and market and month fixed effects (blue) over the range of variation of the latter. The bottom and top centiles of the data are not included in the plot.

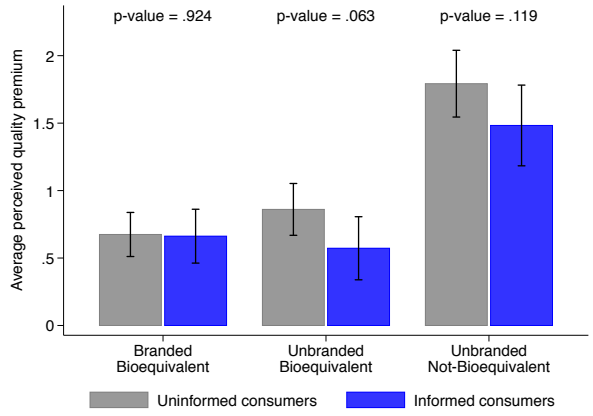
Figure 2.7: Survey Results



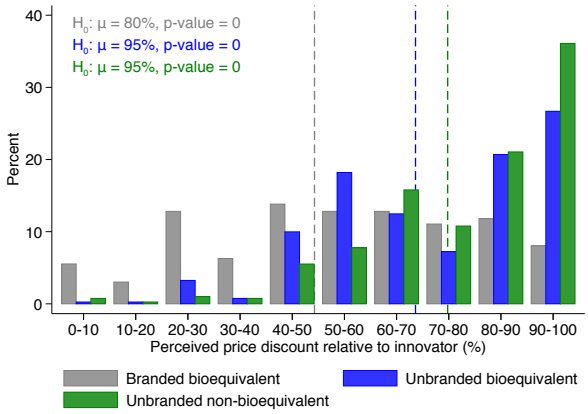
(a) Knowledge about bioequivalence



(b) Perceived quality premium



(c) Knowledge and perceived quality



(d) Perceived price premium

Notes: Panel (a) displays the distribution of consumer knowledge about bioequivalence in a 1-5 scale, where 1 means the consumer is not familiar with bioequivalence at all, and 5 means the consumers is able to provide a good definition of what it is. Panel (b) displays the distribution of perceived quality premiums for different drug types relative to the innovator drug. The premium is calculated as the difference between the perceived quality of the innovator drug and the perceived quality for each drug type, where premium is recorded in a 1-7 scale. Panel (c) displays average quality premium for each drug type across uninformed and informed consumers, where the former are those with knowledge between 1 and 2 in panel (a), and the latter are those with knowledge between 3 and 5 in it. The figure displays 95% confidence intervals for each mean, as well as p-values from a two-sided test of equality between average perceived quality premiums of uninformed and informed consumers. Finally, panel (d) displays the distribution of perceived price discounts of each drug type relative to the innovator drug. Dashed lines in panels (b) and (d) indicate the average for each drug type in the figure.

Table 2.1: Timing of Reform: Policy Variables and Descriptive Statistics

		Panel A: Relevant policy dates				*Panel B: Market characteristics*					
	Number of	First decree		Last decree		Number of	Average	Average	Share of drugs by segment		
Group	Molecules	Decree	Deadline	Decree	Deadline	drugs	price	revenue	Innovator	Branded	Unbranded
1	4	2011-01	2011-02	2013-06	2013-12	67	101	29,190	0.26	0.63	0.10
2	20	2011-01	2012-02	2013-06	2013-12	193	562	29,900	0.31	0.55	0.14
3	11	2012-10	2013-10	2013-10	2014-04	91	485	19,650	0.17	0.58	0.25
4	25	2012-12	2013-12	2012-12	2013-12	378	302	20,607	0.24	0.66	0.10
5	20	2012-12	2013-01	2013-06	2013-12	354	218	23,754	0.22	0.74	0.04
6	10	2012-12	2014-12	2015-12	2016-12	108	1,280	29,255	0.24	0.76	0.00
7	15	2012-12	2014-12	2016-12	2017-06	227	390	21,407	0.26	0.71	0.03
8	16	2012-12	2014-12	2016-12	2017-12	133	672	18,165	0.25	0.64	0.10
9	10	2014-02	2015-12	2016-12	2017-12	28	10	8,414	0.04	0.33	0.63

Notes: Panel A in this table displays the dates of announcement and deadlines of BE requirements for different groups of molecules. The groups are defined as a unique combination of decrees and deadlines. Panel B in this table displays average product characteristics in 2011, by groups of molecules. Prices per gram and revenues are measured in 2013 U.S. dollars.

Table 2.2: Descriptive Statistics for IMS Data

Variable	N	Mean	SD	p10	p50	p90
Panel A: Price per gram						
All drugs	144,106	461.1	4,183.2	2.3	36.0	583.3
Innovators	33,251	900.2	3,886.7	4.3	73.7	1,868.0
Branded generics	96,909	365.8	4,552.7	3.1	36.9	391.9
Unbranded generics	13,946	76.1	327.3	0.4	3.0	130.3
Bioequivalents	17,455	164.3	594.4	2.2	22.6	278.6
Panel B: Market shares						
Innovators	12,576	0.30	0.30	0.00	0.22	0.80
Branded generics	12,576	0.43	0.34	0.00	0.44	0.89
Unbranded generics	12,576	0.27	0.36	0.00	0.04	0.99
Bioequivalents	12,576	0.07	0.16	0.00	0.00	0.29
Panel C: Number of drugs						
All drugs	12,576	12.56	11.30	2.00	9.00	29.00
Innovators	12,576	2.92	2.61	0.00	2.00	6.00
Branded generics	12,576	8.44	9.57	0.00	5.00	23.00
Unbranded generics	12,576	1.20	1.38	0.00	1.00	3.00
Bioequivalents	12,576	1.46	3.88	0.00	0.00	5.00
Panel D: Number of laboratories						
All drugs	12,576	4.77	3.25	1.00	4.00	10.00
Innovators	12,576	0.82	0.50	0.00	1.00	1.00
Branded generics	12,576	3.38	3.05	0.00	2.00	8.00
Unbranded generics	12,576	0.57	1.36	0.00	0.00	2.00

Notes: This table displays descriptive statistics from the IMS data. Statistics for prices are displayed in 2013 U.S. dollars and calculated at the drug level, while the remainder are calculated at the market level. Market shares are only observed for markets in which at least one drug is sold in the period. Statistics for the number of drugs and laboratories are computed using only observations for which the drug or laboratory is found to be active in the corresponding market.

Table 2.3: Hazard Model for Bioequivalence and Exit

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	Panel A: Bioequivalence approval hazard					*Panel B: Exit hazard*				
After first decree	2.52***				1.29**	0.16				-0.05
	(0.48)				(0.58)	(0.16)				(0.16)
After first deadline		1.78***			1.63***		0.42***			0.41***
		(0.31)			(0.31)		(0.14)			(0.16)
After last decree			-0.11		-0.23			0.17**		0.02
			(0.20)		(0.26)			(0.08)		(0.12)
After last deadline				-0.22	-0.25				0.16*	0.09
				(0.21)	(0.27)				(0.08)	(0.12)
Reference						-0.68***	-0.69***	-0.67***	-0.68***	-0.68***
						(0.14)	(0.14)	(0.14)	(0.14)	(0.14)
Branded	0.08	0.08	0.07	0.07	0.07	-0.36***	-0.35***	-0.35***	-0.35***	-0.35***
	(0.10)	(0.10)	(0.10)	(0.10)	(0.10)	(0.07)	(0.06)	(0.06)	(0.06)	(0.06)
Imported	0.15	0.14	0.17	0.17	0.15	0.52***	0.52***	0.52***	0.52***	0.51***
	(0.13)	(0.12)	(0.13)	(0.13)	(0.12)	(0.07)	(0.07)	(0.07)	(0.07)	(0.07)
log(Market revenue)	0.62***	0.57***	0.65***	0.66***	0.58***	-0.02	-0.03	-0.02	-0.02	-0.03
	(0.14)	(0.13)	(0.14)	(0.14)	(0.13)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)
log(Number of branded)	-0.30*	-0.28	-0.30*	-0.31*	-0.29*	0.01	0.01	0.01	0.01	0.01
	(0.17)	(0.17)	(0.17)	(0.17)	(0.16)	(0.06)	(0.06)	(0.06)	(0.06)	(0.06)
log(Number of unbranded)	-0.30*	-0.36**	-0.26	-0.24	-0.30*	-0.09	-0.10	-0.11	-0.11	-0.11
	(0.17)	(0.17)	(0.17)	(0.17)	(0.17)	(0.08)	(0.08)	(0.08)	(0.07)	(0.08)
Time FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Observations	230,971	230,971	230,971	230,971	230,971	288,594	288,594	288,594	288,594	288,594
ln L	-3,420	-3,387	-3,447	-3,446	-3,372	-11,081	-11,073	-11,079	-11,079	-11,071

Notes: This table displays results from hazard models in equation (2.1) for bioequivalence approval and market exit. Estimation is implemented through maximum likelihood. All specifications include time fixed effects. Standard errors in parentheses are clustered at molecule level. *p<0.10, **p<0.05, ***p<0.01.

Table 2.4: Effects of Quality Regulation on Market Structure: Number of Drugs

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Dep. var.: log(1 + Number of drugs)							
	All	Innovator	Branded generics			Unbranded generics		
			All	BE	Non-BE	All	BE	Non-BE
Panel A: Average effects								
Share of market under regulation	-0.29***	-0.10	-0.30***	0.59***	-0.43***	-0.29***	0.61***	-0.41***
	(0.07)	(0.07)	(0.05)	(0.15)	(0.07)	(0.07)	(0.11)	(0.08)
R^2	0.95	0.94	0.96	0.70	0.96	0.92	0.64	0.92
Panel B: Heterogeneity by baseline market size								
Share of market under regulation × Low revenue	-0.44***	-0.21***	-0.41***	0.20	-0.46***	-0.43***	0.32***	-0.43***
	(0.08)	(0.08)	(0.07)	(0.16)	(0.08)	(0.09)	(0.12)	(0.09)
Share of market under regulation × High revenue	-0.16**	-0.00	-0.20***	0.92***	-0.40***	-0.17**	0.85***	-0.39***
	(0.08)	(0.07)	(0.06)	(0.19)	(0.08)	(0.09)	(0.14)	(0.09)
R^2	0.95	0.95	0.96	0.73	0.96	0.92	0.66	0.92
Pre-regulation average	31.25	3.43	17.36	0.10	17.26	10.45	0.01	10.45
Observations	12,576	12,576	12,576	12,576	12,576	12,576	12,576	12,576
Market FE	Y	Y	Y	Y	Y	Y	Y	Y
Month FE	Y	Y	Y	Y	Y	Y	Y	Y

Notes: Each column in this table is a regression of the log number of drugs in a segment on the policy roll-out variable constructed using the first decree deadline. Panel B provides results by baseline revenue. Markets are classified as having a low or high revenue according to the average level of the variable in 2010 relative to the median across markets in that year. Clustered standard errors in parentheses. ***p<0.01, **p<0.05, *p<0.1.

Table 2.5: Effects of Quality Regulation on Market Structure: Number of Laboratories

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Dep. var.: log(1 + Number of Laboratories)							
	All	Innovator	Branded generic			Unbranded generic		
			All	BE	Non-BE	All	BE	Non-BE
Panel A: Average effects								
Share of market under regulation	-0.15***	-0.01	-0.13***	0.52***	-0.19***	-0.18***	0.55***	-0.25***
	(0.04)	(0.02)	(0.04)	(0.12)	(0.03)	(0.06)	(0.09)	(0.06)
R^2	0.93	0.96	0.96	0.71	0.96	0.92	0.65	0.92
Panel B: Heterogeneity by baseline market size								
Share of market under regulation × Low revenue	-0.26***	-0.05*	-0.23***	0.21	-0.24***	-0.30***	0.31***	-0.26***
	(0.05)	(0.03)	(0.05)	(0.13)	(0.05)	(0.07)	(0.11)	(0.08)
Share of market under regulation × High revenue	-0.06	0.03**	-0.05	0.79***	-0.14***	-0.09	0.75***	-0.24***
	(0.05)	(0.01)	(0.04)	(0.15)	(0.04)	(0.07)	(0.12)	(0.07)
R^2	0.96	0.99	0.98	0.88	0.98	0.95	0.89	0.90
Pre-regulation average	10.63	0.96	6.85	0.08	6.83	5.64	0.01	5.63
Observations	12,576	12,576	12,576	12,576	12,576	12,576	12,576	12,576
Market FE	Y	Y	Y	Y	Y	Y	Y	Y
Month FE	Y	Y	Y	Y	Y	Y	Y	Y

Notes: Each column in this table is a regression of the log number of firms that are active in a segment on the policy roll-out variable constructed using the first decree deadline. Panels B provides results by pre-reform revenue. Markets are classified as having a low or high revenue according to the average level of the variable in 2010 relative to the median across markets in that year. Clustered standard errors in parentheses. ***p<0.01, **p<0.05, *p<0.1.

Table 2.6: Effects of Quality Regulation on Drug Prices

	(1)	(2)	(3)	(4)
	Dep. var.: Drug Price Index ($\hat{P}_{mt}$)			
	All drugs	Innovator	Generic	
			Branded	Unbranded
Panel A: Average effects				
Share of market under regulation	0.099**	0.032	-0.007	0.140***
	(0.049)	(0.030)	(0.055)	(0.048)
R^2	0.99	1.00	0.99	0.99
Panel B: Heterogeneity by baseline market size				
Share of market under regulation × Low revenue	0.260***	0.072*	0.053	0.183***
	(0.075)	(0.037)	(0.066)	(0.059)
Share of market under regulation × High revenue	-0.037	0.008	-0.053	0.089
	(0.050)	(0.037)	(0.059)	(0.062)
R^2	0.992	0.996	0.992	0.995
Panel C: Decomposition of price effects				
Dep. var.: Contribution of changes in prices ($\hat{P}_{PC}$)	0.074***	0.012	0.009	0.129***
	(0.023)	(0.021)	(0.023)	(0.047)
R^2	0.64	0.67	0.62	0.67
Dep. var.: Contribution of changes in market shares ($\hat{P}_{RW}$)	0.006	0.017	0.018	0.004
	(0.034)	(0.044)	(0.034)	(0.009)
R^2	0.47	0.50	0.78	0.45
Dep. var.: Contribution of correlation between market shares and prices ($\hat{P}_{CS}$)	-0.002	0.007	-0.042	0.001
	(0.010)	(0.014)	(0.031)	(0.008)
R^2	0.47	0.53	0.44	0.31
Dep. var.: Contribution of drug entry ($\hat{P}_{E}$)	0.023*	0.035	0.011	0.002
	(0.014)	(0.034)	(0.024)	(0.004)
R^2	0.54	0.49	0.66	0.53
Dep. var.: Contribution of drug exit ($\hat{P}_{X}$)	-0.003	-0.039*	-0.003	0.003**
	(0.003)	(0.020)	(0.007)	(0.001)
R^2	0.27	0.35	0.60	0.23
Observations	12,576	9,634	9,903	6,481
Market FE	Y	Y	Y	Y
Month-Type FE	Y	Y	Y	Y

Notes: Panel A displays regressions of share-weighted logged prices for each molecule on the policy roll-out variable constructed using the first decree deadline. The average is taken over all drugs within each market. Panel B provides results by baseline market size. Markets are classified as having a low or high revenue according to their average revenue in 2010 relative to the median revenue across markets in 2010. Panel C displays results for each component of the decomposition of price changes in equation (2.5). Each coefficient in Panel C comes from a separate regression of the component indicated in the left for the drug type indicated in the top row on the policy roll-out variable constructed using the first decree deadline. Clustered standard errors in parentheses. ***p<0.01, **p<0.05, *p<0.1.

Table 2.7: Effects of Quality Regulation on Drug Market Shares and Sales

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
	Panel I: Dep. var.: Market share					*Panel II:* Dep. var.: log(1 + Sales)					
	Innovator	Generic				All	Innovator	Generic			
		Branded			Unbranded			Branded			Unbranded
		Total	BE	Non-BE				Total	BE	Non-BE	
Panel A: Average effects											
Share of market under regulation	0.04	-0.04	0.10***	-0.14***	-0.00	-0.23*	-0.11	-0.48*	2.92***	-1.22***	-0.08
	(0.03)	(0.03)	(0.03)	(0.03)	(0.02)	(0.12)	(0.18)	(0.25)	(0.67)	(0.36)	(0.23)
R^2	0.91	0.93	0.53	0.86	0.96	0.97	0.97	0.94	0.64	0.90	0.95
Panel B: Heterogeneity by baseline market size											
Share of market under regulation × Low revenue	0.08**	-0.02	0.04	-0.07	-0.05	–0.37**	-0.17	-0.54**	1.55**	-1.04**	-0.76**
	(0.04)	(0.03)	(0.04)	(0.04)	(0.04)	(0.15)	(0.23)	(0.23)	(0.76)	(0.47)	(0.36)
Share of market under regulation × High revenue	0.02	-0.06*	0.15***	-0.21***	0.04**	-0.12	-0.06	-0.44	4.06***	-1.37***	0.49**
	(0.03)	(0.03)	(0.03)	(0.04)	(0.02)	(0.13)	(0.19)	(0.38)	(0.80)	(0.39)	(0.23)
R^2	0.92	0.93	0.55	0.86	0.96	0.97	0.97	0.94	0.66	0.90	0.95
Pre-regulation average	0.19	0.55	0.00	0.55	0.26	-	-	-	-	-	-
Observations	12,576	12,576	12,576	12,576	12,576	12,576	12,576	12,576	12,576	12,576	12,576
Market FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Month FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y

Notes: Columns (1) through (5) in this table is a regression of the market share of a segment on the policy roll-out variable constructed using the first decree deadline. Columns (6) through (11) display regressions of logged sales of a segment on the policy roll-out variable constructed using the first decree deadline. Panel B provides results by baseline revenue. Markets are classified as having a low or high revenue according to the average level of the variable in 2010 relative to the median across markets in that year. Clustered standard errors in parentheses. ***p<0.01, **p<0.05, *p<0.1.

Table 2.8: Effects of Quality Regulation on Market Structure: Drug Quality

	(1)	(2)	(3)	(4)
	Drug adverse effects			Drug
	Admissions	Hospital Days	Surgeries	recalls
Panel A: Average effects				
Share of market under regulation	-0.023	-0.120	-0.000	0.001
	(0.023)	(0.112)	(0.000)	(0.001)
R^2	0.849	0.869	0.142	0.223
Panel B: Heterogeneity by baseline market size				
Share of market under regulation × Low revenue	-0.072	-0.235	-0.000	0.003
	(0.071)	(0.224)	(0.000)	(0.003)
Share of market under regulation × High revenue	0.022	-0.016	-0.000	-0.001
	(0.024)	(0.014)	(0.000)	(0.002)
R^2	0.850	0.871	0.142	0.224
Pre-regulation average	0.073	0.132	0.000	0.000
Observations	568	568	568	1021
Market FE	Y	Y	Y	Y
Month FE	Y	Y	Y	Y

Notes: Each column in this table is an outcome related to drug quality on the policy roll-out variable constructed using the first decree deadline, as in equation (2.6). Outcomes are constructed as the ratio of the variable of interest over drug sales measured in thousands of daily doses. Columns (1) through (3) are related to adverse health effects, whereas Column (4) is related to drug recalls, and in particular include all recalls. Panel B provides results by baseline revenue. Markets are classified as having a low or high revenue according to the average level of the variable in 2010 relative to the median across markets in that year. Clustered standard errors in parentheses. ***p<0.01, **p<0.05, *p<0.1.

CHAPTER 3

VERTICAL INTEGRATION BETWEEN HOSPITALS AND INSURERS

3.1 Introduction

The recent trend towards vertical integration in health care markets has become a major concern for policymakers and researchers.[1] There has been speculation about whether vertical integration will reduce health care costs through better management and cost control, or increase market power of integrated firms and induce exclusionary practices towards rival firms (VOX, 2017). Yet, there is limited empirical work informing this debate, at least partly due to a lack of compelling settings and data (Gaynor et al., 2015). The wave of hospital mergers in the U.S. during the last two decades motivated substantial research on horizontal mergers in health care (Gaynor and Town, 2011; Dafny, 2014; Gowrisankaran et al., 2015). However, such evidence is not informative about the effects of vertical integration, since the trade-offs associated with them differ.

The effects of vertical integration on equilibrium outcomes are theoretically ambiguous. The main arguments in favor of vertical integration involve solving the double marginalization problem, and aligning incentives within the vertical chain to induce the efficient use of resources (Spengler, 1950; Williamson, 1971; Grossman and Hart, 1986). However, vertical integration also grants market power to integrated firms and may induce foreclosure, which could harm consumers (Hart et al., 1990; Ordover et al., 1990). In this line, integrated firms may find it profitable to increase prices to rivals to steer consumers to their related partners. The extent to which this distortion arises in insurance and health care markets, and whether it outweighs the potential benefits of vertical integration are empirical questions that have received limited attention.

To study the equilibrium effects of vertical integration between insurers and hospitals,

1. A recent event was the acquisition of Aetna by CVS in 2018. There are several examples of recent vertical mergers in the U.S.: Anthem acquired Simply Healthcare in Florida, United Health acquired DaVita and Monarch Healthcare in California, and Highmark acquired West Penn Allegheny System in Pennsylvania. Outside the U.S., Aetna acquired Indian Health Organization, and Cigna announced a similar strategy to enter the Indian and Chinese markets. Moreover, United Health recently bought the largest health care player in Brazil, Amil, and agreed to acquire Banmédica in Chile. Bossert et al. (2014) provides a discussion of increasing concentration in Latin American health care markets.

we develop and estimate a model for these markets. We use this model to identify incentives for integrated firms to steer demand towards partners, through which vertical integration affects equilibrium outcomes. To quantify the implications of vertical integration, we estimate the model primitives using detailed administrative data from Chile and use these estimates to study the counterfactual policy of banning vertical integration. Vertical integration induces higher negotiated hospital prices by integrated hospitals and reduces overall welfare in our setting.

The Chilean private health care market provides a unique setting for studying the effects of vertical integration. The market displays an oligopolistic structure where a small number of hospitals and insurers compete, and vertically integrated firms account for almost half of hospital admissions. Consumers choose among a variety of plans and, whenever they require health care, choose hospitals from their network and pay their share of the bill. The market features a stable set of hospitals and insurers, complete networks and limited variation in plan design, which ease the study of the effects of vertical integration. For this setting, we have access to detailed individual-level panel administrative data for 2013–2016. These data contain linked hospital claims, insurance plan choices, hospital prices, consumer out-of-pocket payments, plan premiums, and consumer demographics.

Our analysis starts by describing differences in market outcomes for integrated and non-integrated firms. The full price of admissions at an integrated hospital is 7.9% lower when the patient comes from an integrated insurer, and patients from integrated insurers pay 23% less out-of-pocket than patients from non-integrated insurers. Additionally, we show that new enrollees of integrated insurers are 10% more likely to visit hospitals integrated with that insurer, despite facing unrestricted networks. These facts suggest that vertical integration affects prices and choices in this market. However, vertical integration affects the behavior of all firms in equilibrium, which complicates the interpretation of these effects. The limitations of this analysis motivate the structural approach we adopt in the rest of the paper to evaluate the effects of vertical integration.

We develop a model that captures the main features of vertical integration in health care. The vertical structure of this market is non-standard and induces particular incentives. In a typical vertical market, downstream firms sell products acquired from upstream firms.

In health care markets, consumers acquire an insurance plan that gives them an option to access upstream hospitals and purchase services directly from them at a given price schedule (Capps et al., 2003). This structure creates incentives for integrated insurers to use negotiated hospital prices to steer patients towards their integrated partners.

We model the interaction between hospitals, insurers and consumers as a four-stage game. In the first stage, hospitals and insurers engage in bilateral bargaining over hospital prices. In the second stage, insurers set premiums taking hospital prices as given. In the third stage, households choose an insurance plan. Finally, in the fourth stage, consumers' health risk is realized and, upon becoming sick, they choose a hospital within a choice set that depends on their insurance plan. This model is broadly similar to leading models in the literature (Gowrisankaran et al., 2015; Prager, 2016; Ho and Lee, 2017), but connects those to recent developments in the study of vertical markets (Lee, 2013; Crawford et al., 2018). In particular, our model accommodates vertical integration between insurers and hospitals, which allows for analyzing its effects. Integrated firms set both hospital prices and premiums to maximize their joint profits.

The main insights from our model are two mechanisms that emerge in equilibrium, which we study and quantify. Integrated firms have incentives to increase hospital prices to steer demand from rival hospitals and insurers towards their related partners. First, integrated hospitals have incentives to steer demand to their integrated insurers by negotiating higher hospital prices with rival insurers, which we call the *enrollee-steering* effect. This effect has been previously referred to as *raising your rival's cost* (Salop and Scheffman, 1983). Second, integrated insurers have incentives to steer demand to their integrated hospitals by negotiating higher prices with rival hospitals, which we call the *patient-steering* effect. To our knowledge, this effect has not been studied previously, as it stems from the aforementioned non-standard structure of the health care market. Both patient- and enrollee-steering effects can be broadly identified with partial foreclosure, as their limit case leads to downstream and upstream exclusion (Hart et al., 1990).

We exploit our data and variation in vertical integration to estimate the model. First, we estimate discrete choice demand models for hospitals and plans using data on hospital and plan choices, prices, premiums, and consumer demographics. We exploit our individual-

level data to control for unobservables, using rich sets of fixed effects to deal with price and premium endogeneity concerns. Our hospital demand estimates indicate that consumers are price sensitive and value the proximity of hospitals. Moreover, our plan demand estimates imply that consumers trade-off premiums and the overall quality of the network offered by insurers when choosing plans. Second, we estimate the parameters of the supply side of the model, namely marginal costs and bargaining weights. We develop a GMM estimator based on three sets of moments related to (i) optimality conditions for premium setting, (ii) firms' profitability as measured by financial statements, and (iii) orthogonality conditions between instruments and unobservable determinants of hospital costs. Using the estimated model, we show that enrollee- and patient-steering effects are empirically relevant in this setting.

Using our structural estimates, we quantify the effects of banning vertical integration on prices, market shares and welfare. Banning vertical integration increases total welfare in our setting, largely driven by a reduction in the gap between prices of integrated and non-integrated hospitals. This change in prices lowers insurer costs, which is partially passed through to consumers. The welfare effect of banning vertical integration is $146 million per year, which combines a decrease in hospital profits with increases in both insurer profits and consumer surplus. Underlying these welfare effects, we show that this policy involves substantial changes in the distribution of consumers across hospitals and insurers. This analysis does not account for potential cost efficiencies associated with vertical integration. We study a wide range of such efficiencies within integrated firms and show that while the welfare effects of banning vertical integration become smaller, they remain positive. However, vertical integration is welfare enhancing if cost efficiencies of the same magnitude within an integrated hospital were shared with rival insurers.

The strength of the steering incentives depends on consumer price sensitivity. More price sensitive consumers make this mechanism more effective and allow integrated firms to take advantage of this source of market power. However, more price sensitive consumers also make the market more competitive and induce prices to decrease. We explore the role of consumer price sensitivity for the effects of vertical integration. We find the the effect of banning vertical integration on consumer surplus depends on whether consumers are

more sensitive to prices then to premiums, or the converse. When consumers respond more to hospital prices, integrated firms can steer patients by increasing prices to rival insurers which generates a consumer surplus loss. Alternatively, when consumers are more responsive to premiums then integrated insurers steer enrollees by lowering premiums, which increases consumer surplus.

Our paper contributes to three branches of the literature. First, we contribute to the empirical literature on the effects of vertical integration (Chipty, 2001; Hastings, 2004; Hortaçsu and Syverson, 2007; Atalay et al., 2014), which is still unsettled (Bresnahan and Levin, 2012). Closest to this paper is the work by Crawford et al. (2018) on vertical integration in multichannel television markets. Although our model is similar to theirs, the vertical structure of the health market we study creates new forces. We study a non-standard vertical market in which consumers interact with both upstream hospitals and downstream insurers, and it turns out that this feature has relevant implications for the effects of vertical integration. In effect, we find that demand steering incentives have significant implications on outcomes, which are not present in the vertical relationships between content providers and TV broadcasters where foreclosure is the predominant force.

Second, we contribute to the literature on competition in health care markets by providing new evidence on the effects of vertical integration. Within the vast literature on competition in health care (Gaynor and Town, 2011; Gaynor et al., 2015), the analysis of mergers focuses mostly on horizontal mergers between hospitals (Dafny, 2009; Dafny et al., 2012; Gowrisankaran et al., 2015; Lewis and Pflum, 2017; Craig et al., 2018; Dafny et al., 2018) and between insurers (Chorniy et al., 2016). Despite the increasing number of vertical mergers, work on vertical integration in health care is limited, and we contribute by expanding it. An exception is Diebel (2018), who studies the effect of vertical integration on foreclosure using the approach of Lewis and Pflum (2015).[2]

Finally, we contribute to the empirical literature on bargaining in vertical markets by studying the role of steering effects. The bargaining literature has covered a variety of

2. Additionally, there is some literature on vertical contracting between insurers and hospitals, although it focuses mostly on the possibility of foreclosure (Gal-Or, 1997; Gal-Or, 1999).

research topics such as vertical integration and foreclosure (Crawford et al., 2018), horizontal mergers (Gowrisankaran et al., 2015), insurer competition (Ho and Lee, 2017), bundling (Crawford and Yurukoglu, 2012), price discrimination (Grennan, 2013), and network design and formation (Ho, 2009; Prager, 2016; Ghili, 2017; Ho and Lee, 2018; Liebman, 2018). Our paper focuses on the estimation of price distortions associated with vertical integration and their welfare implications.

Overall, we provide a theoretically grounded approach for quantifying the effects of vertical integration, that can inform antitrust analysis. We accommodate vertical integration in a bargaining framework that identifies demand steering incentives that distort negotiated hospital prices, and propose an approach to empirically assess the welfare consequences of banning vertical integration. Importantly, our approach can be used to study vertical integration in other industries where downstream consumers also interact with upstream firms. For example, our framework could be applied to analyze the role of Pharmacy Benefit Managers (PBM) in the U.S. market.

The remainder of the paper is organized as follows. Section 3.2 describes the institutional framework and data from the Chilean health market, and provides descriptive evidence for the role of vertical integration. Section 3.3 proposes a model of competition and bargaining in health markets. Then, Section 3.4 discusses the identification and estimation of the parameters of the model and the main results from estimation. Section 3.5 develops a welfare analysis of banning vertical integration in this market. Finally, Section 3.6 concludes.

3.2 Institutions and Data

3.2.1 The Chilean Health Care Market

Health Insurance. The insurance system in Chile combines public and private provision.[3] The public insurer is the National Health Fund (*Fondo Nacional de Salud*, FONASA), a pay-as-you-go system financed by individual contributions and the government. The private sector has a small number of insurers (*Instituciones de Salud Previsional*, ISAPREs) that

3. Our description of the Chilean insurance markets borrows from Duarte (2012a) and Atal (2015).

compete in a regulated environment. FONASA serves around 70% of the population and ISAPREs serve roughly 15% of it, while the remaining 15% of is either enlisted in the army or uninsured (Bitrán et al., 2010b; Duarte, 2012a). Insurance is mandatory for those in the labor market. Workers entering the labor force for the first time must automatically enroll in FONASA. After a month, they must actively choose to stay in FONASA or switch to a private insurer. Hence, workers, and then retirees, must contribute 7% of their taxable income to the public system or to purchase a private plan with a premium of at least that amount, with a maximum of $264 per month.[4]

Private and public plans differ in premiums, networks, coinsurance structure, coverage caps, risk pricing, and selection. In both sectors, plans offer separate coinsurance rates for inpatient and outpatient care. Unlike in the U.S., plans do not include deductibles and out-of-pocket maximums. For details on the interaction between the public and private systems, see Appendix C.2.1.

Private Health Insurance. Private insurers were introduced in 1981, in a context of broader privatization and market-oriented policies. Private insurers developed a variety of contracts to attract consumers, mainly from the top of the income distribution. Contracts in the private market are mostly individual arrangements among insurers and consumers. Contracts are annual, and once consumers choose one, they must remain under it for at least one year. After that period, consumers are allowed to switch to a different plan in the private or public sector. We focus on the six *open* insurers available to all workers, which account for 96% of the private market. We denote these insurers by m_1–m_6. Insurers m_1 and m_6 are horizontally integrated, which motivates treating them as a single firm for our supply side analysis.[5]

Insurance plans are regulated and are composed of the following elements. First, they have a monthly premium that is age- and gender-specific. Second, insurers may reject consumers based on their health status, although plans have guaranteed renewability by

4. All monetary amounts are measured in U.S. dollars using the exchange rate on December 30, 2014.

5. We account for the distinction between m_1 and m_6 when relevant. In particular, when estimating plan demand we allow consumers to hold different preferences for m_1 and m_6.

which policyholders can always re-enroll on their plan regardless of changes in health status. Third, each plan has separate coinsurance rates for inpatient and outpatient care. Fourth, plans offer either unrestricted open networks or tiered networks.[6] Hospitals cannot deny health care to patients, and therefore all consumers have access to all hospitals, although they may have zero coverage from their plan. For further details regarding the regulation of insurance plans, see Appendix C.2.2.

Hospitals. The health care system combines public and private provision. The public hospital network is broader than the private one, with 185 public hospitals compared to 85 private hospitals in 2012 (Clínicas de Chile, 2012). We focus on the interaction between private insurers and private hospitals, given the private and public sectors are mostly segmented. Private insurers primarily cover admissions to private hospitals, whereas the public insurer mostly covers admissions to public hospitals. In fact, 97% of private insurer payments are to private hospitals, whereas only 3% are to public hospitals (Galetovic and Sanhueza, 2013). An important feature of this market is price transparency, as consumers are often able to obtain price quotes before choosing a hospital.

For our analysis, we focus on a particular segment of the market. Geographically, we focus on the city of Santiago, which is the largest health care market and where more than a third of private hospitals and around half of the capacity is located (Galetovic and Sanhueza, 2013). Additionally, we only consider inpatient care, which represents more than half of health care expenditure. This segment is comprised of remarkably fewer players than the outpatient care sector and therefore strategic concerns associated with vertical integration are more relevant in it. We focus on the 12 main hospitals in Santiago, and denote them by h_1–h_{12}. We discuss this selection in Section 3.2.2.

6. Unrestricted network plans provide the same coverage for all hospitals. Tiered networks offer differentiated coverage across sets of private hospitals, as PPO plans in the U.S.. Few plans offer restricted networks, as HMO plans in the U.S., and they are rarely observed in the data and not offered publicly. We do not consider them in our analysis.

Vertical Integration. Vertically integrated firms are structured into *holdings* that control both insurers and hospitals.[7] Insurers and hospitals have strong vertical linkages: 48% of the private hospital capacity was controlled by holdings that also owned an insurer in 2012 (Galetovic and Sanhueza, 2013). There are two integrated insurers, each of which is integrated with three hospitals in Santiago. In particular, insurer m_1 is integrated with hospitals h_4 , h_7 and h_{11}, whereas insurer m_3 is integrated with hospitals h_2, h_3 and h_8.[8] Importantly, vertically integrated hospitals remain open to patients from all insurers in the market.

3.2.2 Data

Administrative Records

We exploit administrative data collected by the regulator of the insurance market (*Superintendencia de Salud*, SS). Insurers must report data on individual claims. These data cover every health service provided to a private plan policyholder in 2013–2016, including financial and medical attributes along with consumer, plan and hospital identifiers. We complement these data with list prices paid by the public insurer for each service. Additionally, we access data on all private plans offered in 2013–2014. We have data on plan premiums, copayment rates, preferential networks, coverage caps, and availability in the market over time. Furthermore, we can match plans and their enrollees and observe basic demographics of policyholders and their dependents.[9]

We restrict our analysis to the 12 hospitals with highest market share, which account

7. The Chilean law forbids insurers from having ownership and control over hospitals. However, the law does not forbid a third party to own insurers and hospitals simultaneously. Hence, firms have circumvented the regulation by establishing vertical relations through holdings that own both insurers and hospitals.

8. We define vertically integrated firms as those for which the holding owns more than 50% of the hospital and more than 98% of the insurer. Information on vertical linkages is based on Copetta (2013). The case of m_2 is unclear, as the holding only controls 50% of the hospital. We do not consider this hospital-insurer pair as vertically integrated. The list of vertical linkages we provide is limited to the market we examine. Private insurers also hold vertical linkages with hospitals in other geographic markets (Tobar et al., 2012; Copetta, 2013; Galetovic and Sanhueza, 2013). We assume that geographic markets are independent and therefore focus only on linkages between insurers and hospitals in Santiago.

9. When appropriate, we distinguish between policyholders, who choose and pay for insurance, and the full set of enrollees, which also cover policyholders' dependents.

for 76% of the admissions in the data. The remaining hospitals are relatively small, and we group them into the outside option along with public hospitals. All these hospitals receive patients from all insurers in the market. Figure C.1 displays their locations in the market.

Admissions. We exploit administrative claims data to construct hospital admissions. Using claim dates and patient identifiers, we identify unique medical episodes of inpatient care which we label as *admissions*. The data contain detailed financial and medical information for each admission. Financial information includes the hospital charges, insurer coverage, and consumer copayment. Medical information includes the diagnosis and the list of claims for different services provided by the hospital. We code admissions to diagnoses using ICD-10 codes, resulting in medical episodes that cover 16 diagnoses groups.[10] These diagnoses account for 90% of admissions and 92% of hospital revenue. Finally, we combine these data with plan attributes and consumer covariates, such as age, income, gender, and the number of dependents. We describe this process in detail in Appendix C.1. The resulting dataset contains 641,392 admissions for 2013–2016.[11]

Insurance Plans. Our administrative data on insurance plans contain detailed characteristics of 68,625 coded plans. The proliferation of plan codes is due to incentives faced by insurers due to guaranteed-renewability requirements (Atal, 2015). However, consumers face a much smaller choice set when choosing insurance. By grouping plans that are identical in financial attributes, we reduce the number of distinct plans to 4,358 over our four years of data.[12] As insurers offer different plans by gender, age, and household structure, consumers choose on average among 1,603 plans, of which only 43 have a market share above 0.5%. We provide more details on how we construct plan choice sets for demand estimation in Section 3.4.3.

10. The list of diagnosis covers infections and parasites, neoplasms, blood diseases, endocrine diseases, nervous system diseases, ocular diseases, ear diseases, circulatory diseases, respiratory diseases, digestive diseases, skin diseases, musculoskeletal diseases, genitourinary diseases, pregnancy, perinatal treatments, and congenital malformation.

11. We face limitations that preclude us from using outpatient claims data. The main issue is that those services are provided by entities typically different from hospitals (mostly physicians groups or other firms such as laboratories). Thus, tracking the identity of these providers is not feasible.

12. We group plans by insurer, coverage rates, network structure and deciles in the base premium.

Descriptive Statistics

Table 3.1-A describes policyholders. The average policyholder is 40 years old, has a monthly income of $1,600, and pays $160 in insurance premiums every month.[13] There is substantial variation in the household composition of policyholders, with 34% and 22% being single males and single females respectively, while the remaining 43% of the enrollees have at least one dependent.

Table 3.1-B displays plan attributes where the coverage rates are, on average 85% and 72% for inpatient and outpatient care. Moreover, 87% of plans have a coverage cap and 86% offer at least one preferential hospital. Table 3.1-C summarizes the market shares and monthly premiums.[14] We document substantial variation in premiums across insurers, with the difference between the highest and the lowest average premium being 66% of the average premium. Furthermore, we observe significant variation in premiums within an insurer, which is partly explained by the ability of insurers to adjust premiums by policyholder gender, age and number of dependents.

Table 3.2-A describes admissions, and shows that the average admission bill is $3,790, of which the patient pays almost a third. However, there is significant dispersion in total bill and insurer coverage. Nearly 38% of admissions are at preferential hospitals in the plan's tiered network. The average patient is 37 years old, although the data span from infants to elderly. Finally, about 70% of the patients have dependents, and 14% and 17% are single males and females, respectively.

Table 3.2-B documents hospital market shares and prices. Four hospitals have market shares between 10% and 13%, while the rest have market shares of 5% or lower. The outside option—minor private hospitals and all public ones—has a market share of 24%. There is substantial dispersion in total bill across hospitals. For example, hospitals h_1 and h_6 charge average prices more than double those charged by h_4 and h_{11}. This price dispersion is explained by differences in location, infrastructure, real and perceived quality, among

13. This contribution is on average slightly larger than the mandatory 7%, because additional contributions are allowed.

14. Market shares in our sample closely track national market shares. According to the regulator, insurer market shares in 2015 were 19.3% for m_1, 16.2% for m_2, 19.6% for m_3, 21.2% for m_4, 16.4% for m_5, 3.9% for m_6 and 3.3% for other insurers (Superintendencia de Salud, 2015).

others.

Finally, Table 3.3-A displays the breakdown of hospital admissions by insurer. Among all integrated hospitals, integrated insurers are the dominant source of admissions, representing between 40% and 70% of the hospitals' admissions. Nevertheless, all integrated hospitals receive a substantial share of patients from non-integrated insurers.

3.2.3 Descriptive Evidence on Vertical Integration

In this section, we provide descriptive evidence consistent with integrated firms steering demand towards their partners. We focus on how hospital prices and insurer coverage correlate with vertical integration, and on how being enrolled in an integrated insurer affects hospital choice.

Vertical Integration and Payments

To study how vertical integration correlates with admission payments, we exxploit within-hospital variation in admission outcomes for patients insured by integrated and non-integrated insurers. If integrated firms use prices to steer demand towards their partners, we should observe that hospital charges and patient copayments are lower for integrated admissions relative to non-integrated admissions within an integrated hospital.

The estimating equation is:

$$y_{idjh} = \beta VI_{m(j)h} + X'_{ij}\gamma + \tau_d + \eta_{m(j)} + \zeta_h + \varepsilon_{idjh} \tag{3.1}$$

where y_{idjh} is the outcome of interest for patient i admitted for diagnosis d under plan j in hospital h; $VI_{m(j)h}$ is an indicator of whether the insurer m and the hospital h are integrated; and X_{ij} is a vector of controls that includes patient i demographics and plan j attributes. Demographics include gender, age, income, number of dependents, an indicator for being an independent worker, and county of residence. Plan attributes include plan premium, coinsurance rate for inpatient and outpatient admissions, and indicators for whether the plan has a coverage cap and a preferential hospital. We also include prices in the public

system for each admission as a proxy of costs, which we interact with hospital dummies. Moreover, we control for time-invariant heterogeneity by including diagnosis, insurer and hospital fixed effects, denoted by τ_d, $\eta_{m(j)}$ and ζ_h respectively.

Table 3.4 displays results for the full hospital bill, patient out-of-pocket copayment, and insurer coverage.[15] Each column shows estimates using an increasingly broader set of controls, and column (5) is our preferred specification. Estimates in Panel A show that the hospital bill is 7.9% lower for integrated admissions. Moreover, estimates in Panel B show that patient copayments are 23% lower for integrated admissions, whereas estimates in Panel C show that the amount paid by the insurer is 3.9% higher for integrated admissions.

These differences are not sufficient to determine the impact of vertical integration. On the one hand, we can rationalize this gap as an increase in integrated hospital prices to rival insurers to steer demand to their integrated insurer. On the other hand, it could be driven by cost efficiencies and the elimination of double marginalization within integrated firms. Our structural model will distinguish and quantify these two mechanisms.

Integrated Hospitals and Hospital Choices

Vertical integration creates incentives to steer demand towards integrated partners. To test whether the availability of integrated hospitals affects hospital choice, we study the outcomes of policyholders who switched to integrated insurers. The hospital choice set for switchers is constant over time. However, whether a hospital is integrated with a policyholder's insurer changes over time with switches. We exploit that variation to study the role of vertical integration in hospital choice.

Thus, we estimate the following event study regression for the subpopulation of switchers:

$$y_{iht} = \sum_{\tau} \beta_\tau D_{ih\tau} + \alpha_i + \delta_{ht} + \varepsilon_{iht} \tag{3.2}$$

where y_{iht} is an outcome for patient i in hospital h at year t. The main explanatory variables

15. We use the log of the outcomes plus some fixed amount to ensure no zeros in the dependent variable, which is frequent for patient copayments. The results are similar when using the inverse hyperbolic sine transformation of the dependent variable, $y = \log(\widetilde{y} + \sqrt{\widetilde{y}^2 + 1})$, as displayed in Table C.1.

are the indicators $D_{ih\tau} = 1\{h \in \mathcal{H}_{m(is_i)}, \tau = s_i - t\}$, where $\mathcal{H}_{m(is_i)}$ is the set of hospitals integrated with the patient insurer, and s_i is the date at which patient i switched insurers. Each dummy variable indicates whether hospital h is integrated with the patient's insurer, τ periods after year t. The coefficients of interest are β_τ, which measure the effect of changing the integrated status of a hospital on the outcomes of interest τ years after the patient switched insurers. We include patient fixed effects α_i to control for differences in outcomes across patients that are constant through time (e.g., permanent differences in health), and hospital-time fixed effects δ_{ht} to control for differences in outcomes across hospitals and time that are constant across patients (e.g., seasonality in health shocks, quality differences). The coefficient for the year before the patient switches is set to zero.

Policyholders switching to an integrated insurer are more likely to choose hospitals integrated with their insurer. Figure 3.1 displays results from estimating equation (3.2). In particular, Figure 3.1-a shows that when the patient switches insurers, the probability of choosing a hospital integrated with the new insurer increases by almost 10%. Moreover, Figure 3.1-b shows that expenditure in hospitals integrated to the new insurer increases by more than 50% relative to the year before. Both effects remain two years after the patient switches insurers.

These results should be interpreted with caution. First, patients may switch insurers precisely to improve their access to integrated hospitals. Second, the results do not imply that hospital admissions or expenditure increased, but may reflect reallocation from non-integrated to integrated hospitals. Finally, this analysis does not identify which aspect of the chosen plan affects hospital choice, which could be prices, networks, or other.

3.3 A Model of Bargaining between Hospitals and Insurers

We model the market as a four-stage game. First, hospital prices are set for each insurer-hospital pair in the market. If the hospital-insurer pair is integrated, the hospital prices are set by joint profit maximization, whereas if firms are not integrated, they engage in negotiation. Second, insurers determine plan premiums taking hospital prices as given. Third, households choose an insurance plan based on premiums and the expected utility

from the plan network of health care services. Fourth, consumers' health risk is realized. Upon becoming sick, consumers choose a hospital given hospital characteristics and out-of-pocket payment as determined by their insurance plan.

Our model builds upon recent work in the literature (Gowrisankaran et al., 2015; Prager, 2016; Ho and Lee, 2017), though differing in two critical aspects. First, central to our analysis, we allow for vertical integration, which alters incentives in upstream and downstream equilibrium prices. Second, we assume that insurers set premiums taking hospital prices as given, which is different from Ho and Lee (2017) who assume those pricing decisions to be simultaneous.[16]

3.3.1 Setup

Each insurer $m \in \mathcal{M}$ offers a menu of insurance plans denoted by $\mathcal{J}_m$, where $\mathcal{M}$ is the set of insurers. Each plan $j \in \mathcal{J}_m$ charges a premium ϕ_j for a given coverage structure and a specific hospital network. Offered hospital networks are unrestricted, but plans may offer tiered networks that differ in coinsurance rates across tiers. Throughout the paper, we assume an exogenous and fixed set of available plans, keeping their coinsurance rates and network structure constant.

Hospitals $h \in \mathcal{H}$ provide health services. Each hospital h charges a price p_{mh} to patients enrolled with insurer m. Patient out-of-pocket payments are a fraction of the hospital price, determined by the patient's plan coverage at the hospital. Given that hospital networks are unrestricted and hospitals cannot refuse health care to patients, consumers can choose any hospital in the market regardless of their plan. We also allow hospitals to organize into systems which we denote by $s \in \mathcal{S}$, where each system s consists of a set of hospitals $\mathcal{H}_s \subset \mathcal{H}$. Hospital systems negotiate the prices of their hospitals as a single entity but are allowed to set different prices for each hospital-insurer.

16. We allow insurers to set premiums after observing hospital prices, as insurance contracts in Chile are signed throughout the year. In contrast, hospital contracts have fixed lengths and are renegotiated only once per term.

Timing of the Game

Our model consists of a four-stage game with the following timing:

1. Hospital prices $\boldsymbol{p}$ are determined either by bilateral negotiation between insurers and hospitals or by joint profit maximization if the hospital and the insurer are integrated.

2. Profit maximization of the insurers determines the vector of plan premiums $\boldsymbol{\phi}$, taking hospital prices $\boldsymbol{p}$ as given.

3. Households choose an insurance plan j of insurer m based on premiums and the expected utility from health care services provided by the plan. Household choices determine the aggregate demand for plans, $D_j^M(\boldsymbol{\phi}, \boldsymbol{p})$.

4. Health risk is realized, and consumers choose among hospitals given their attributes and the out-of-pocket expenditure determined by their plan. Consumer choices determine aggregate demand for each hospital h by household type enrolled in each plan j, $D_{hj}^H(\boldsymbol{\phi}, \boldsymbol{p})$.

Note that the timing of the game implies that premiums are set conditional on hospital prices. This allows insurers to respond to out-of-equilibrium offers made in the bargaining stage, which disciplines equilibrium prices.

Hospital Profits

Hospital system $\mathcal{H}_s$ maximizes profits by setting prices with integrated insurers and negotiating prices with non-integrated insurers. Profits for the hospital system s are given by:

$$\pi_s^H(\boldsymbol{\phi}, \boldsymbol{p}) = \sum_{h \in \mathcal{H}_s} \sum_{m \in \mathcal{M}} \sum_{j \in \mathcal{J}_m} D_{hj}^H(\boldsymbol{\phi}, \boldsymbol{p})(p_{mh} - c_{mh}^H) \tag{3.3}$$

where $D_{hj}^H(\boldsymbol{\phi}, \boldsymbol{p})$ is health care demand, c_{mh}^H is the hospital marginal cost and p_{mh} is the price charged by hospital h to patients enrolled with insurer m. For simplicity, we assume scalar costs and prices for each hospital-insurer pair. In the empirical application, we use

condition-specific weights to allow the hospital prices and costs to vary across diagnosis and consumer characteristics.

Insurer Profits

Insurer m maximizes expected profits by choosing plan premiums for the set of offered plans, conditional on hospital prices. Profits for the insurer m are given by:

$$\pi_m^M(\boldsymbol{\phi}, \boldsymbol{p}) = \sum_{j \in \mathcal{J}_m} D_j^M(\boldsymbol{\phi}, \boldsymbol{p})(\phi_j - c_j^M) \tag{3.4}$$

where ϕ_j is the premium and c_j^M is the expected marginal cost per household enrolled in plan j. We abstract from insurer administrative costs. Hence, the insurer expected marginal cost is the fraction of the hospital bill covered by the insurer.

Profits for Vertically Integrated Firms

We use the term holding for a firm that owns an insurer and a hospital system. These vertically integrated firms or holdings set both hospital prices and plan premiums to maximize the joint profits of their related partners. In doing so, these firms internalize the effects of changes in premiums and hospital prices in steering demand towards their affiliated insurer and hospitals.

The profits of integrated firm $a \in \mathcal{V}$ that controls hospital system $s(a)$ and insurer $m(a)$ are:

$$\pi_a^{VI}(\boldsymbol{\phi}, \boldsymbol{p}) = \pi_{s(a)}^H(\boldsymbol{\phi}, \boldsymbol{p}) + \theta_a \pi_{m(a)}^M(\boldsymbol{\phi}, \boldsymbol{p}) \tag{3.5}$$

where both hospital and insurer profits are as in equations (3.3) and (3.4). The VI weight θ_a scales the objective of the firm, allowing it to differently value profits from health care than those from insurance.[17] This can be justified by differences in regulation between

17. Crawford et al. (2018) justify a similar weighting in their context, as a measure of the extent of the integration.

both markets; by the internal organization of the integrated firm and ability to transfer rents across its insurer and system; or simply by the nature of the contracts written when merging. We allow the weight to differ between integrated systems, and consider $\boldsymbol{\theta} = 1$ as the special case where integrated firms value all rents equally. For ease of exposition, we present the rest of the model for this special case, and reintroduce θ when discussing the supply side estimation in Section 3.4.4.

Bargaining over Hospital Prices

We assume that hospital systems and insurers that are not integrated engage in sequential bargaining over hospital prices as in Collard-Wexler et al. (2017). Thus, the negotiated hospital price, p_{mh}, between non-integrated hospital h and a non-integrated insurer m is given by:

$$p_{mh} = \arg\max_{p_{mh}} \quad (\pi_s^H - \pi_{s\setminus m}^H)^{(1-\lambda_{ms})}(\pi_m^M - \pi_{m\setminus s}^M)^{\lambda_{ms}} \tag{3.6}$$

where $\pi_{s\setminus m}^H$ are the profits for hospital system s upon disagreement with insurer m, and $\pi_{m\setminus s}^M$ are the profits for insurer m upon disagreement with hospital system s. The negotiated price maximizes the Nash product that is the weighted product of the marginal gains from the relationship for each firm. Under the standard assumption of passive beliefs, we keep all other hospital prices constant at their equilibrium level (Horn and Wolinsky, 1988). The parameter $\lambda_{ms} \in (0, 1)$ is the normalized bargaining weight of insurer m relative to hospital system s and measures relative bargaining skill. For instance, if λ_{ms} increases, then the split of surplus skews towards insurer m.

We generalize equation (3.6) to accommodate negotiations between holdings a and b, since integrated and non-integrated firms are particular cases of holdings with and without related firms. The negotiated hospital price, $p_{m(a)h(b)}$, between insurer $m(a)$ and hospital $h(b)$ is given by:

$$p_{m(a)h(b)} = \arg\max_{p_{m(a)h(b)}} \quad (\pi_a^{VI} - \pi_{a\setminus s(b)}^{VI})^{\lambda_{m(a)s(b)}}(\pi_b^{VI} - \pi_{b\setminus m(a)}^{VI})^{(1-\lambda_{m(a)s(b)})} \tag{3.7}$$

where $\pi^{VI}_{a\setminus s(b)}$ are the profits of holding a upon disagreement with hospital system $s(b)$, and $\pi^{VI}_{b\setminus m(a)}$ are the profits of holding b upon disagreement with insurer $m(a)$. As before, the parameter $\lambda_{m(a)s(b)}$ is the normalized bargaining weight between insurer $m(a)$ and hospital system $s(b)$. Notice that the disagreement between two firms from different holdings does not block the agreement between the other related firms of those holdings. For instance, a disagreement between insurer $m(a)$ and hospital system $s(b)$ does not prevent an agreement between insurer $m(b)$ and hospital system $s(a)$.

Vertical Integration and Disagreement Profits. We analyze the components of disagreement profits for vertically integrated firms, as they provide relevant insights regarding the role of vertical integration in the bargaining outcomes.

Profits for holding a upon disagreement with hospital system $s(b)$ are given by:

$$\pi^{VI}_{a\setminus s(b)} \quad = \quad \pi^{H}_{s(a)\setminus s(b)}(\boldsymbol{\phi}, \boldsymbol{p}) + \pi^{M}_{m(a)\setminus s(b)}(\boldsymbol{\phi}, \boldsymbol{p})$$

where $\pi^{H}_{s(a)\setminus s(b)}(\boldsymbol{\phi}, \boldsymbol{p})$ and $\pi^{M}_{m(a)\setminus s(b)}(\boldsymbol{\phi}, \boldsymbol{p})$ are the hospital and insurer profits of holding a upon disagreement with hospital system $s(b)$.

The term $\pi^{H}_{s(a)\setminus s(b)}(\boldsymbol{\phi}, \boldsymbol{p})$ is novel and highlights that integrated holdings have incentives to steer demand towards their hospitals. Indeed, the disagreement between holding a and hospital system $s(b)$ can be beneficial to their related hospital system, $s(a)$. Thus, holding a has incentives to deter enrollees of insurer $m(a)$ from choosing hospitals in system $s(b)$. Unlike non-integrated insurers, holding a internalizes that high hospital prices to rival hospitals may steer demand towards their related hospital system. Therefore, we denote this as the *patient-steering* effect.

Disagreement profits $\pi^{M}_{m(a)\setminus s(b)}(\boldsymbol{\phi}, \boldsymbol{p})$ capture the standard loss in a vertical relationship when the insurer loses a hospital system from its network, making the network less valuable for enrollees. This loss stems from the ensuing decrease in insurance demand for plans offered by insurer $m(a)$, mitigated by the ability of insurers to adjust premiums after disagreements.

Analogously, profits for holding b upon disagreement with insurer $m(a)$ are given by:

$$\pi^{VI}_{b\setminus m(a)} = \pi^{H}_{s(b)\setminus m(a)}(\boldsymbol{\phi}, \boldsymbol{p}) + \pi^{M}_{m(b)\setminus m(a)}(\boldsymbol{\phi}, \boldsymbol{p})$$

where $\pi^{H}_{s(b)\setminus m(a)}(\boldsymbol{\phi}, \boldsymbol{p})$ and $\pi^{M}_{m(b)\setminus m(a)}(\boldsymbol{\phi}, \boldsymbol{p})$ are hospital and insurer profits of holding b upon disagreement with a rival insurer $m(a)$. The usual loss from disagreement is given by $\pi^{H}_{s(b)\setminus m(a)}(\boldsymbol{\phi}, \boldsymbol{p})$, which captures the change in hospital profits when removed from insurer $m(a)$ networks. Unless enough patients leave insurer $m(a)$ following the disagreement and access hospitals in $H_{s(b)}$ through other plans, the hospital system will obtain lower profits under disagreement.

Disagreement profits $\pi^{M}_{m(b)\setminus m(a)}(\boldsymbol{\phi}, \boldsymbol{p})$ capture a novel effect of vertical integration. Under vertical integration, disagreement with rival insurer $m(a)$ may benefit the holding's insurer $m(b)$, as not having hospital system $s(b)$ in the network of $m(a)$ makes that network less valuable and increases demand for insurer $m(b)$. Unlike non-integrated insurers, holding b internalizes that high hospital prices with rival insurers worsen its rival's network and benefit their integrated insurers.[18] Therefore, we refer to this as the *enrollee-steering* effect.

3.3.2 *Equilibrium*

Equilibrium Negotiated Hospital Prices

Negotiated prices between non-integrated hospitals and insurers come from equation (3.6), and solve:

$$\frac{\partial \pi^{H}_{s}}{\partial p_{mh}}(1 - \lambda_{ms}) = -\lambda_{ms}\left(\frac{\pi^{H}_{s} - \pi^{H}_{s\setminus m}}{\pi^{M}_{m} - \pi^{M}_{m\setminus s}}\right)\frac{\partial \pi^{M}_{m}}{\partial p_{mh}} \quad \forall m \in \mathcal{M}, h \in \mathcal{H} \tag{3.8}$$

which we generalize to accommodate different combinations of vertical structures between the two negotiating firms. In particular, let $1_{v \in \mathcal{V}}$ indicate whether an integrated holding owns firm v. Then, based on equation (3.7), the negotiated prices between insurer $m(a)$ and

18. The usual incentive to foreclose rivals from accessing upstream services arises when negotiated prices tend to infinity (Hart et al., 1990).

hospital $h(b)$ are:

$$\frac{\partial \pi^H_{s(b)}}{\partial p_{m(a)h(b)}} + 1_{b\in\mathcal{V}}\frac{\partial \pi^M_{m(b)}}{\partial p_{m(a)h(b)}} = -\frac{\lambda_{m(a)h(b)}}{1-\lambda_{m(a)h(b)}}\left(\frac{\pi^H_{s(b)} - \pi^H_{s(b)\setminus m(a)} + 1_{b\in\mathcal{V}}(\pi^M_{m(b)} - \pi^M_{m(b)\setminus m(a)})}{\pi^M_{m(a)} - \pi^M_{m(a)\setminus s(b)} + 1_{a\in\mathcal{V}}(\pi^H_{s(a)} - \pi^H_{s(a)\setminus s(b)})}\right) \times\left(\frac{\partial \pi^M_{m(a)}}{\partial p_{m(a)h(b)}} + 1_{a\in\mathcal{V}}\frac{\partial \pi^H_{s(a)}}{\partial p_{m(a)h(b)}}\right) \quad \forall m\in\mathcal{M}, h\in\mathcal{H} \tag{3.9}$$

which is the standard condition in the bargaining literature, extended to consider the incentives related to vertical integration between insurers and hospitals. For integrated firms, a change in hospital prices affects both hospital and insurer profits.

Negotiated prices in equation (3.9) can be rewritten in matrix form. After rearranging and stacking equations, the vector of negotiated prices for hospital system $s(b)$ is given by:

$$\boldsymbol{p}_{s(b)} = \boldsymbol{c}^H_{s(b)} - (\Omega_{s(b)} + \Lambda_{s(b)})^{-1}(D^H_{s(b)} + \Gamma_{s(b)}) \tag{3.10}$$

where $\boldsymbol{p}_{s(b)}$ contains negotiated prices between each hospital $h \in \mathcal{H}_{s(b)}$ and each insurer. On the right-hand side, $\boldsymbol{c}^H_{s(b)}$ contains hospital marginal costs for each hospital and insurer. Thus, equilibrium mark-ups over marginal cost combine several elements that we analyze in detail.[19]

First, the matrix $\Omega_{s(b)}$ captures demand price sensitivity for hospital h from enrollees of insurer m. Each entry in this matrix is given by:

$$\Omega_{s(b)[h,m]} = \sum_{j\in\mathcal{J}_m}\frac{\partial D^H_{hj}(\phi, p)}{\partial p_{m(a)h(b)}}$$

19. Equation (3.10) nests other models as particular cases. First, in the absence of vertical integration ($\mathcal{V} = \emptyset$) and when hospitals have all the bargaining power ($\lambda_{ms} = 0$), we recover the usual Nash-Bertrand conditions for hospital pricing, $\boldsymbol{p}_{s(b)} = \boldsymbol{c}^H_{s(b)} - \Omega^{-1}_{s(b)}D^H_{s(b)}$. Second, if all bargaining power is granted to insurers ($\lambda_{ms} = 1$), then hospital prices are equal to hospital marginal costs, $\boldsymbol{p}_{s(b)} = \boldsymbol{c}^H_{s(b)}$. Finally, allowing for both players to hold some bargaining power ($\lambda_{ms} \in (0, 1)$) in absence of vertical integration ($\mathcal{V} = \emptyset$), equilibrium hospital prices are set at a mark-up over marginal costs, $\boldsymbol{p}_{s(b)} = \boldsymbol{c}^H_{s(b)} - (\Omega_{s(b)} + \Lambda_{s(b)})^{-1}D^H_{s(b)}$, such that mark-ups depend on price sensitivity augmented by bargaining, similar to that in Gowrisankaran et al. (2015). An identical conclusion can be drawn from equation (3.8): as λ_{ms} goes to zero, optimal prices solve $\frac{\partial \pi^H_s}{\partial p_{mh}} = 0$, whereas as $(1 - \lambda_{ms})$ goes to zero, optimal prices solve $\frac{\partial \pi^M_m}{\partial p_{mh}} = 0$.

which measures standard demand responses to hospital prices across plans of insurer m. The more price sensitive hospital demand is, the lower the equilibrium mark-up in hospital price $p_{m(a)h(b)}$.

Second, the matrix $\Lambda_{s(b)}$ captures additional considerations of price sensitivity of the insurer's holding. Each entry in this matrix is:

$$\Lambda_{s(b)[h,m]} = \underbrace{\frac{\lambda_{m(a)h(b)}}{1-\lambda_{m(a)h(b)}}}_{\substack{\text{Relative} \\ \text{bargaining skill}}} \underbrace{\sum_{j \in \mathcal{J}_m} [D^H_{jh}(\phi, p) - D^H_{jh\setminus m}(\phi, p)]}_{\substack{\text{Contribution of insurer } m \\ \text{to demand for hospital } h}} \underbrace{\left(\frac{\frac{\partial \pi^M_{m(a)}}{\partial p_{m(a)h(b)}} + 1_{a \in \mathcal{V}} \frac{\partial \pi^H_{s(a)}}{\partial p_{m(a)h(b)}}}{\pi^M_{m(a)} - \pi^M_{m(a)\setminus s(b)} + 1_{a \in \mathcal{V}} (\pi^H_{s(a)} - \pi^H_{s(a)\setminus s(b)})} \right)}_{\text{Sensitivity of holding } a \text{ profits to negotiated price}}$$

where the first term depends on the relative bargaining weights, and shows that insurers with higher bargaining skill obtain lower hospital mark-ups. The second term captures the marginal contribution of insurer m to demand for hospital h. The larger the contribution of insurer m, the lower the hospital mark-up. Finally, the third term measures the sensitivity of holding a's profits to the negotiated price and captures the patient-steering effect in integrated insurers, when $a \in \mathcal{V}$.[20]

Finally, $\Gamma_{s(b)}$ captures the enrollee-steering effect in integrated hospitals, when $b \in \mathcal{V}$. This term measures the ability of hospital systems to leverage their integrated insurers for higher prices:

20. The numerator is the marginal effect of an increase in hospital prices on holding a's profits. It includes not only the standard negative effect on insurer $m(a)$ but also the positive effect on the profits of affiliated hospitals $s(a)$. The more sensitive holding a's profits are to hospital prices, the lower the equilibrium mark-up charged by hospital $s(b)$ to insurer $m(a)$. The denominator is the marginal value of the relationship with system $s(b)$ to holding a's profits. As discussed above, disagreement payoffs not only include the standard negative effect of losing the patients from hospital system $s(b)$ but also the positive effect of such disagreement on the profits of its affiliated hospital system $s(a)$. The lower the marginal value of $s(b)$ to the profits of holding a, the lower the mark-ups charged by the hospital $s(b)$ to insurer $m(a)$.

$$\Gamma_{s(b)[m]} = 1_{b\in\mathcal{V}}\Bigg[\underbrace{\frac{\partial \pi^M_{m(b)}}{\partial p_{m(a)h(b)}}}_{\text{Effect on integrated insurer profits}} + \underbrace{\left(\pi^M_{m(b)\setminus m(a)} - \pi^M_{m(b)}\right)}_{\text{Integrated insurer disagreement profit}} \underbrace{\frac{\lambda_{m(a)h(b)}}{1-\lambda_{m(a)h(b)}}}_{\text{Relative bargaining skill}} \underbrace{\left(\frac{-\frac{\partial \pi^M_{m(a)}}{\partial p_{m(a)h(b)}} - 1_{a\in\mathcal{V}}\frac{\partial \pi^H_{s(a)}}{\partial p_{m(a)h(b)}}}{\pi^M_{m(a)} - \pi^M_{m(a)\setminus s(b)} + 1_{a\in\mathcal{V}}(\pi^H_{s(a)} - \pi^H_{s(a)\setminus s(b)})}\right)}_{\text{Sensitivity of holding } a \text{ to negotiated price}}\Bigg]$$

where the first term measures the effect of higher hospital prices for rival insurer $m(a)$ on the integrated insurer $m(b)$. Higher hospital prices to rival insurer $m(a)$ steer demand towards the integrated insurer $m(b)$, thus increasing holding b's profits. The stronger this effect, the higher the prices $p_{m(a)h(b)}$. The second term captures the benefits of insurer $m(b)$ from the disagreement between insurer $m(a)$ and hospital $s(b)$. The larger the benefits of $m(b)$ under the disagreement with $m(a)$, the higher the prices $p_{m(a)h(b)}$. This effect is augmented by the bargaining weight of insurer $m(a)$ and the marginal loss in profit of holding a from an increase in the negotiated price. This adjustment captures the incentives to foreclose $m(a)$, which is larger when facing a counterpart with high bargaining skill, or when hospital $s(b)$ is relevant for holding a's profits. In the limit case in which the insurer $m(a)$ has all the bargaining power, this last component implies perfect foreclosure by hospital system $s(b)$ through an infinite $p_{m(a)h(b)}$.

Integrated holdings set hospital prices for their insurer and hospitals to maximize joint profits in equation (3.5). Although not arising from a bargaining problem, the first order condition for optimality of that problem is identical to that of a bargaining problem in which the bargaining weight λ is set to zero. This implies that equation (3.10) nests the optimal hospital prices of the integrated firm, and therefore we can solve for all hospital prices from a single matrix equation.

Premium Setting by Non-Vertically Integrated Insurers

Insurers compete in premiums taking negotiated hospital prices as given. Optimal premiums for a non-integrated insurer m are those that maximize insurer profits in equation

(3.4). The first order condition is:

$$\phi_j^*(\boldsymbol{p}) = c_j^M - \underbrace{\frac{1}{\frac{\partial D_j^M(\boldsymbol{\phi},\boldsymbol{p})}{\partial \phi_j}}\left[D_j^M(\boldsymbol{\phi},\boldsymbol{p}) + \sum_{j'\neq j, j'\in\mathcal{J}_m} \frac{\partial D_{j'}^M(\boldsymbol{\phi},\boldsymbol{p})}{\partial \phi_j}(\phi_{j'}^* - c_{j'}^M)\right]}_{\text{Standard multiproduct insurer mark-up}} \quad \forall j \in \mathcal{J}_m \tag{3.11}$$

which is the standard Bertrand-Nash pricing for a multiproduct insurer that offers differentiated plans. Optimal premiums are set as a mark-up over marginal costs that depends on two terms: price sensitivity of demand for plan j to changes in its premium, and price sensitivity of demand for other plans $j' \in \mathcal{J}_m$ to changes in premium of plan j. Since plans are substitutes, the insurer internalizes substitution across its plans when optimally setting premiums.

Premium Setting by Vertically Integrated Insurers

Vertically integrated insurers face more complex incentives when setting premiums, as they internalize the steering effect that premiums have on their integrated hospitals. Optimal premiums in this case are given by:

$$\begin{aligned}\phi_j^*(\boldsymbol{p}) \;=\; c_j^M - \frac{1}{\frac{\partial D_j^M(\boldsymbol{\phi},\boldsymbol{p})}{\partial \phi_j}}\Bigg[& \underbrace{D_j^M(\boldsymbol{\phi},\boldsymbol{p}) + \sum_{j'\neq j, j'\in\mathcal{J}_{m(a)}} \frac{\partial D_{j'}^M(\boldsymbol{\phi},\boldsymbol{p})}{\partial \phi_j}(\phi_{j'}^* - c_{j'}^M)}_{\text{Standard multiproduct insurer mark-up}} \\ & + \underbrace{\sum_{h\in\mathcal{H}_s}\left(\sum_{j'\in\mathcal{J}_{m(a)}} \frac{\partial D_{hj'}^H(\boldsymbol{\phi},\boldsymbol{p})}{\partial \phi_j}(p_{m(a)h} - c_{m(a)h}^H)\right)}_{\text{Steering from integrated insurer}} \\ & + \underbrace{\sum_{h\in\mathcal{H}_s}\left(\sum_{m'\neq m(a), m'\in\mathcal{M}}\sum_{j'\in\mathcal{J}_{m'}} \frac{\partial D_{hj'}^H(\boldsymbol{\phi},\boldsymbol{p})}{\partial \phi_j}(p_{m'h} - c_{m'h}^H)\right)}_{\text{Steering from rival insurers}}\Bigg] \quad \forall j \in \mathcal{J}_{m(a)}\end{aligned} \tag{3.12}$$

Besides the standard effects shown in equation (3.11), the integrated insurer faces two additional effects associated to the profits of its integrated hospitals. The first term captures

the effect of premiums on hospital profits coming from the enrollees of integrated plans. The second term captures the effect of premiums on hospital profits coming from the enrollees of competing plans. Therefore, integrated insurers have stronger incentives for premium competition as they account for the potential benefits of steering demand towards their integrated hospitals from the integrated insurer, but also from rival insurers.

3.4 Econometric Model

Our goal is to estimate the model to study equilibria under counterfactual market structures and assess the welfare effects of banning vertical integration. The parameters of interest are the preferences over insurance plans and hospitals, hospital costs, and bargaining weights. Estimation proceeds in four stages. First, we estimate negotiated prices, resource intensity weights and consumer health risk. Second and third, we estimate discrete choice models of demand for hospitals and insurance plans. Fourth, we estimate hospital marginal costs and bargaining weights.

3.4.1 Negotiated Prices, Resource Intensity Weights and Health Risk

Our data collects the price for each admission, along with identifiers for hospital, insurer, diagnosis and consumer. However, our model focuses on a scalar negotiated price for each insurer and hospital. This simplifying feature is common in bargaining models (Horn and Wolinsky, 1988; Gowrisankaran et al., 2015; Ho and Lee, 2017), and suggests a decomposition of observed payments into a negotiation index over which bargaining takes place, and a resource intensity weight that scales the index to match payments. For a particular admission price p_{ihmdt}, this decomposition is:

$$p_{ihmdt} = p_{hmt}\omega_{ihmdt} \tag{3.13}$$

which is fully generic without further restrictions. However, it clearly precludes the identification of the components of interest. Therefore, we impose restrictions on weights ω. In particular, we assume that it is common across insurers, hospitals and years, but varies

with diagnosis and consumer attributes to capture heterogeneity in admission complexity. In a similar exercise, Gowrisankaran et al. (2015) used the resource intensity weights (DRG) calculated by the authorities (CMS) to reimburse hospitals in the U.S. as a proxy for ω, allowing it to vary only by diagnosis.[21] Since a standardized weight is unavailable in Chile, we estimate them from the data. By constructing detailed utilization metrics based on public system prices, we are able to separate the consumer-diagnosis component $\omega_{\kappa(i)d}$ from the negotiated price component p_{hmt}. Appendix C.1.2 describes this procedure and its advantages relative to estimating this equation using fixed effects.[22][23]

Table 3.3-B displays full admission prices in the raw data as a share of total payments, while Panel 3.3-C displays estimated hospital prices as a share of total payments. Controlling for demographics and diagnosis matters, but only marginally. Estimated prices fit average observed prices well for each combination of insurer-hospital-year, as shown in Figure C.3. Additionally, Table C.3 displays the estimated resource intensity weights by diagnosis and demographic group.

Finally, we also estimate consumer health risk, which is then used to compute the distribution of insurer costs, hospital revenues, and consumer expected utility. We use a frequency estimator of admission probabilities for the population of enrollees. We allow for time-invariant admission probabilities to vary across diagnoses and gender-age. Table C.4 shows the estimated probabilities.

21. Additionally, Ho and Lee (2017) construct negotiated price by imposing similar restrictions based on DRG weights.

22. Using fixed effects or imposing functional forms to separate the bargaining component from the observable price is common in structural bargaining models (Cooper et al., 2018; Crawford et al., 2018).

23. Limiting the variation of resource intensity weights has implications. First, by assuming constant weights across hospitals, they cannot differ in their relative charges across medical conditions, implying that targeted investments in treatment efficiency are only priced on average. Since the hospitals in our sample are large general hospitals, we consider this a mild assumption. Second, we assume time-invariant weights. Since we do not observe substantial technological changes in inpatient care during our period of study, we expect minor variation over time in weights. Finally, we also assume that these weights are common knowledge. This seems appropriate in a mature market like the one we study. Moreover, it allows us to label the weights as resource intensity weights, which map average hospital resource utilization for each condition and consumer type to service costs.

3.4.2 *Demand for Health Care*

The next step in our analysis is health care demand. We specify a hospital choice model conditional on diagnosis and insurance plan. A consumer who faces a medical condition chooses a hospital from the hospital network available in his current plan based on the out-of-pocket cost of the treatment and the distance to the hospital. Importantly, we allow for observable heterogeneity in preferences and control for unobserved preferences for hospitals.

The utility of consumer i enrolled in insurance plan j of choosing hospital h for diagnosis d at time t is given by:

$$u^H_{ijhdt} = \alpha^H_i c_{jh} p_{ijhdt} + \beta_v v_{ih} + \delta^H_{h\kappa(i)d} + \varepsilon_{ijhdt}$$

where $\kappa(i)$ is the consumer type and $p_{ijhdt} = \omega_{\kappa(i)d} p_{mht}$ is the weighted negotiated price described in the previous section; c_{jh} is the coinsurance rate obtained by the enrollees of plan j at hospital h, and v_{ih} is the distance from the consumer residence to hospital h. Additionally, $\delta^H_{h\kappa(i)d}$ is the hospital h fixed effect for consumers of type $\kappa(i)$ under diagnosis d, which captures time-invariant unobserved hospital attributes for that specific group of patients. Finally, ε_{ijhdt} is an idiosyncratic preference shock assumed to follow an i.i.d. T1EV distribution. As described in Section 3.2, consumers choose among hospitals, including those of the public system. The outside option, defined as the nearest public hospital, delivers a utility given by:

$$u^H_{ij0dt} = \alpha^H_i c_{j0} p_{j0dt} + \vartheta_{l(i)} + \varepsilon^H_{ij0dt}$$

where p_{j0dt} is the price of the public option for diagnosis d at time t, and $\vartheta_{l(i)}$ is a county-fixed effect that accounts for the heterogeneity in the outside option across consumer locations.

Since we assume preference shocks are i.i.d. T1EV, the probability that consumer i

enrolled in plan j chooses hospital h given diagnosis d is:

$$\sigma^H_{ijht|d} = \frac{\exp(\alpha^H_i(c_{jh}p_{ijhdt} - c_{j0}p_{j0dt}) + \beta_v v_{ij} + \delta^H_{h\kappa(i)d} - \vartheta_{l(i)})}{1 + \sum_{r\in\mathcal{H}} \exp(\alpha^H_i(c_{jr}p_{ijrdt} - c_{j0}p_{j0dt}) + \beta_v v_{ir} + \delta^H_{r\kappa(i)d} - \vartheta_{l(i)})}$$

We model observed heterogeneity in the price coefficient as:

$$\alpha^H_i = D'_{age,i}\alpha^H_{age} + D'_{HH,i}\alpha^H_{HH} + \alpha^H_{income} income_i \tag{3.14}$$

where $D_{age,i}$ and $D_{HH,i}$ are dummies for whether consumer i belongs to specific bins of age and household characteristics, respectively. The age bins match the definitions of $\kappa(i)$ while the household characteristics include gender, marital status, and whether she has dependents.[24]

We estimate the demand model via maximum likelihood using the detailed data on admissions described in Section 3.2.2, which includes admission medical and financial attributes, along with patient attributes. We measure the distance between households and hospitals as the linear distance between the centroid of the household's county of residence and the hospital location.

Identification. Hospital demand is identified off variation in prices and distances across consumers within and between hospitals. We face three threats to identification. First, the correlation between prices and unobservable consumer preferences for hospitals. Given that estimation requires prices for all hospitals in the choice set, we use predicted hospital prices as estimated in the previous section. By construction, these prices are free of much of the individual-specific heterogeneity that might be correlated with unobserved preferences. By adding fixed effects for hospital-diagnosis, we control for systematic tastes for particular hospitals for certain conditions. By further estimating the model by age groups, we account for the possibility that these tastes vary across consumer age groups. The remaining potentially endogenous taste variation is within hospital-diagnosis and

24. We consider single males as the baseline group. Consistent with that specification, the coefficients on the age-group dummies capture price sensitivity for single men, while the coefficients for other groups (e.g., single women or consumers with dependents) should be added to the correspondent age-group estimate.

across gender. Given our restrictions on the resource intensity weights, price variation for a given diagnosis across age and gender is independent of the hospital and the consumer insurer, which makes this form of endogeneity unlikely.[25]

The second issue is that consumer location may be based on hospital unobservables that affect consumer choice. We assume that consumer location is exogenous based on the facts that inpatient events are relatively rare, and that there is a broad set of options for outpatient care in the market. The third issue is that unobserved preferences could drive selection into plans. In our setting, all plans offer complete networks and differ only in the coinsurance rates offered across hospitals. To our knowledge, there are no other systematic differences among plans in terms of hospital access. Given this variation is observable and priced, it should not threaten our strategy.[26]

Results. Table 3.5-A shows the estimates of the hospital demand model, where each column includes an increasingly richer set of fixed effects $\delta^H_{h\kappa(i)d}$. For the rest of the paper, we use the results in columns (4) and (5), which include estimates the model separately for young and old consumers and includes hospital-diagnosis fixed effects.

Overall, we find that all consumers are price sensitive, with young, single males, and lower-income consumers being more price sensitive. Additionally, consumers value hospital location and prefer hospitals close to their residences. Note that adding richer sets of fixed effects delivers larger estimates of price sensitivity, which suggests that those fixed effects indeed capture unobserved drivers of hospital choice that are correlated with prices, such as hospital quality.[27] Table 3.5-B summarizes price elasticities, while Figure 3.2-a shows a histogram of them. The average and median price elasticities are -2.40 and -1.88,

25. Price endogeneity at this level would require variation in preferences for a hospital-diagnosis across young females and males. Moreover, this variation should be considered by insurers and hospitals when negotiating a price that applies across diagnoses and consumers, conditional on the average young consumer preference for the hospital.

26. The same assumption has been used extensively in the literature (e.g. Ho 2006; Ho and Lee 2017).

27. To evaluate whether hospital fixed effects in hospital demand capture meaningful differences across hospitals, we study their correlation with observable hospital attributes. Figure C.2 shows the relationship between estimated hospital fixed effects in Column (2) of Table 3.5 and an objective measure of quality, which comes from the position of the hospital in the Webometrics Ranking of World Hospitals (Cybermetrics Lab, 2016). Both variables display a positive correlation, which suggests that our strategy indeed captures relevant hospital attributes that might drive choices.

which are larger than those in the literature on the intensive margin for inpatient care in the U.S.[28]

3.4.3 *Demand for Insurance Plans*

We develop a model of insurance plan choice. Households take into account the expected utility from the plan for all household members. Therefore, households choose among available plans based on premiums, hospital networks, and the expected quality and price of health care.[29]

Using our estimates of hospital demand, we compute the expected utility from the hospital network offered by each plan, in line with Capps et al. (2003). Given the hospital demand model in Section 3.4.2, the expected utility of consumer i from the hospital network of plan j at time t is:

$$EU^H_{ijt} = \sum_{d \in \mathcal{D}} \gamma_{d\kappa(i)} \log \sum_{h \in \mathcal{H}} \exp(\alpha^H_i (c_{hj} p_{ijhdt} - c_{j0} p_{j0dt}) + \beta_v v_{ih} + \delta^H_{h\kappa(i)d} - \vartheta_i) \tag{3.15}$$

where $\gamma_{d\kappa(i)}$ is the probability of consumer type $\kappa(i)$ of being diagnosed with condition d.

Let the utility of household-type f from choosing insurance plan j at time t be:

$$u^M_{fjt} = \alpha^M_f \phi_{fjt} + \beta_f \sum_{i \in f} EU^H_{ijt} + \delta^M_{m(j)\kappa(f)} + \varepsilon^M_{fjt} \tag{3.16}$$

where ϕ_{fjt} is the premium charged to household-type f if choosing plan j at time t; $\delta^M_{m(j)\kappa(f)}$ is the mean utility that household-type $\kappa(f)$ obtains from plans offered by insurer $m(j)$ other than premiums and expected health care services (e.g., insurer customer service); and ε^M_{fjt} is an idiosyncratic preference shock i.i.d. T1EV.[30] Under these assumptions, the

28. The utilization elasticity in the RAND experiment is -0.2 (Aron-Dine et al., 2013), whereas Prager (2018) estimates price elasticities across hospitals of between -0.03 and -0.12. This pattern may be driven by two differences that make prices more salient in Chile than in the U.S.: (i) consumers in the former share the cost of treatment at the margin, given there are no caps on out-of-pocket expenditure, and (ii) hospital prices are more transparent in Chile, because prices and insurance coverage are mostly available to consumers when choosing hospitals.

29. We assume that household decisions equally weight the welfare of all household members.

30. Recall that consumers must spend at least 7% of their taxable income (up to a cap) on insurance, as

choice probability of plan j by household-type f at time t is given by:

$$\sigma^M_{fjt} = \frac{\exp(\alpha^M_f \phi_{fjt} + \beta_f \sum_{i \in f} EU^H_{ijt} + \delta^M_{m(j)\kappa(f)})}{\sum_{k \in \mathcal{J}} \exp(\alpha^M_f \phi_{fkt} + \beta_f \sum_{i \in f} EU^H_{ikt} + \delta^M_{m(k)\kappa(f)})}$$

We estimate the model by maximum likelihood. We allow for observable heterogeneity on preferences over premiums and the expected utility of hospital networks across age, household composition and income, with a structure similar to that in our hospital demand model. As discussed in Section 3.2.2, there is a large number of plans in the market with negligible market share. To reduce the size of the choice set, we let each consumer choose among the 30 most popular plans offered in their market segment each year.[31] In total, each insurer offers up to 40 plans over all markets, each year. Appendix C.1.3 describes the construction of plan choice sets.

Identification. Plan demand is identified by variation in premiums and offered hospital networks across plans within an insurer and household type. However, endogeneity of premiums and networks might threaten identification. In particular, insurers could provide additional services to their clients that are not captured by plan attributes and are unobserved to us. If these services affect premium setting or the contracting phase with hospitals, it would cause an endogeneity concern. We address this by assuming that these unobserved attributes are constant over time, and add plan-level fixed effects to capture these unobservables.

Results. Table 3.6-A shows plan demand estimates. Columns (1)-(5) report results for premium sensitivity, whereas columns (6)-(10) report results for willingness to pay for hospital networks covered by a plan. For the rest of the paper, we use the results in columns (4), (5), (9) and (10), which estimate the model separately for young and old consumers and include plan fixed effects.

discussed in Section 3.2.1. This mandatory payment is not choice-specific and is therefore omitted from the equation above.

31. We allow consumers to re-enroll in their plan even if it was no longer available, consistent with the guaranteed renewability regulation.

Our estimates suggest that all consumers are premium sensitive, but young single, males, with no dependents, and lower-income consumers are more price sensitive. This heterogeneity is consistent with patterns of premium sensitivity previously found in literature (e.g. Ho and Lee 2017; Tebaldi 2017). As expected, households have a preference for plans offering a higher expected utility from health care services. Table 3.6-B summarizes premium elasticities, and Figure 3.2-b shows a histogram of them. The average and median premium elasticities are -1.32 and -1.01, which are within the range of estimates in the literature.[32]

3.4.4 Marginal Costs and Bargaining Weights

We turn to the estimation of supply-side parameters, namely hospital marginal costs and bargaining weights. The estimation procedure builds on optimality conditions for insurer premiums and negotiated hospital prices in Section 3.3.2, to propose a GMM estimator of the form:

$$\min_{\boldsymbol{\lambda},\boldsymbol{\theta},\boldsymbol{c},\boldsymbol{\phi}} \quad g(\boldsymbol{\lambda},\boldsymbol{\theta},\boldsymbol{c},\boldsymbol{\phi})' W^{-1} g(\boldsymbol{\lambda},\boldsymbol{\theta},\boldsymbol{c},\boldsymbol{\phi}) \tag{3.17}$$

$$\text{s.t} \quad \boldsymbol{c} = C(\boldsymbol{\phi},\boldsymbol{\lambda},\boldsymbol{\theta}) \tag{3.18}$$

$$\boldsymbol{\phi} = \boldsymbol{\phi}^*(\boldsymbol{c},\boldsymbol{\theta}) \tag{3.19}$$

where $\boldsymbol{c}$ and $\boldsymbol{\phi}$ are auxiliary variables that capture the equilibrium constraints (3.18) and (3.19), $g(\cdot)$ is a vector of moment conditions, and W is a weighting matrix. Ignoring the auxiliary variables, the problem is optimized over bargaining weights $\lambda_{ms} \in (0,1)$ and VI weights $\theta_m \geq 0$.

The first constraint (3.18) is that hospital costs, conditional on premiums and weights, must satisfy the first order condition of the Nash-in-Nash bargaining problem in equation (3.10). This optimality condition allows us to build moments related to marginal costs that

32. For instance, Abaluck and Gruber (2011) estimate mean elasticities around -1 for Medicare Part D; Curto et al. (2015) estimate mean elasticities around -1.1 for Medicare Advantage; Ho and Lee (2017) estimates mean elasticities between -2.95 and -1.23 for enrollees of CalPERS; and Tebaldi (2017) estimates mean elasticities between -1.2 and -0.8 for subsidized buyers of Silver plans in the California ACA exchange. Atal (2015) estimates a lower mean elasticity of -0.2 in the Chilean market.

discipline the estimator based on how $\boldsymbol{\lambda}$ and $\boldsymbol{\theta}$ determine costs. The second constraint (3.19) is that premiums, in equilibrium and under all pair-wise disagreements between hospitals and insurers, must satisfy the first order condition of insurer premium setting in equations (3.11) and (3.12). This condition allows us to evaluate the disagreement profit of hospitals and insurers, and to build moment conditions based on equilibrium premiums.[33]

The first set of moment conditions matches the equilibrium and observed premiums. We impose this moment condition separately for each insurer-year, such that:

$$g^{\phi}_{mt}(\boldsymbol{\phi}) = \frac{1}{|\mathcal{J}_m|} \sum_{j \in \mathcal{J}_m} (\tilde{\phi}_{jt} - \phi_{jt}) \quad \forall m \in \mathcal{M}, t \in \mathcal{T}$$

where $\tilde{\phi}_{jt}$ are observed premiums, ϕ_{jt} are equilibrium premiums, and $\mathcal{T}$ is the set of years in our data. Note that in the absence of vertical integration, these conditions do not inform the estimates of marginal costs.[34]

The second set of moment conditions builds on firms' financial records. We construct moment conditions that match equilibrium mark-ups to observed profit to revenue ratios, displayed in Table C.5. Denoting the observed ratios $\tilde{\mu}_{lt}$, we define these conditions as:

$$\begin{aligned}
g^{\mu}_{m}(\boldsymbol{c}, \boldsymbol{\phi}) &= \frac{1}{|\mathcal{T}|} \sum_{t \in \mathcal{T}} \left(\tilde{\mu}_{mt} - \frac{\sum_{j \in \mathcal{J}_m} D^{M}_{jt}(\boldsymbol{\phi}_t, \boldsymbol{p}_t)(\phi_{jt} - c^{M}_{jt})}{\sum_{j \in \mathcal{J}_m} D^{M}_{jt}(\boldsymbol{\phi}_t, \boldsymbol{p}_t)\phi_{jt}} \right) \quad \forall m \in \mathcal{M} \\
g^{\mu}_{h}(\boldsymbol{c}, \boldsymbol{\phi}) &= \frac{1}{|\mathcal{T}|} \sum_{t \in \mathcal{T}} \left(\tilde{\mu}_{ht} - \frac{\sum_{m \in \mathcal{M}} \sum_{j \in \mathcal{J}_m} D^{H}_{hjt}(\boldsymbol{\phi}_t, \boldsymbol{p}_t)(p_{mht} - c^{H}_{mht})}{\sum_{m \in \mathcal{M}} \sum_{j \in \mathcal{J}_m} D^{H}_{hjt}(\boldsymbol{\phi}_t, \boldsymbol{p}_t)p_{mht}} \right) \quad \forall h \in \mathcal{H}
\end{aligned}$$

Finally, the third set of moments exploits orthogonality conditions. Consider a set of instruments $\boldsymbol{Z}$ that is independent of within hospital-year variation in marginal cost and predicts negotiated prices. In particular, $\boldsymbol{Z}$ includes four metrics of willingness to pay for hospitals by enrollees of each insurer: (i) willingness to pay for each hospital, (ii) for

33. The nested fixed point for premiums problem distinguishes our estimator from that in recent work (Ho and Lee, 2017; Gowrisankaran et al., 2015). This provides us with additional identifying moments at the expense of substantial computational cost.

34. These conditions inform the estimator because of the pass-through of marginal costs to premiums, the strategic interaction of insurers, and how premiums affect the profits of integrated firms.

each hospital system, (iii) for all rival hospitals, and (iv) for all rival systems. Each metric computes willingness to pay relative to the actual network offered by each plan, as in Capps et al. (2003), and then averages by insurer. This creates four instruments that vary across hospitals, insurers and years, similar to those used in previous work (e.g., Gowrisankaran et al. 2015, Ho and Lee 2017). However, we exploit prices in the public system instead of negotiated prices to compute these metrics. This departure from the traditional BLP-style instruments (Berry et al., 1995) weakens the assumption needed to claim exogeneity, as it removes any dependence with the bargaining process between hospitals and insurers, but keeps enough hospital and consumer heterogeneity as to predict prices.[35]

To construct this final set of moments, we decompose the marginal cost of hospital h as:

$$c_{hmt} = \bar{c}_h + \bar{c}_t + \eta_{hmt}$$

where $\bar{c}_h$ and $\bar{c}_t$ capture cost differences across hospitals and years.[36] The remaining variation η_{hmt} is assumed to be independent of instruments Z_{hmt}. We construct moment conditions as:

$$g_k^Z(\boldsymbol{c}) = \frac{1}{|\mathcal{H}| \times |\mathcal{M}| \times |\mathcal{T}|} \sum_{h \in \mathcal{H}} \sum_{m \in \mathcal{M}} \sum_{t \in \mathcal{T}} Z_{k,hmt} \eta_{hmt}$$

where k indexes the instruments, which include the willingness to pay metrics and hospital and year dummies, so as to match the decomposition of marginal cost. Our assumption relies on the connection between the public and private systems: public system prices correlate with hospitals' costs, but are exogenous to the contracting process between private hospitals and insurers.

We optimize the GMM objective by replacing the equilibrium constraints within the moment conditions.[37] To reduce the dimensionality of the problem and to guide our

35. Willingness to pay instruments are associated to BLP instruments, as it is considered an attribute of plans.

36. We decompose marginal costs this way to reduce computational burden. Decomposing cost as $c_{hmt} = \bar{c}_{ht} + \eta_{hmt}$ instead does not affect the results to a relevant extent, but increases the time to convergence substantially.

37. Using a constrained, derivative-free numerical optimizer, we account for the support of $(\boldsymbol{\lambda}, \boldsymbol{\theta})$. We use the COBYLA algorithm (Powell, 2007). Computing numerical gradients for this problem is prohibitively costly.

counterfactuals, we decompose bargaining weights into a convex combination of insurer and hospital components. In particular, if insurer a is negotiating with hospital b, the bargaining weight takes the form:

$$\lambda_{ab} = \alpha^{\lambda}(a,b)\lambda_a + (1 - \alpha^{\lambda}(a,b))\lambda_b \tag{3.20}$$

with the weight $\alpha^{\lambda}(a,b)$ being an additional parameter to be estimated, but restricted to be the same for each type of negotiation. In particular, there are four types of negotiations: non-integrated hospitals bargaining with non-integrated insurers, integrated hospitals with integrated rival insurers, integrated hospitals with non-integrated insurers, and integrated insurers with non-integrated hospitals.[38] This specification has two main advantages. First, it guides how bargaining weights adjust under counterfactual integration scenarios. Second, it reduces the number of bargaining weights to estimate from $|\mathcal{S}| \times |\mathcal{M}| - |\mathcal{V}|$ to $|\mathcal{S}| + |\mathcal{M}| + 4$, which in our case reduces the number of bargaining parameters from 38 to 17. Further details regarding the implementation and estimation of the bargaining and VI weights are provided in Appendix C.3.1.

Identification. We start by focusing on the identification of $\boldsymbol{\lambda}$ given $\boldsymbol{\theta}$. First, note that the unobserved hospital marginal cost $\boldsymbol{c}$ uniquely determines $\boldsymbol{\lambda}$ using the first order condition of the Nash bargaining problem in equation (3.10). Second, hospital financial moments directly determine hospital average marginal cost, therefore $\boldsymbol{c}$ is known up to variation within hospital-year. This implies we can decompose hospital marginal costs into their average and unknown variation:

$$\boldsymbol{c} = \bar{\boldsymbol{c}} + \boldsymbol{\eta}$$

38. As the previous literature has emphasized, accurate estimation of bargaining weights often requires additional structure. For example, cross-sectional studies such as Gowrisankaran et al. (2015) and Ho and Lee (2017) assume that bargaining weights are insurer-specific. This increases the sample size over which the same weights are valid to all negotiations in which an insurer participates in the sample year. In our estimation, we let bargaining weights to be fixed for each pair of insurer and hospital system and follow the structure of equation (3.20) and leverage the panel structure of our data to increase the sample size in terms of number of negotiations.

and we can therefore rewrite equation (3.10) as:

$$\boldsymbol{p} - \bar{\boldsymbol{c}} = F(\boldsymbol{p}, \boldsymbol{c}, \boldsymbol{\eta})$$

where the right hand side mark-up is a known non-linear function of prices $\boldsymbol{p}$ and within hospital-year variation in costs $\boldsymbol{\eta}$. This leads us to a non-linear instrumental variable problem (Hansen and Singleton, 1982), in which we seek to identify the residual $\boldsymbol{\eta}$, to which the regressor $\boldsymbol{p}$ is endogenous due to the bargaining process. Thus, our willingness to pay instruments using public system prices are valid, as they are correlated with negotiated prices $\boldsymbol{p}$ but uncorrelated with $\boldsymbol{\eta}$.[39] Therefore, the orthogonality condition moments identify the remaining cost variation, which in turn identifies $\boldsymbol{\lambda}$.

Finally, the VI weights $\boldsymbol{\theta}$ are identified by the premium and insurer financial moments. Given hospitals marginal costs and equilibrium premiums, the Nash-Bertrand first order condition for the premium of an integrated plan is linear in θ. Thus, we jointly solve for the VI weights and cost vector $\boldsymbol{c}$. The premium matching condition implements this constraint by requiring $\boldsymbol{\theta}$ to match predicted and observed premiums. Moreover, insurer financial moments further discipline this link by providing a different level of aggregation over which premiums must match the data. The additional information contained in these moments helps to identify bargaining and VI weights.[40]

Results. Table 3.7 summarizes our estimates for bargaining weights and marginal costs.[41] Table 3.7-A shows that, on average, negotiations weigh insurer and hospitals gains from trade equally. However, these weights vary depending on the negotiation: integrated

39. Our instrument predicts hospital costs. It varies across hospitals and insurers due to compositional changes in demand caused by the joint distribution of preferences and risk. Public system prices are good predictors of hospital costs, because they capture common cost shocks across the public and private systems (e.g., physician labor costs).

40. As in the related literature, we cannot rule out the possibility of multiple solutions to the marginal cost equilibrium constraint (3.18), as $F(\cdot)$ may not be injective in $\boldsymbol{\eta}$. We explore a variety of starting values in the optimization procedure and obtain similar estimates upon convergence. Our results seem robust to the multiplicity concern, and our numerical analysis suggests that equation (3.18) is a contraction, at least locally to the only solution we find.

41. Table C.6 displays results for the first stage of negotiated prices on willingness-to-pay instruments, which show that our instruments are strong predictors of negotiated prices, with an F-test of 437.

hospitals and insurers have lower bargaining weights than non-integrated ones. One way to interpret this dispersion is by recognizing that Nash-in-Nash bargaining might overstate the ability of hospital systems to leverage their set of hospitals when negotiating. For example, if hospital systems cannot credibly threaten to remove all hospitals from the insurer's network upon disagreement, the bargaining objective will overstate the hospital system's ability to command higher prices. As the integrated firms in our setting also own horizontally integrated hospital systems, a reduction in hospital bargaining weights might be associated with this group to adjust for their increased leverage. This argument is consistent with the finding that the bargaining weight of integrated hospitals when negotiating with separately integrated insurers is less skewed, as both negotiating firms incorporate the bargaining surplus of their hospital systems. Regardless of the source of this heterogeneity, as bargaining weights are not strategic components and have no clear non-cooperative counterpart, they do not affect the interpretation of results from our counterfactuals.

Table 3.7-B displays estimates of hospital marginal costs, negotiated prices, and hospital mark-ups. Our estimates indicate that hospitals average mark-up is around 38%, which is mostly driven by our financial moment conditions. Interestingly, integrated hospitals charge a mark-up to their integrated insurer. Note that this does not imply a failure of vertical integration to eliminate double marginalization. Recall that this vertical market differs from those of retail goods, where consumers pay only to downstream firms. In this case, consumers pay in both upstream and downstream markets and are elastic to both hospital prices and premiums. This particularity implies that it is optimal for integrated firms to balance charges to consumers on both ends. The incentive to remove double marginalization, in this case, is reflected in the lower mark-up hospitals charge to their integrated consumers relative to those enrolled in plans of rival insurers.[42]

These results also show that integrated hospitals have lower marginal costs than their rival hospitals, but face a slightly higher cost of serving their integrated insurer. The first finding is partly driven by two high-quality and high-price hospitals among non-integrated

42. The reduction is also driven by incentives to steer patients to the integrated hospital, discussed in Section 3.4.5.

hospitals.The second result relates to the motivation for vertical integration, which we do not model. In particular, our work is silent on the effects of vertical integration on firms' organization. Therefore, we are unable to determine whether integrated firms provide better care to patients at a higher cost, or if insurers decide to integrate precisely with hospitals with higher costs. In both cases, vertical integration might lead to changes in costs which we do not capture. To assess the robustness of our welfare calculations, our counterfactuals explore the role of cost efficiencies .[43]

Finally, the estimated VI weights θ are 0.474 and 0.206 for the two integrated firms, m_3 and m_1. These estimates imply that both firms value hospital profits more than insurer profits. This finding is consistent with a heavier regulatory burden for insurers relative to hospitals in our setting;[44] or with commitments between the insurer and the hospital at the time of the merger.

3.4.5 Patient and Enrollee Steering Effects

Our model illustrates how vertical integration affects incentives in the market through patient-steering effects and enrollee-steering effects. In this section, we quantify such incentives.

From equation (3.7), the bargaining surplus of an integrated insurer m with a rival hospital system $s(b)$ is $(\pi^A_m - \pi^{NA}_{m\setminus s(b)} + \pi^A_{s(m)} - \pi^{NA}_{s(m)\setminus s(b)})$, which combines the surplus it obtains from both the insurance and health care markets. The bargaining surplus of the integrated hospital system from the negotiation $(\pi^A_{s(m)} - \pi^{NA}_{s(m)\setminus s(b)})$ is non-positive, as under the event of foreclosure the integrated system would benefit from the steering of patients to its hospitals. In the extreme, if this incentive to steer dominates the gains of the insurer

43. Notice that we do not identify fixed and sunk costs in the hospital industry or the insurance sector. Therefore, we cannot make statements regarding the overall profitability of the health sector. Also, fixed and sunk costs are irrelevant for the bargaining stage of the game since they are not contingent on reaching an agreement with a particular firm.

44. As an example of the asymmetric regulation between insurers and hospitals, our rich data comes from the regulator of insurers, whereas very little data exists on the private hospitals we study. Moreover, the regulator publishes quarterly reports on insurer profits, which generate adverse reactions from consumers and calls for stronger regulations on health-insurance providers. See La Tercera (2017) for an example discussing high profits in the industry in 2016—in which profits increased by 40.6% relative to 2015—, with several quotes from senators manifesting their concern.

m from bargaining with $s(b)$, then no agreement should take place between the two players. Therefore, we can measure the patient-steering effect as:

$$\frac{\pi^A_{s(m)} - \pi^{NA}_{s(m)\backslash s(b)}}{\pi^A_m - \pi^{NA}_{m\backslash s(b)}}$$

which is the fraction of the integrated insurer bargaining surplus from the insurance market that is lost due to the vertical incentive to steer patients away from a rival hospital.[45] We measure this incentive for the two integrated insurers in our setting as its average across non-integrated rival hospitals. We find that this fraction is -22.19% for m_1 and -15.85% for m_3.

Similarly, from equation (3.7), the bargaining surplus of an integrated hospital system s with a rival insurer $m(b)$ is $(\pi^A_s - \pi^{NA}_{s\backslash m(b)} + \pi^A_{m(s)} - \pi^{NA}_{m(s)\backslash m(b)})$. In this case, the bargaining surplus from the insurance market $(\pi^A_{m(s)} - \pi^{NA}_{m(s)\backslash m(b)})$ is non-positive, as the integrated insurer would benefit from its hospital foreclosing rivals and leading patients to switch away from $m(b)$. Thus, the enrollee-steering effect is given by:

$$\frac{\pi^A_{m(s)} - \pi^{NA}_{m(s)\backslash m(b)}}{\pi^A_s - \pi^{NA}_{s\backslash m(b)}}$$

which is the fraction of the integrated hospital bargaining surplus lost due to the incentive to steer enrollees away from a rival insurer. We measure this incentive for the two integrated insurers in our setting as its average across non-integrated rival hospitals. We find these fractions to be -29.84% for hospitals integrated with m_1 and -1.67% for hospitals integrated with m_3.

There is substantial heterogeneity in the patient- and enrollee-steering effects, driven by the horizontal differentiation among firms. The differentiation is starker in the enrollee-steering effect as we estimate unobserved consumer preference for m_1 to be substantially

45. This ratio relates to vertical gross upward pricing pressure indices (vGUPPIs) used in antitrust analysis (Moresi and Salop, 2013). vGUPPIs measure the potential harm of vertical integration captured by unilateral pricing incentives, which compare the value of sales diverted to the downstream merging partner to the revenue on volume lost by the upstream merging partner.

larger than that of m_3. Hence, while m_1 can count on steering patients away from rivals to work in their favor, m_3 cannot do so to the same extent. Overall, the fact that incentives associated with vertical linkages are quantitatively relevant suggests that the distortions related to vertical integration might be substantial and motivates our welfare analysis of banning vertical integration in the next section.[46]

3.5 Equilibrium Effects of Vertical Integration

We use our estimated model to solve for equilibrium in a scenario in which integrated insurers and hospitals are broken up. Banning vertical integration induces adjustments in plan premiums and hospital negotiated prices. Consumers react to these price changes in both markets by adjusting their hospital and insurance demand. We compute hospital profits, insurer profits, and consumer surplus in both scenarios to measure the welfare effects of vertical integration in this setting.

3.5.1 Simulation Details

The simulation consists of solving equation (3.10) for negotiated prices conditional on estimated hospital costs. The procedure is as follows: for each guess of hospital prices, we find the equilibrium premiums and demands, and evaluate equation (3.10) until reaching a fixed point. The root of this non-linear problem yields the new equilibrium negotiated hospital prices, insurance premiums, and demands. For our main results, we hold hospital marginal costs fixed at their estimated levels. We then explore the role of potential cost efficiencies in Section 3.5.3.

The specification of bargaining weights as in equation (3.20) offers the advantage of determining the bargaining weights for integrated firms in our counterfactual scenarios. In particular, we use the mixing coefficient α^{λ} of non-integrated hospitals and insurers to compute counterfactual bargaining weights for the simulated scenarios. Hence, the ban on

46. Importantly, the fact that these effects are larger than -100% supports the absence of foreclosure in this market. For example, if an integrated hospital system benefits more from a disagreement of its integrated insurer than the profit generated by the insurer from the agreement in question, than disagreement should happen and foreclosure take place.

vertical integration alters both the gains from trade and the heterogeneity in bargaining parameters.

3.5.2 Main Results

Banning vertical integration would have sizable effects in the hospital market, as shown by Table 3.8-A. Formerly integrated hospitals decrease negotiated prices across the board: the average price to non-integrated insurers falls by 19.84%, and that to their previously affiliated insurer by 2.44%. These results are consistent with the enrollee-steering effect: integrated hospitals had an incentive to increase prices to rival insurers to steer demand to their integrated insurer, which is not the case when vertical integration is banned. As a result, the market share of hospitals integrated at baseline coming from non-integrated insurers increases by 16.68%, whereas their market share coming from integrated insurers at baseline decreases by 18.53%.

Moreover, hospital profits decrease for both integrated and non-integrated hospitals. Non-integrated hospitals now bargain in a less distorted market with cheaper competing hospitals. As a consequence, non-integrated hospitals decrease their prices to integrated insurers by 0.88%, which is consistent with the patient-steering effect: integrated insurers had the incentive to increase prices to rival hospitals, as their integrated hospitals would recapture part of that hospital demand.

The insurance market is also affected by banning vertical integration, as shown in Table 3.8-B. After banning vertical integration, integrated insurers increase their premiums by 4.72% on average, while non-integrated insurers decrease theirs by 0.32%. Driven by these changes in premiums, consumers substitute towards insurers that were not integrated at baseline. Lower negotiated prices imply lower payments to hospitals increasing insurer profits.

Consumers on average benefit from banning vertical integration, as shown by Table 3.8-C.[47] Average consumer surplus increases by $55 per year. However, there is substantial heterogeneity, partly driven by premium sensitivity. Less premium sensitive consumers

47. We measure consumer surplus from the insurance market, given the utility from plans captures benefits from both markets by including the expected utility of hospital networks as an attribute. In particular, we

gain the most: elderly females and males increase their consumer surplus by $200 and $132 per year. On the other hand, young females and males are sufficiently price-sensitive as to lose from banning vertical integration due to increases in premiums, although their losses are small. On average, consumers are willing to pay 4% higher premiums to ban vertical integration.

3.5.3 Welfare Effects and Cost Efficiencies

Advocates of vertical integration often argue they induce cost efficiencies. To explore this margin, we evaluate how the welfare effects of vertical integration vary under a range of cost efficiencies, which are lost under our counterfactual ban. In practice, we consider cost efficiencies in a range of -10% to 30% relative to our estimated hospital costs, which only apply to admissions within integrated hospitals and insurers.[48] Figure 3.3-a shows results for this analysis.

Banning vertical integration increases overall welfare by $146 million per year, which accrue increases of $90.1 million in consumer surplus and $100.7 million in insurer profits, and a decrease of $44.8 million in hospital profits. Consumer surplus increases, driven by a reduction in negotiated prices between formerly integrated hospitals and non-integrated insurers, as reflected in the changes in hospital market shares shown in Table 3.8. Both integrated and non-integrated insurers are better off without integration. On the one hand, integrated insurers are better off because they set baseline premiums below the individually profit-maximizing levels to attract enrollees and steer them to their integrated hospitals. On the other hand, non-integrated insurers face lower hospital prices from formerly integrated

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compute expected consumer surplus following Small and Rosen (1981), as:

$$CS_f = \frac{1}{\alpha_f^M} \log \sum_{k \in \mathcal{J}} \exp\left(-\alpha_f^M \phi_{fk} + \beta_f \sum_{i \in f} EU_{ik}^H + \delta_{m(k)\kappa(f)}^M \right) + \iota$$

where ι is the Euler-Mascheroni constant.

48. To put this range in context, we use the case of childbirth, which has been exploited by other studies of efficiency in health care (e.g., Johnson and Rehavi 2016). One way through which integrated firms can affect spending is by engaging in fewer C-sections, which are often costlier than natural births. In our setting, the average C-section has a 13% higher price and a 15% higher insurer payment, both of which are within the range of cost efficiencies we study.

hospitals when vertical integration is banned, and they no longer compete with integrated insurers that set premiums aggressively to steer demand. Hospitals are worse off without integration due to the decrease in prices of formerly integrated hospitals, which no longer have incentives to increase hospital prices to rivals to steer enrollees to their plans. As a result of this decrease in hospital prices, rivals face more competition and either lower prices or lose market share, reducing overall hospital profits. Therefore, banning vertical integration increases overall efficiency and shifts rents from hospitals to consumers and insurers.

Cost efficiencies in the range we consider modify this result only quantitatively. For larger cost efficiencies to the right of Figure 3.3-a, the welfare effect of banning vertical integration remains positive but decreases, reflecting those cost efficiencies are lost in the counterfactual. The effect of banning vertical integration on consumer surplus is smaller for larger cost efficiencies, but that on hospital and insurer profits is mostly constant across them, such that cost efficiencies are mostly passed-through to consumers. However, there is underlying heterogeneity across integrated and non-integrated firms. Figures 3.3-b and 3.3-c show that integrated hospitals (insurers) get more losses (gains) than non-integrated hospitals (insurers) from banning vertical integration, and that the gap increases with cost efficiencies. These patterns can be explained by the pass-through of cost efficiencies to consumers in the form of lower prices by integrated hospitals. By decreasing hospital prices under higher cost efficiencies, integrated firms can increase premiums and still increase profits, bringing them closer to the premiums they would set if non-integrated.[49]

3.5.4 Cost Efficiencies at the Hospital Level as an Antitrust Remedy

Our analysis of cost efficiencies in the previous section is not explicit about their source and assumes they only affect admissions within the integrated firm. Better processing of claims, integration of information systems and reduced managerial costs are all firm-specific sources of synergies. However, vertical integration might also improve the use of resources

49. Integrated firms are more likely to decrease hospital prices than premiums because consumers are more sensitive to the former, making it more appealing for integrated firms to attract consumers with lower hospital prices.

within the hospital and the management of cases. These changes may induce improvements that could potentially lower the costs of admissions of enrollees coming from rival insurers. Regulators may be able to force firms to share such efficiencies with non-integrated insurers through non-discrimination clauses, which may mitigate the potentially adverse effects of vertical integration.

Cost efficiencies at the hospital level do modify our results from the previous section, as shown by Figure C.4. Results for the scenario under no cost efficiencies remains unchanged, by construction. However, we find that higher cost efficiencies partially compensate the distortions introduced by vertical integration. In particular, cost efficiencies higher than 17% imply that banning vertical integration is welfare detrimental in Chile.

3.5.5 The Role of Price Sensitivity for the Effects of Vertical Integration

Our discussion of the effects of vertical integration focuses on steering incentives. Consumer price sensitivity determines the strength of these incentives as integrated firms' ability to steer demand increases when consumers are more price sensitive. However, price sensitivity intensifies competition, limiting the profitability of steering. Which of these effects dominates is theoretically unclear, yet relevant to the overall impact of vertical integration.

We study how our results depend on consumer price sensitivity. In particular, we scale price and premium preferences as $(\tau^M \times \alpha_i^M, \tau^H \times \alpha_f^H)$ for a grid $(\tau^M, \tau^H) \in \{0.5, 0.75, 1, 1.25, 1.5\}^2$, while holding all other estimates fixed. This analysis is therefore comparable with our previous counterfactual analysis, up to consumer price sensitivity. Figures 3.4 and 3.5 display the main results from this analysis, which we implement for a case without cost efficiencies.[50]

Steering incentives vary substantially with consumer price sensitivity. Figure 3.4-a shows that patient-steering incentives are stronger when consumers are more price sensitive and less premium sensitive. In that case, integrated insurers can steer hospital demand by negotiating higher hospital prices with rivals, without decreasing the demand

50. For reference, Figure C.5 displays baseline average hospital prices and plan premiums across our range of scenarios.

for their plans substantially. Similarly, Figure 3.4-b shows that enrollee-steering incentives are stronger when consumers are less price sensitive and more premium sensitive, as in such case integrated hospitals can steer enrollees by negotiating higher hospital prices with rivals and compensating consumers with lower premiums.[51]

Our analysis of steering incentives highlights two channels through which vertical integration distorts outcomes: (i) increasing hospital prices to rivals and (ii) adjusting premiums. However, the extent by which integrated firms exploit each channel vitally depends on consumer price sensitivity. When consumers are more sensitive to prices than to premiums, integrated firms steer demand by increasing hospital prices to rival insurers and decreasing prices to the integrated insurer, as shown by Figures 3.4-c and 3.4-d. Unlike in our baseline scenario, these firms increase their premiums driven by the increase in the relative value of their integrated plan networks with respect to rival insurers', as shown by Figure 3.4-g. On the other hand, when consumers are less responsive to prices than to premiums, integrated insurers decrease premiums to attract enrollees.

The welfare effects of banning vertical integration also vary with price and premium sensitivity, as shown in Figure 3.5. Whereas our main results in Section 3.5.2 show that banning vertical integration would benefit consumers in our setting, the opposite could happen depending on consumers price sensitivity. When consumer price sensitivity is low, and premium sensitivity is high, consumer surplus *decreases* upon banning vertical integration, regardless of the existence of cost efficiencies. This result is partly driven by the substantial increase in premiums of integrated insurers, which more than compensates the decrease in hospital prices by integrated hospitals.[52][53]

51. Additionally, steering becomes costlier when both price and premium sensitivity are low, as it requires larger changes in price and premiums. On the other hand, when consumers are highly price and premium sensitive, rivals can offset steering incentives by slightly reducing premiums and prices. Thus, both of these scenarios limit steering incentives.

52. Interestingly, this can provide some rationale for the success of the integrated system Kaiser, since estimates for hospital price sensitivity are relatively low in the U.S. and close to our case with $(\tau^M, \tau^H) = (1.0, 0.5)$ in Figure 3.5-c.

53. Cost efficiencies caused by vertical integration reduce the benefits from banning vertical integration. Figure C.7 shows the total welfare in a simulation where vertical integration induces a cost efficiency of 20% within the integrated firm. All qualitative results remain the same in this case, yet the benefits from banning vertical integration are smaller, and we can find a scenario in which banning vertical integration would reduce overall welfare.

The connection among price sensitivity, steering incentives and the effects of vertical integration is strong and quantitatively relevant. Consumer price sensitivity limits the ability of integrated firms to exercise the additional market power granted by integration. As a consequence, the effects of vertical integration on consumer surplus vary both in magnitude and sign with consumer price sensitivity, suggesting this dimension might be relevant for antitrust discussions.

3.5.6 Discussion and Limitations

Given the complexity of our counterfactual exercises, we require various assumptions and simplification, thus, we discuss some of the main caveats and limitations. First, we assume that insurers can only adjust premiums while keeping constant the menu of plans, and their coinsurance rates and networks. One could argue that insurers would optimize along these margins in the absence of vertical integration. However, legal restrictions in our application limit this concern: guaranteed renewability of plans implies that plan switching must be voluntary, which limits the extent to which insurers may alter existing insurance plans. In any case, we should expect that having more margins of adjustment would dampen the adverse effects of banning vertical integration on insurer profits, and our estimates of welfare effects may change.

Second, our analysis does not consider changes in fixed costs. If integrated firms share capital that is not captured in per-consumer costs, vertical integration might have implications for entry and consolidation. This aspect lies beyond the scope of our work. However, we observe no entry or exit of large hospitals or insurers during our sample, which suggests these effects might be of second order in our setting.

3.6 Conclusion

This paper proposes an empirical approach for the assessment of the equilibrium effects of vertical integration on market outcomes and welfare. On the theoretical side, we develop an equilibrium model of bargaining between hospitals and insurers, which accommodates vertical integration. Our model highlights two relevant incentives that affect pricing at

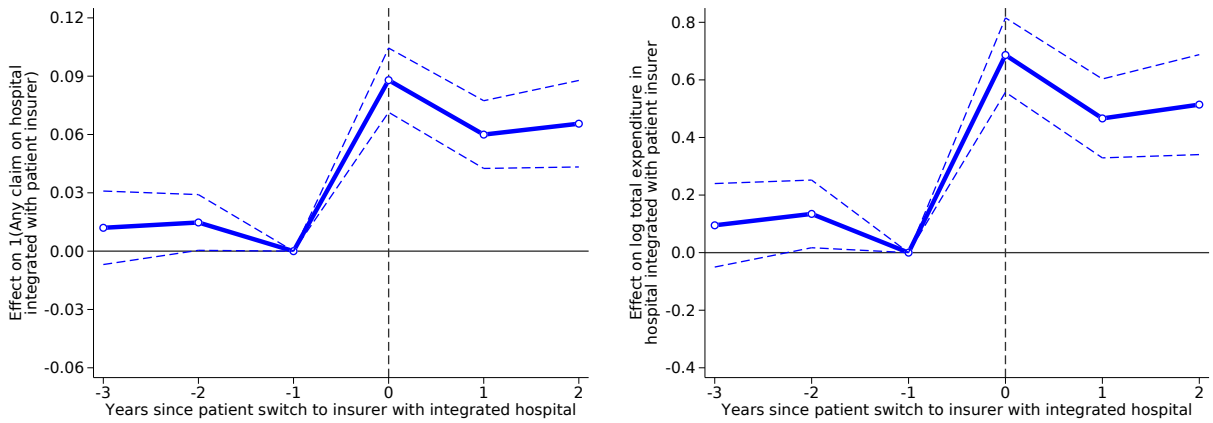
the margin under vertical integration. First, the *patient-steering* effect, by which integrated insurers induce demand to their integrated hospitals by negotiating higher prices with competing hospitals, thus reducing their value within the network. Second, the *enrollee-steering* effect, by which integrated hospitals induce demand to their integrated insurers by negotiating higher hospital prices with competing insurers, thus reducing the value of competing networks.

On the empirical side, our model has implications for researchers and policymakers about the desirability of vertical integration in health markets. We estimate our model using individual-level data from Chile, where private insurers own about half of the private health care sector. Using our estimated model, we quantify the equilibrium effects of vertical integration. We find that banning vertical integration is welfare enhancing in our setting, as the gains in consumer surplus and insurer profit more than compensate decreases in hospital profits. Furthermore, this result does not change qualitatively for a range of cost efficiencies induced by vertical integration.

To further inform the regulation of vertical integration, we explore how our results change under alternative scenarios. First, we examine a situation in which vertical integration creates cost efficiencies at the hospital level that apply to all insurers. In this case, vertical integration can increase welfare for moderate cost efficiencies. We see this as a useful result for antitrust regulation as anti-discrimination clauses can be enforced. Second, we explore the role of price and premium sensitivity for the effects of vertical integration. We find that when consumers are more sensitive to premiums than to hospital prices, integrated firms optimally decrease premiums to attract consumers, enough to increase consumer surplus. This analysis suggests that price and premium sensitivity are relevant inputs in the assessment of vertical mergers.

We see clear avenues to extend our work. First, identifying and quantifying the organizational features that are affected by vertical integration, could clarify the mechanisms of action of vertical integration. For example, specific work could be done on changes to physician incentives and hospital spending under vertical integration. Second, we see the study of the industry characteristics that lead to vertical integration, as a critical pending question for the determination of the desirability of integration in health care markets.

Figure 3.1: Relationship between integration and hospital choices

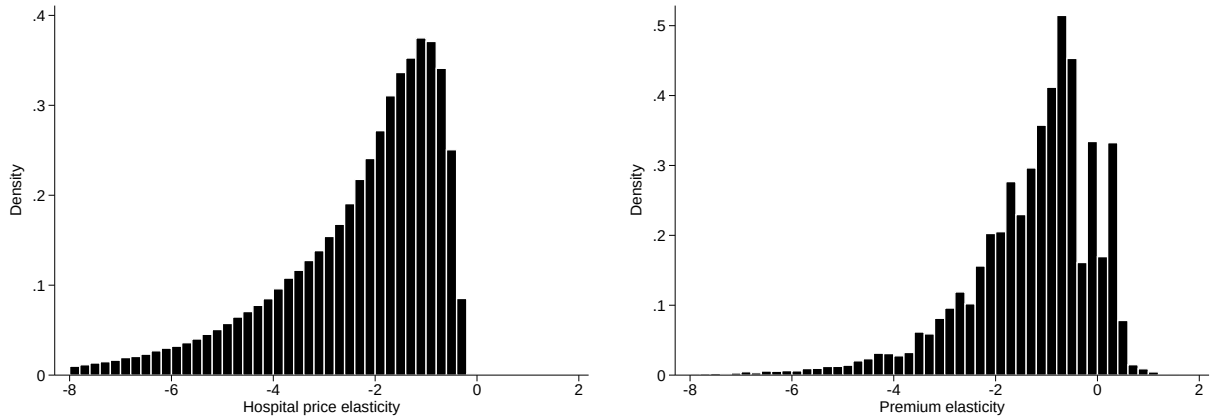


(a) Any claims in integrated hospital

(b) Expenditure in integrated hospital

Notes: This figure displays event study estimates from equation (3.2). Each dot is a coefficient estimate for a year around patients switching insurer. Dashed lines indicate 95% confidence intervals.

Figure 3.2: Histogram of Health Care Price Elasticities and Plan Premium Elasticities elasticities

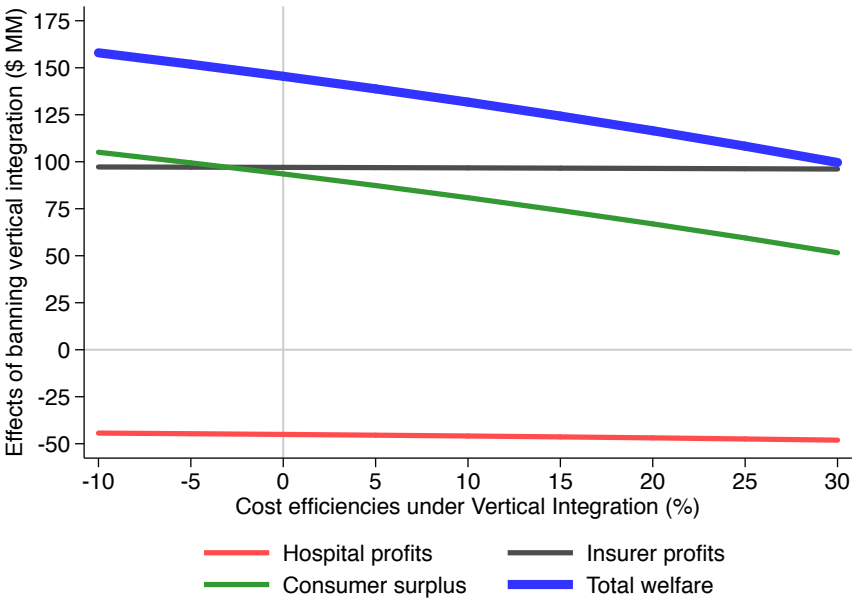


(a) Hospital price elasticity

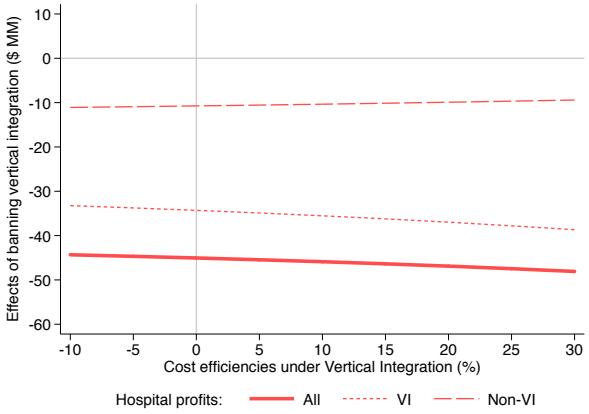
(b) Plan premium elasticity

Notes: Panel (a) displays the histogram of estimated price elasticities for hospitals using estimates in column (1) of Table 3.5. The logit elasticities are given by: $\hat{\eta}_{iht} = \hat{\alpha}_i^H c_{jh} p_{jhdt}(1 - \hat{\sigma}_{ijhdt}^H)$, where $\hat{\sigma}_{ihjt}^H$ is the predicted choice probability of hospital h by consumer type i enrolled in plan j at time t. Panel (b) displays the histogram of premium elasticities for insurance plans using estimates in column (1) of Table 3.6. The logit elasticities are given by: $\hat{\eta}_{fjt} = \hat{\alpha}_f^M \phi_{fjt}(1 - \hat{\sigma}_{fjt}^M)$ is the predicted choice probability of household type f enrolled in plan j at time t.

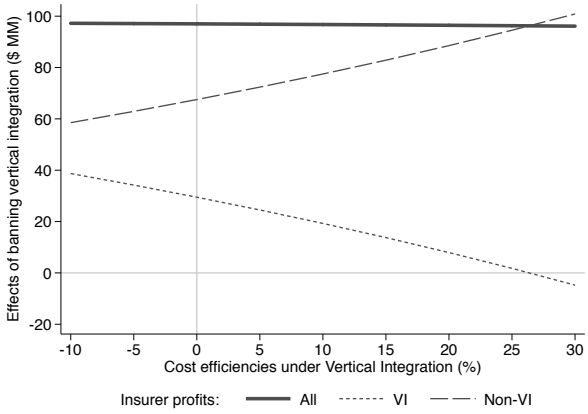
Figure 3.3: Welfare Effects of Banning Vertical Integration for Cost Efficiencies: Chilean Market



(a) Aggregate effects



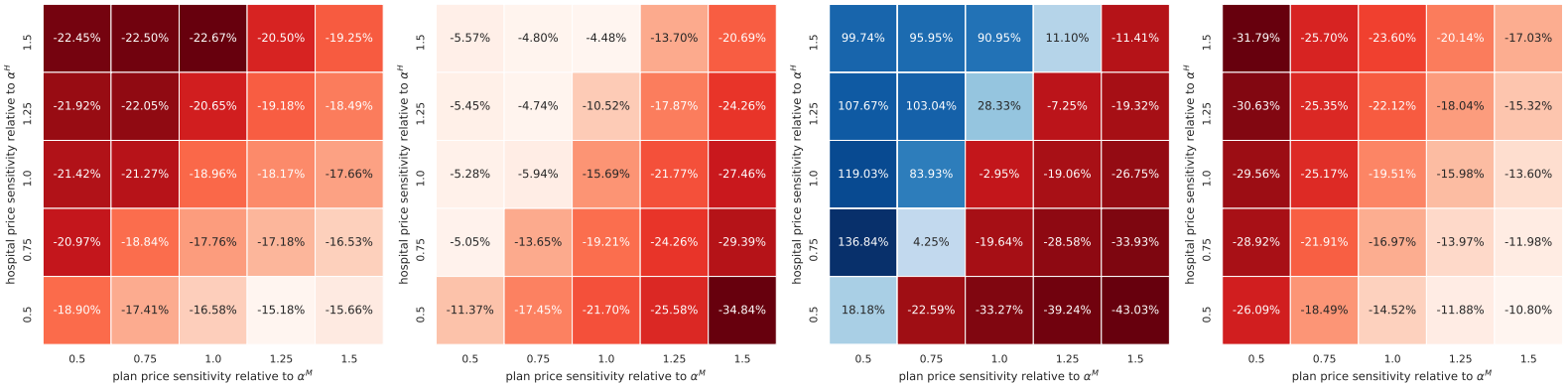
(b) Effects on hospitals, by vertical integration



(c) Effects on insurers, by vertical integration

Notes: This figure shows the effect of banning vertical integration on equilibrium welfare outcomes for different levels of cost efficiencies, for the Chilean market. Panel (a) displays aggregate effects, Panels (b) and (c) respectively decompose effects on hospitals and insurers profits by vertical integration at baseline. The x-axis in each graph measure cost efficiencies induced by vertical integration on integrated hospital-insurer pairs. Blue lines show overall welfare effects, green lines show effects on consumer surplus, red lines show effects on hospital profits, and black lines show effects on insurer profits.

Figure 3.4: The Role of Price Sensitivity for the Effects of Banning Vertical Integration: Outcomes

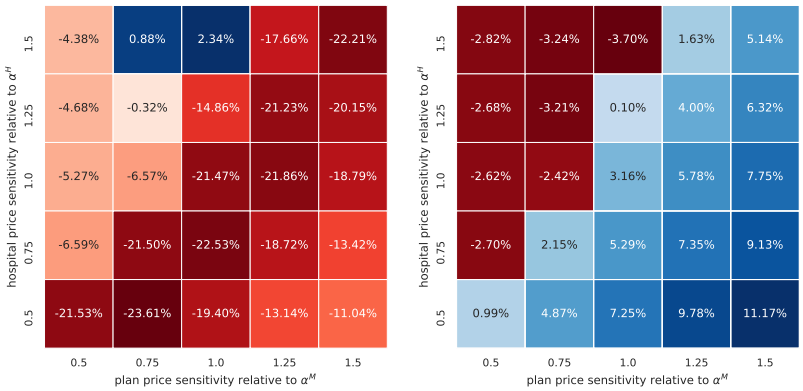


(a) Patient-steering effects (b) Enrollee-steering effects (c) Effects on prices VI to VI (d) Effects on prices VI to non-VI

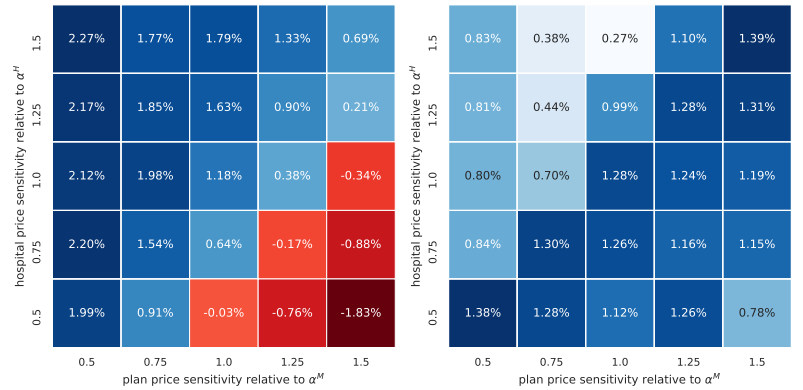
(e) Effects on prices non-VI to VI (f) Effects on prices non-VI to non-VI (g) Effects on VI plan premiums (h) Effects on non-VI plan premiums

Notes: This figure shows the effect of banning vertical integration on a variety of outcomes for a grid of consumer price sensitivity. For each plot, we show results for a 5×5 grid of hospital and plan demand sensitivity defined by $(\tau^M \times \alpha_i^M, \tau^H \times \alpha_f^H)$ for $\tau = \{0.5, 0.75, 1, 1.25, 1.5\}$. Panels (a) and (b) quantify patient- and enrollee-steering effects respectively in the baseline scenario, as described in Section 3.4.5. Panels (c) through (f) display the effect of banning vertical integration on quantity-weighted average hospital prices from a VI/non-VI hospital to a VI/non-VI insurer, and panels (g) and (h) display plan premiums for VI/non-VI insurers. For each such figure, blue (red) indicates increases (decreases) in the outcome, and the intensity of the color indicates the relative magnitude of the change.

Figure 3.5: The Role of Price Sensitivity for the Effects of Banning Vertical Integration: Welfare Effects



(a) Effects on hospital profits (b) Effects on insurer profits



(c) Effects on consumer surplus (d) Effects on total welfare

Notes: This figure shows the effect of banning vertical integration on a variety of outcomes for a grid of consumer price sensitivity. For each plot, we show results for a 5×5 grid of hospital and plan demand sensitivity defined by $(\tau^M \times \alpha_i^M, \tau^H \times \alpha_f^H)$ for $\tau = \{0.5, 0.75, 1, 1.25, 1.5\}$. Panels (a) through (d) display the effect of banning vertical integration on a variety of equilibrium outcomes, measure as a percentage change relative to the baseline level. The outcomes are hospital and insurer profits, consumer surplus and overall welfare. For each such figure, blue (red) indicates increases (decreases) in the outcome, and the intensity of the color indicates the relative magnitude of the change.

Table 3.1: Descriptive Statistics for Plans Dataset

	Panel A - Policyholders attributes					
Variable	N	Mean	SD	p10	p50	p90
Paid premium	1,104,344	0.16	0.09	0.08	0.14	0.27
Policyholder age	1,104,344	40.38	13.41	26.00	37.00	60.00
Policyholder income	1,104,344	1.61	1.13	0.00	1.54	3.03
Single male	1,104,344	0.34				
Single female	1,104,344	0.22				
Has dependents	1,104,344	0.43				
	Panel B - Plan attributes					
Attribute	N	Mean	SD	p10	p50	p90
Inpatient coverage rate	4,358	85.35	23.67	70.00	90.00	100.00
Outpatient coverage rate	4,358	71.83	21.73	60.00	70.00	90.00
Has coverage cap	4,358	0.87				
Has preferential hospital	4,358	0.86				
	Panel C - Insurer market shares and premiums					
	Market	Paid premium				
Insurer	share	Mean	SD	p10	p50	p90
m_1	20.42	0.15	0.09	0.07	0.12	0.26
m_2	17.11	0.19	0.11	0.11	0.17	0.32
m_3	13.72	0.14	0.07	0.06	0.12	0.23
m_4	19.63	0.16	0.08	0.08	0.14	0.26
m_5	25.50	0.15	0.06	0.09	0.14	0.24
m_6	3.63	0.27	0.16	0.13	0.21	0.47

Notes: This table displays descriptive statistics for our estimating plans dataset. Panel A displays statistics across all policyholders in the sample. Panel B displays statistics for plan attributes across al plans in the sample. Panel C displays market shares and premiums paid by policyholders for each insurer in the market. All prices are measured in thousands of U.S. dollars for December, 2014.

Table 3.2: Descriptive Statistics for Admissions Dataset

	Panel A - Admission attributes					
Variable	N	Mean	SD	p10	p50	p90
Full price	641,392	3.79	6.21	0.08	2.25	8.61
Copayment	641,392	1.22	3.03	0.00	0.33	3.25
Coverage	641,392	3.05	4.91	0.34	1.94	6.29
Preferential hospital	641,392	0.38	0.49	0.00	0.00	1.00
Patient age	641,392	37.43	19.44	6.00	37.00	64.00
Policyholder income	641,392	1.88	1.22	0.00	1.95	2.95
Single male	641,392	0.14				
Single female	641,392	0.17				
Has dependents	641,392	0.69				

	Panel B - Hospital market shares and prices					
	Market	Full price				
Hospital	share	Mean	SD	p10	p50	p90
h_1	13.03	6.80	7.91	0.89	4.96	13.50
h_2	4.20	2.70	3.39	0.81	2.04	4.95
h_3	3.82	3.13	4.98	0.75	2.14	6.30
h_4	10.98	3.38	5.48	0.63	2.12	6.22
h_5	9.87	4.32	4.62	1.18	3.61	7.26
h_6	7.51	8.09	10.39	1.39	5.59	16.56
h_7	12.23	5.01	6.71	0.87	3.48	9.69
h_8	2.79	4.37	4.92	0.95	2.99	9.39
h_9	1.37	4.28	6.83	0.32	2.77	8.70
h_{10}	2.34	5.07	5.25	1.22	4.02	8.79
h_{11}	2.67	2.91	3.57	0.95	2.21	5.16
h_{12}	5.14	2.98	5.91	0.58	1.89	5.85
Other	24.05	0.52	1.25	0.01	0.13	1.58

Notes: This table displays descriptive statistics for our estimating admissions dataset. Only admissions on the hospitals in the sample are considered for these statistics. Panel A displays statistics across all hospitals in the sample. Panel B displays statistics for market shares and full prices by hospital. All prices are measured in thousands of U.S. dollars for December, 2014.

Table 3.3: Admission Market Shares and Prices between Hospitals and Insurers

	Panel A - Admission Market Shares						
Hospital	m_1	m_2	m_3	m_4	m_5	m_6	VI share
h_1	15.24	37.53	5.56	24.34	7.57	9.77	0.00
h_2	10.05	10.34	52.62	22.26	3.12	1.61	52.62
h_3	6.30	6.55	63.17	21.80	1.89	0.29	63.17
h_4	67.89	5.21	12.24	9.43	1.86	3.38	71.27
h_5	11.57	25.46	8.46	24.28	27.61	2.62	0.00
h_6	17.98	37.42	5.33	21.12	9.06	9.09	0.00
h_7	44.73	17.88	4.59	17.45	6.14	9.21	53.94
h_8	12.14	17.25	43.38	18.71	4.57	3.95	43.38
h_9	0.43	11.13	22.36	65.14	0.78	0.15	0.00
h_{10}	7.84	64.03	3.20	15.49	5.90	3.54	64.03
h_{11}	63.30	6.30	16.64	9.63	2.51	1.62	64.92
h_{12}	21.60	9.34	46.20	19.78	1.81	1.26	0.00

	Panel B - Admission Full Prices (% of Total Industry Payments)						
Hospital	m_1	m_2	m_3	m_4	m_5	m_6	Total
h_1	2.19	2.10	2.19	2.19	1.34	2.21	12.23
h_2	1.04	0.93	0.81	0.99	1.03	1.09	5.88
h_3	1.26	1.07	0.94	1.08	0.97	1.01	6.33
h_4	1.07	1.06	1.01	1.09	0.80	1.12	6.16
h_5	1.55	1.51	1.36	1.45	1.14	1.63	8.63
h_6	2.37	2.34	2.53	2.47	1.97	2.32	14.00
h_7	1.62	1.57	1.66	1.53	1.12	1.59	9.09
h_8	1.53	1.73	1.31	1.46	1.27	1.51	8.80
h_9	1.70	2.12	1.30	1.21	1.32	0.95	8.60
h_{10}	1.70	1.71	1.31	1.33	1.27	1.77	9.09
h_{11}	1.02	0.99	0.79	0.92	0.70	0.92	5.34
h_{12}	1.06	1.08	0.87	0.84	1.01	0.99	5.84
Total	18.10	18.21	16.08	16.55	13.95	17.12	100.00

	Panel C - Estimated Negotiated Prices (% of Total Industry Payments)						
Hospital	m_1	m_2	m_3	m_4	m_5	m_6	Total
h_1	2.21	1.91	2.86	2.21	1.53	2.26	12.98
h_2	0.82	0.66	0.76	0.83	1.11	0.90	5.09
h_3	0.97	0.91	1.03	0.96	1.00	0.90	5.76
h_4	0.75	0.78	1.07	0.87	0.80	0.79	5.06
h_5	1.38	1.37	1.72	1.34	1.33	1.45	8.59
h_6	2.37	2.27	3.20	2.25	2.22	2.35	14.67
h_7	1.38	1.19	1.86	1.47	1.22	1.39	8.52
h_8	1.24	1.39	1.33	1.30	1.24	1.30	7.79
h_9	2.37	1.98	1.57	1.18	2.10	2.06	11.26
h_{10}	1.64	1.68	2.02	1.78	1.40	1.63	10.15
h_{11}	0.91	0.94	1.03	0.90	0.79	0.87	5.44
h_{12}	0.74	0.74	0.88	0.69	0.99	0.66	4.70
Total	16.79	15.82	19.35	15.75	15.72	16.56	100.00

Notes: The table displays a breakdown of the admissions market shares in Panel A, admission negotiated prices in Panel B, and estimated admission negotiated prices by hospital and insurer pair in Panel C. Vertically integrated pairs are underlined. Panels A and B are calculated from the raw data. Panel C is estimated using the procedure described in Section 3.4.1. The prices are expressed as a percentage of the industry payments.

Table 3.4: Vertical Integration, Hospital Prices and Coverage

	(1)	(2)	(3)	(4)	(5)
	Panel A - OLS estimates on Total bill				
Vertically integrated	-0.291***	-0.031	-0.002	-0.073***	-0.079***
	(0.100)	(0.026)	(0.022)	(0.018)	(0.017)
Observations	545,718	545,718	545,718	545,718	545,716
R-squared	0.026	0.118	0.408	0.411	0.430
	Panel B - OLS estimates on Patient copayment				
Vertically integrated	-0.369***	-0.105***	-0.094**	-0.227***	-0.230***
	(0.103)	(0.039)	(0.040)	(0.031)	(0.031)
Observations	545,718	545,718	545,718	545,718	545,716
R-squared	0.041	0.176	0.304	0.430	0.444
	Panel C - OLS estimates on Insurer coverage				
Vertically integrated	-0.148*	0.021	0.048***	0.044*	0.039*
	(0.076)	(0.023)	(0.016)	(0.022)	(0.022)
Observations	545,718	545,718	545,718	545,718	545,716
R-squared	0.007	0.055	0.320	0.370	0.384
Hospital FEs	N	Y	Y	Y	Y
Diagnosis FEs	N	N	Y	Y	Y
Diagnosis public prices	N	N	Y	Y	Y
Insurer controls	N	N	N	Y	Y
Patient controls	N	N	N	N	Y

Notes: This table shows results from estimating equation (3.1) using the log amount of total bill (Panel A), patient copayment (Panel B), and insurer coverage (Panel C) as the dependent variables (we add 500 USD to avoid zero amounts). Each column includes a different set of control variables. Diagnosis fixed effects are based on ICD10 chapters, and diagnosis public system prices are the prices of the same admissions in public hospitals. Insurer controls include insurer fixed effect, plan premium, coinsurance rate for inpatient and outpatient admissions, and dummies for whether the plan has a coverage cap and a preferential hospital. Patient controls include gender, age, income, number of dependents, an indicator for independent worker and fixed effects by county of residence. The sample considers the admissions in the 12 main private hospitals. Standard errors in parentheses are clustered by insurer-hospital combination. P-values notation: *** p<0.01, ** p<0.05, * p<0.1.

Table 3.5: Demand and Elasticities for Health Care

	(1)	(2)	(3)	(4)	(5)
	All	All	All	Age ≤ 45	Age > 45
Panel A - Preferences estimates					
α^H - Hospital price					
Age ≤ 25	-0.829***	-2.150***	-2.133***	-2.639***	
	(0.010)	(0.011)	(0.011)	(0.017)	
Age ∈ (25, 45]	-0.903***	-2.126***	-2.168***	-2.644***	
	(0.009)	(0.010)	(0.010)	(0.015)	
Age ∈ (45, 60]	-0.984***	-2.214***	-2.078***		-1.558***
	(0.009)	(0.011)	(0.011)		(0.013)
Age > 60	-0.884***	-2.135***	-1.970***		-1.489***
	(0.009)	(0.011)	(0.011)		(0.013)
Single female	0.250***	0.454***	0.441***	0.796***	0.165***
	(0.009)	(0.010)	(0.010)	(0.015)	(0.012)
Dependents	0.224***	0.477***	0.375***	0.682***	0.169***
	(0.008)	(0.009)	(0.009)	(0.014)	(0.011)
Income 2nd quartile	-0.298***	-0.273***	-0.294***	-0.285***	-0.288***
	(0.007)	(0.007)	(0.007)	(0.010)	(0.010)
Income 3rd quartile	0.144***	0.061***	0.083***	0.167***	-0.040***
	(0.007)	(0.007)	(0.007)	(0.009)	(0.010)
Income 4th quartile	0.504***	0.456***	0.495***	0.631***	0.295***
	(0.006)	(0.006)	(0.007)	(0.009)	(0.010)
β_v - Distance to hospital	-0.086***	-0.091***	-0.094***	-0.101***	-0.083***
	(0.000)	(0.000)	(0.000)	(0.001)	(0.001)
Panel B - Price elasticities					
Mean	-1.290	-2.322	-2.396	-2.607	-2.015
SD	0.993	1.702	1.762	1.905	1.487
p10	-2.640	-4.666	-4.827	-5.242	-4.048
p50	-1.001	-1.819	-1.877	-2.050	-1.574
p90	-0.354	-0.686	-0.701	-0.762	-0.594
Observations	7,899,554	7,899,554	7,899,554	5,098,860	2,800,694
Hospital FEs	N	Y	N	N	N
Hospital-diagnosis FEs	N	N	Y	Y	Y

Notes: Panel A shows demand estimates for hospitals. The price coefficient varies across age groups, household composition and income. The single male category is the baseline. The price coefficient for another specific group is the sum of the age-group for single male plus the price coefficient of that group. $income_i$ corresponds to the taxable income of the consumer as recorded in the administrative data. Panel B displays the summary statistics for the individual estimated price elasticities. Namely, $\hat{\eta}_{iht} = \hat{\alpha}_i^H c_{jh} p_{ijhdt}(1 - \hat{\sigma}_{ijhdt}^H)$, where $\hat{\sigma}_{ijhdt}$ is the predicted choice probability of hospital h by consumer type i enrolled in plan j under diagnosis d at time t. Robust standard errors in parentheses. P-value notation: ***p<0.01, ** p<0.05, * p<0.1.

Table 3.6: Demand Estimates and Elasticities for Insurance Plans

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	α^M - Plan premium					β - Expected utility from health care				
	All	All	All	Age ≤ 45	Age > 45	All	All	All	Age ≤ 45	Age > 45
Panel A - Preferences estimates										
Age ≤ 25	-11.038***	-4.330***	-15.839***	-19.889***		3.062***	2.865***	5.871***	11.027***	
	(0.161)	(0.177)	(0.223)	(0.321)		(0.056)	(0.061)	(0.071)	(0.106)	
Age ∈ (25, 45]	-6.016***	-2.646***	-6.486***	-14.385***		4.331***	4.105***	5.492***	10.347***	
	(0.054)	(0.055)	(0.097)	(0.253)		(0.025)	(0.028)	(0.034)	(0.083)	
Age ∈ (45, 60]	-7.840***	-5.210***	-8.552***		-3.945***	3.975***	3.733***	4.910***		5.246***
	(0.060)	(0.060)	(0.092)		(0.133)	(0.026)	(0.030)	(0.036)		(0.048)
Age > 60	-3.831***	-2.217***	-4.805***		0.375***	2.187***	1.879***	2.283***		2.416***
	(0.057)	(0.056)	(0.082)		(0.108)	(0.024)	(0.026)	(0.030)		(0.038)
Single female	-1.053***	-0.971***	-0.409***	0.606**	0.724***	-1.385***	-0.821***	-0.116***	-5.464***	0.781***
	(0.058)	(0.061)	(0.087)	(0.257)	(0.113)	(0.026)	(0.026)	(0.029)	(0.076)	(0.037)
Dependents	-3.200***	-4.497***	-2.747***	-2.484***	-1.609***	-2.777***	-2.350***	-2.630***	-8.175***	-2.076***
	(0.044)	(0.043)	(0.068)	(0.244)	(0.082)	(0.023)	(0.016)	(0.028)	(0.077)	(0.032)
Income 2nd quartile	-8.422***	-8.746***	-9.407***	-7.743***	-8.128***	-0.203***	-0.163***	-0.059***	0.309***	-0.192***
	(0.067)	(0.068)	(0.070)	(0.103)	(0.095)	(0.016)	(0.025)	(0.017)	(0.022)	(0.025)
Income 3rd quartile	1.467***	1.334***	1.268***	8.157***	-3.287***	0.648***	0.657***	0.748***	1.269***	0.492***
	(0.055)	(0.056)	(0.058)	(0.088)	(0.077)	(0.015)	(0.016)	(0.016)	(0.022)	(0.023)
Income 4th quartile	8.817***	8.664***	9.101***	19.940***	2.569***	0.731***	0.688***	0.674***	1.199***	0.714***
	(0.049)	(0.049)	(0.053)	(0.086)	(0.058)	(0.016)	(0.015)	(0.017)	(0.024)	(0.024)
Panel B - Premium elasticities										
Mean	-1.216	-0.847	-1.319	-1.484	-1.038					
SD	1.314	1.286	1.411	1.807	1.065					
p10	-2.757	-2.344	-2.976	-3.580	-2.293					
p50	-0.926	-0.503	-1.012	-1.330	-0.842					
p90	0.180	-0.503	0.173	0.668	0.081					
Observations	44,276,610	44,276,610	44,276,610	30,234,540	14,042,070	44,276,610	44,276,610	44,276,610	30,234,540	14,042,070
Insurer FEs	N	Y	N	N	N	N	Y	N	N	N
Plan FEs	N	N	Y	Y	Y	N	N	Y	Y	Y

Notes: Panel A shows the logit estimates of the demand for insurance plans. The premium and expected utility of health care coefficients vary across age groups, household composition, and income. Different columns show estimates considering a different set of fixed effects. Columns (1) and (3) consider insurer fixed effects, while columns (2) and (4) consider insurance plan fixed effects. Panel B displays the summary statistics for the individual estimated price elasticities for the chosen plans. Namely, $\hat{\eta}_{fjt} = \hat{\alpha}^M_f \phi_{fjt}(1 - \hat{\sigma}^M_{fjt})$, where $\hat{\sigma}^M_{fjt}$ is the predicted choice probability of plan j by household type f in time t. Robust standard errors in parentheses. P-value notation: *** p<0.01, ** p<0.05, * p<0.1.

Table 3.7: Estimated Bargaining Weights and Marginal Costs

		(1)	(2)
Negotiating firms		Mean	SD
Panel A - Hospital Bargaining Weights ($1-\lambda$)			
All Hospitals and Insurers		0.515	0.374
Non-VI hospital and Non-VI insurer		0.696	0.025
Non-VI hospital and VI insurer		0.949	0.038
VI hospital and Non-VI insurer		0.083	0.083
VI hospital and VI insurer from different holding		0.423	0.286
Panel B - Marginal Costs and Mark-ups	Outcome	Mean	SD
All Hospitals	Marginal cost	3.032	2.053
	Negotiated price	4.504	2.041
	Mark-up	0.383	0.254
Integrated hospitals only	Marginal cost	2.113	1.137
	Negotiated price	3.404	1.103
	Mark-up	0.400	0.231
Non-integrated hospitals only	Marginal cost	3.950	2.338
	Negotiated price	5.605	2.171
	Mark-up	0.366	0.276
Integrated hospital to own VI insurer only	Marginal cost	2.316	1.573
	Negotiated price	3.332	1.203
	Mark-up	0.332	0.363
Integrated hospital to other insurers	Marginal cost	2.062	1.004
	Negotiated price	3.422	1.082
	Mark-up	0.416	0.182

Notes: Non-VI stands for non-integrated, and VI stands for vertically integrated. Panel A displays summary statistics for the estimates of hospital bargaining weights (i.e. $1-\lambda_{ms}$). Each row provides statistics for a different combinations of negotiators. Panel B displays summary statistics for the estimated hospital marginal costs; the estimated negotiated prices as estimated in Section 3.4.1, and the implied hospital mark-up different for different subsample of hospitals.

Table 3.8: Counterfactual Simulation of Banning Vertical Integration

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	Hospital prices			Market shares			Total profits			Mark-ups		
Panel A - Hospitals	Base	CF	Δ%	Base	CF	Δ%	Base	CF	Δ%	Base	CF	Δ%
VI to own-VI	3.368	3.279	-2.629%	0.174	0.143	-18.169%	48.800	40.109	-17.809%	0.527	0.528	0.181%
VI to non-VI	3.470	2.793	-19.517%	0.327	0.380	16.108%	75.264	49.647	-34.036%	0.420	0.288	-31.444%
Non-VI to VI	5.609	5.560	-0.881%	0.143	0.142	-0.880%	44.267	34.455	-22.165%	0.348	0.268	-23.059%
Non-VI to non-VI	5.360	5.391	0.588%	0.355	0.335	-5.579%	44.430	43.499	-2.097%	0.148	0.148	0.112%
Non-VI to all	5.431	5.441	0.183%	0.499	0.477	-4.227%	88.697	77.954	-12.113%	0.207	0.185	-11.040%
	Plan premiums			Market shares			Total profits			Costs per enrollee		
Panel B - Insurers	Base	CF	Δ%	Base	CF	Δ%	Base	CF	Δ%	Base	CF	Δ%
VI at baseline	2.210	2.297	4.563%	0.206	0.195	-1.447%	599.858	614.598	3.693%	0.007	0.007	-1.425%
Non-VI at baseline	2.384	2.374	-0.351%	0.196	0.203	3.599%	604.922	627.428	3.751%	0.014	0.014	-3.270%

Panel C - Consumers	Share of market	Premium sensitivity	Δ Consumer surplus
Female 0-24	1.482%	-21.480	-0.002
Female 25-44	22.747%	-11.332	0.025
Female 45-60	9.510%	-7.954	0.075
Female 60+	4.204%	-4.151	0.200
Male 0-24	4.633%	-24.093	-0.007
Male 25-44	36.839%	-9.879	0.043
Male 45-60	14.562%	-7.069	0.070
Male 60+	6.022%	-3.758	0.132
Weighted average		-9.838	0.055

Notes: This table displays results from a counterfactual in which vertical integration is banned in the market and all vertical linkages are removed. Non-VI stands for non-integrated, and VI stands for vertically integrated. Panel A displays outcomes in the health care market, in which shares are weighted by resource intensity weights. Changes are market-size weighted averages per hospital, averaged by the level indicated on the leftmost column. Profits and prices are in USD thousands, with profit being hospital annual averages. Panel B displays yearly averages over insurers, weighted by the market size. Premiums are averaged at the plan level, while share, costs and profits correspond to insurer level averages. Baseline values are expressed in thousands of dollars, with profits being total yearly values averaged over all insurers. Panel C shows consumer surplus change per consumer, measured in thousands of dollars per year.

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APPENDIX A
PRICE REGULATION IN CREDIT MARKETS: A TRADE-OFF BETWEEN CONSUMER PROTECTION AND CREDIT ACCESS

A.1 Robustness Exercises and Additional Results

The empirical strategy developed in Section 1.3.2 exploits policy variation across time and loan-size brackets to measure the effect of interest rate caps on credit market outcomes. The main concern with this strategy is the potential for equilibrium effects on loans larger than \$8,000, which are not directly treated by the policy change, but might be affected through some form of substitution along that margin. In this section, we implement different robustness exercises that provide support to our empirical strategy. Moreover, we include some additional results.

A.1.1 Effects of Placebo Policies

Our approach in Section 1.3.2 relies on a comparison across treated and untreated loan-size brackets. One should not expect to find the same estimated effects across different comparison groups within untreated loan size brackets. We study whether that is the case, by estimating equation (1.4) for placebo policies. Concretely, we use the same definition for the policy and the same policy intensity variables as in Section 1.3.2 to estimate effects of interest rate regulation on different parts of the loan size distribution. In practice, we proceed by replacing the dependent variable y_{krt} to $y_{k+\Delta,rt}$, where Δ defines the placebo policy. We start by policy size brackets defined as being \$8,000 higher than actual ones, and then sequentially increase them by \$2,000 to generate a range of placebo policies.

Figure A.8 displays the results from this exercise for price and quantity outcomes. Each figure displays our main estimates from Table 1.3 and Table 1.4, along with estimates for a range of placebo policies. Figures A.8-a and A.8-b display results for maximum and average interest rates, and the results are stark: estimates from placebo policies are remarkably different from our estimates and close to zero. Figures A.8-c and A.8-d display results for quantity outcomes, for which placebo estimates are noisier but offer a similar

pattern: most of point estimates are close to zero and not statistically different to zero. These results provide evidence against particular patterns of substitution from untreated loan size brackets to treated loan size brackets.

A.1.2 Effects on Distribution of Application Loan Size and Term

An additional robustness exercise we implement is to study the evolution of the distribution of application amount and term. If there substitution across policy size brackets, that should reflected in a change in the distribution of application loan amount. Figure A.9-a shows the evolution of relevant statistics of the distribution of application loan amount separately for loans smaller than $8,000 and loans larger than $8,000. We find no evidence of changes in this distribution around the date of the policy change we study. Moreover, the same is true for application loan term, as displayed by Figure A.9-b.

A.1.3 Effects around Policy Thresholds for Loan Size

The approach proposed in Section 1.3.2 exploits loans larger than $8,000 as a control group for evaluating the effect of the policy. One concern regarding that is that, in response to the change in relative interest rate regulation between loans under and above that threshold, there could be equilibrium effects affecting loans larger than $8,000 despite them not being directly treated by the reform we study. For example, one could argue that, before the reform, borrowers could have sought to get loans right above the $8,000 threshold to benefit from lower interest rate caps on loans in that loan-size bracket than on loans of amounts marginally below such threshold. That incentive would be reduced by the policy, given interest rate caps for both groups were brought closer by it. In such case, we should observe bunching at that threshold from above before the policy, and a decrease in such behavior after it. That in turn would imply that our results in Section 1.3.2 would underestimate the effects of the reform. On the other hand, banks may have opposite incentives to induce borrowers to take marginally smaller loans on the left side of the policy threshold or to take multiple small loan instead of a large one, for which we already provided evidence against in Figure A.7. This incentive would also decrease under stronger regulation.

In order to address this concern about substitution around the threshold, we study the distribution of the number of loans around relevant policy thresholds. Figure A.10 displays the number of loan originations at loan sizes around relevant policy thresholds, before and after the policy. As displayed by Figure A.10-a, The relationship between the density of loan size below the $2,000 threshold is remarkably noisy—this pattern is driven by mass points in the loan size distribution that are observed at certain round number for loan size—, which makes it difficult to conclude anything. However, above the $2,000 threshold, there is no noticeable change in such density before and after the policy. A similar comparison is displayed by Figure A.10-b for the $8,000 policy threshold. While the distribution of the number of loans shows more mass around the policy threshold after the policy, that behavior is similar on both sides of threshold.[1]

In a more systematic attempt to address this concern, we repeat the analysis in Section 1.3.2 dropping loans around the $8,000 policy threshold. Table A.2 displays results from estimating equation (1.4) excluding loans of size between $6,000 and $10,000 from the sample. Estimates are quantitatively similar to those obtained with the full sample in Section 1.3.2, which is reassuring in terms of our empirical strategy. Finally, Figure A.11 repeats this exercise for price and quantity outcomes for a variety of comparison groups which differ in their lower bound, and provides evidence in the same direction: estimates for the effects of the policy do not change substantially when excluding loans close to the policy threshold from the comparison group.

A.1.4 Alternative Comparison Groups

Our analysis in Section 1.3.2 exploits loans between $8,000 and $20,000 as a comparison group for those directly affected by the policy change. In this subsection, we assess how would our estimates change under alternative definitions of compares groups. In particular, we estimate the same specification as in (1.4) but for variety of comparison groups, starting

1. Figures A.10-c and A.10-d complement this analysis by showing that average interest rates shift downwards after the policy, but that there is no discontinuity in average interest rates around policy thresholds. We should mention that when looking at high enough percentiles in the distribution of loan interest, discontinuities at the policy thresholds become evident, which is consistent with bunching at the interest rate cap displayed in Figure 1.2.

with loans between $8,000 and $10,000, and then increasing sequential by $2,000 until a group covering loans between $8,000 and $30,000.

Results from this exercise are displayed in Figure A.12, and show results for price and quantity outcomes. Each figure displays our main estimates from Table 1.3 and Table 1.4, along with estimates for a range of alternative comparison groups. Overall, the main conclusions of our main analysis are unaffected, as point estimates do not change substantially across comparison groups. On the other hand, there are some efficiency gains from using larger comparison groups, which reflects in tighter standard errors.

A.1.5 Heterogeneity across Banks

We have focused so far on the effects of interest rate regulation at the market level. In this subsection, we provide results for heterogeneity in effects across banks. Figure A.13 provides results for marginal effects of interest rate regulation on both number of lines and prices for each of the 8 largest banks in the market. While there heterogeneity in magnitudes, our estimates suggest that the stronger interest rate regulation affects most banks in the same direction, by inducing them to sign less loan contracts and to do so at lower interest rates. This is consistent with our estimates for average effects and with the interpretation we give to them.

Interestingly, there is 1 bank that displays a different behavior, by reducing average interest but simultaneously increasing credit volume as a result of stronger interest rate regulation. Those estimates suggest that either borrowers substituted towards that bank which perhaps had a more lenient screening process or that the bank changed its screening process as a result of the policy change.

A.2 Estimation Details

A.2.1 Preliminary Steps in Estimation of Application Equation

We discuss joint estimation of the application and repayment equations in (1.11) and (1.12) in Section 1.5.3. Estimation proceeds in three steps, of which the first two are related

to estimation of components of the application equation in equation (1.11) that are not observed for every consumer in the sample, and that we then use as inputs in estimation of the key parameters in that equation by maximum likelihood. We provide further detail about those steps in this section.

In the first step,we estimate the conditional distribution of loan amount and term (L_i, T_i) using data from applicants and then draw from that distribution for non-applicants. In the first stage, we estimate a probit model for applications on a rich vector of borrower covariates x_i that includes the level and change of consumer and mortgage debt and default, income, consumer and mortgage debt to income ratio, age and gender; and application shifters z_i that include the total number of branches across banks in the consumer local market and the lagged number of related banks:

$$P(a_i = 1) = \Phi(x_i'\beta_a + z_i'\gamma_a)$$

which we estimate this model on data for the period before the policy change in December 2013. In the second stage, we estimate a regression of loan amount on the same vector of borrower covariates, and include fitted propensity scores $\hat{P}(x_i, z_i)$ as a control function to account for selection into application. This procedure is based on Das et al. (2003) and also used by Attanasio et al. (2008) for studying loan demand. In particular, we estimate:

$$\ln(L_i) = x_i'\beta_L + \lambda(\hat{P}(x_i, z_i)) + \epsilon_i$$

where λ is the control function. In the third stage, we estimate an ordered logit model for loan term monthly bins on borrower covariates and loan size:

$$\begin{aligned} P(T_i = j) &= P(\alpha_{j-1} < x_i'\beta_T + \delta_T L_i \leq \alpha_j) \\ &= \Lambda(\alpha_j - (x_i'\beta_T + \delta_T L_i)) - \Lambda(\alpha_j - (x_i'\beta_T + \delta_T L_i)) \end{aligned}$$

where α_{j-1} and α_j are the cutoffs that define loan term monthly bin j. The advantage of using an ordered logit model in this step is that it accommodates the fact that the empirical distribution of loan term features noticeable spikes at multiples of semesters.

Using estimates from these regressions, we first take draws of loan amount for consumers that did not apply for loans $\tilde{L}_i$, conditional on x_i; and then take draws of loan term for consumers that did not apply for loans $\tilde{T}_i$, conditional on x_i and L_i.

In the second step, we estimate the approval probability P_C and the density of loan prices conditional on approval $f_{p|C}$. We estimate P_C using a probit model for an indicator of application approval on a vector of borrower covariates, previous relationship variables, as well as application amount and term from the first step and month fixed and market effects:

$$P(C_i = 1) = \Phi(x_i'\eta + \zeta_t + \tau_m)$$

which we estimate separately for low- and high-risk borrower to allow for flexibility. We include time and market fixed effects in order to accommodate the possibility that approval probabilities change over time and across markets due to the policy change. We compute expected approval probabilities $\hat{P}_{Ci}$ as the fitted values from this equation for each consumer in the sample. Then, we estimate the density of loan monthly payments conditional on approval $f_{p|C}$, using a kernel density estimator after conditioning on the same vector of variables. We then take draws from this estimated conditional density for estimation of borrower preferences by maximum likelihood below.

A.2.2 Likelihood Function for Application and Repayment

The parameters of interest in the application equation are those in v_{Ci}, δ_p and κ_i, whereas the parameters of interest in the repayment equation are α_S. Moreover, we are interested in the parameters in the joint distribution of application and repayment shocks, namely ρ and σ_S. Recall that σ_A is normalized to 1.

We start by the likelihoods of application choices. Given the normality assumption we impose on ε_{Ai}, the probabilities that a potential borrower chooses to apply and not to

apply are:

$$\begin{aligned}
P_{a_i=1} &= \Pr\left(P_{Ci}\int(x_i'\delta_X+\delta_L L_i+\delta_T T_i-\delta_p p)f_{p|C}(p)dp-z_i'\kappa+\varepsilon_{Ai}>0\right)\\
&= \Phi\left(P_{Ci}\int(x_i'\delta_X+\delta_L L_i+\delta_T T_i-\delta_p p)f_{p|C}(p)dp-z_i'\kappa\right)\\
P_{a_i=0} &= \Pr\left(P_{Ci}\int(x_i'\delta_X+\delta_L L_i+\delta_T T_i-\delta_p p)f_{p|C}(p)dp-z_i'\kappa+\varepsilon_{Ai}<0\right)\\
&= \Phi\left(-P_{Ci}\int(x_i'\delta_X+\delta_L L_i+\delta_T T_i-\delta_p p)f_{p|C}(p)dp+z_i'\kappa\right)
\end{aligned}$$

where Φ is the standard normal cdf. We approximate the integral through simulation by taking $N_A^S = 100$ Halton draws from the estimated conditional density of loan monthly payments conditional on approval, which is:

$$\int(x_i'\delta_X+\delta_L L_i+\delta_T T_i-\delta_p p)f_{p|C}(p)dp \approx \frac{\sum_{s=1}^{N_A^S}(x_i'\delta_X+\delta_L L_i+\delta_T T_i-\delta_p p_i^{(s)})1\{L_i^{(s)}=1\}}{\sum_{s=1}^{N_A^S}1\{L_i^{(s)}=1\}}$$

where $L_i^{(s)}$ is an indicator for whether a loan application by borrower i in simulation draw s was approved.

We now derive the likelihoods of repayment outcomes. The likelihood of observing a given repayment behavior can be written in terms of the distribution of ε_{Si} conditional on the event of application, for which we exploit the properties of conditional normal distributions. There are three cases of interest, one in which borrower i fully repays, one in which borrower i partially repays, and one in which borrower i does not repay at all. The

probabilities for these three cases are:

$$
\begin{aligned}
P_{S=0|a_i=1} &= \Pr(\exp(x_i'\alpha_S + \varepsilon_{Si}) \leq \chi | a_i = 1) \\
&= \int_{-P_{Ci}\int(x_i'\delta_X+\delta_L L_i+\delta_T T_i-\delta_p p)f_{p|C}(p)dp+z_i'\kappa}^{\infty} \Phi\left(\frac{\ln(\chi) - x_i'\alpha_S - \mu_{S|Ai}}{\sigma_{S|A}}\right) f_{\varepsilon_A}(\varepsilon_A)d\varepsilon_A \\
P_{S=s_i|a_i=1} &= \Pr(\exp(x_i'\alpha_S + \varepsilon_{Si}) = s_i | a_i = 1) \\
&= \int_{-P_{Ci}\int(x_i'\delta_X+\delta_L L_i+\delta_T T_i-\delta_p p)f_{p|C}(p)dp+z_i'\kappa}^{\infty} \frac{1}{\sigma_{S|A}}\phi\left(\frac{\ln(s_i) - x_i'\alpha_S - \mu_{S|Ai}}{\sigma_{S|A}}\right) f_{\varepsilon_A}(\varepsilon_A)d\varepsilon_A \\
P_{S=1|a_i=1} &= \Pr(\exp(x_i'\alpha_S + \varepsilon_{Si}) \geq 1 | a_i = 1) \\
&= \int_{-P_{Ci}\int(x_i'\delta_X+\delta_L L_i+\delta_T T_i-\delta_p p)f_{p|C}(p)dp+z_i'\kappa}^{\infty} \Phi\left(\frac{x_i'\alpha_S + \mu_{S|Ai}}{\sigma_{S|A}}\right) f_{\varepsilon_A}(\varepsilon_A)d\varepsilon_A
\end{aligned}
$$

where $\chi = \frac{1}{T_i} \approx 0$ is the repayment share achieved after the first payment on the contract, and ϕ is the standard normal pdf. Given the joint normality assumption of $(\varepsilon_{Ai}, \varepsilon_{Si})$, the conditional distribution of ε_S is given by:

$$\varepsilon_S | \varepsilon_A \sim N\left(\frac{\rho\sigma_S}{\sigma_A}\varepsilon_A, \sigma_S^2(1-\rho^2)\right)$$

which we use for computing the integral in each likelihood by simulation. In particular, we take 100 Halton draws $\varepsilon_{Ai}^{(s)}$ from its truncated marginal distribution, and then compute the conditional mean of ε_{Si} for each draw, $\mu_{S|Ai}^{(s)} = \frac{\rho\sigma_S}{\sigma_A}\varepsilon_{Ai}^{(s)}$.

The likelihood of an observation varies across five combinations of application and repayment outcomes we observe in the data, which we identify by indicators I_i^1 through I_i^5. The log likelihood of the data is:

$$
\begin{aligned}
\log \mathcal{L}^D = \frac{1}{N}\sum_i & I_{1i}\log P_{a_i=0} + I_{2i}\log P_{a_i=1} + I_{3i}[\log P_{a_i=1} + \log P_{S=0|a_i=1}] \\
& + I_{4i}[\log P_{a_i=1} + \log P_{S=s_i|a_i=1}] + I_{5i}[\log P_{a_i=1} + \log P_{S=1|a_i=1}]
\end{aligned}
$$

where I_{1i} indicates non-applicates, I_{2i} indicates rejected applicants, I_{3i} indicates applicants that do not pay any monthly payment, I_{4i} indicates applicants that only partially repay their contract, and finally I_{5i} indicates applicants that fully repay their contract. This log

likelihood can in turn can be written as:

$$
\begin{aligned}
\log \mathcal{L}^D \;=\; & \frac{1}{N}\sum_i I_{1i}\log P_{a_i=0} + (I_{2i}+I_{3i}+I_{4i}+I_{5i})\log P_{a_i=1} \\
& + I_{3i}\left[\log \int_{-P_{Ci}}^{\infty} \int_{(x_i'\delta_X+\delta_L L_i+\delta_T T_i-\delta_p p)f_{p|C}(p)dp+z_i'\kappa} F_{\varepsilon_D|\varepsilon_A}(\ln(\chi)-x_i'\alpha_S|\varepsilon_A) f_{\varepsilon_A}(\varepsilon_A)d\varepsilon_A\right] \\
& + I_{4i}\left[\log \int_{-P_{Ci}}^{\infty} \int_{(x_i'\delta_X+\delta_L L_i+\delta_T T_i-\delta_p p)f_{p|C}(p)dp+z_i'\kappa} f_{\varepsilon_D|\varepsilon_A}(\ln(s_i)-x_i'\alpha_S|\varepsilon_A) f_{\varepsilon_A}(\varepsilon_A)d\varepsilon_A\right] \\
& + I_{5i}\left[\log \int_{-P_{Ci}}^{\infty} \int_{(x_i'\delta_X+\delta_L L_i+\delta_T T_i-\delta_p p)f_{p|C}(p)dp+z_i'\kappa} F_{\varepsilon_D|\varepsilon_A}(x_i'\alpha_S|\varepsilon_A) f_{\varepsilon_A}(\varepsilon_A)d\varepsilon_A\right]
\end{aligned}
$$

We maximize this log-likelihood to estimate the parameters of interest in the application and repayment equations.

A.2.3 Likelihood Function for Banks' Costs

The parameters of interest on the supply side are $\{\tau, \gamma, \sigma_\omega\}$. The likelihood function for prices and loan application outcomes can be obtained using results for the distributions of order statistics of the T1EV distribution assumed for ω_{ij}, as detailed in Appendix A.2.4. We work separately on the corresponding likelihood for each of the three potential outcomes generated by the model and stated in equation (1.9). In terms of notation, we employ uppercase letters for random variables and lowercase variables for data.

Likelihood for Unconstrained Loans. When regulation is not binding, loan price is the optimal unconstrained price for the lowest cost bank. The likelihood of a contract in such

situation, signed at price $p_i \leq \bar{p}_i$ with bank b_i is:

$$\begin{aligned} P(P_i = p_i, P_i < \bar{p}, B_i = b_i|\mathcal{J}_i) &= P(P_i^u = p_i, P_i^u < \bar{p}_i, B_i = b_i|\mathcal{J}_i) \\ &= \left(g_{i(1)} \left(\frac{f_i - \frac{E_\varepsilon[\varphi(S_i)]}{\varphi(T_i)} p_i}{l_i} | \mathcal{J}_{i\setminus j} \right) \right. \\ &\quad \left. + (\rho_{ib_i}(\mathcal{J}_i) - 1) g_{i(1)} \left(\frac{f_i - \frac{E_\varepsilon[\varphi(S_i)]}{\varphi(T_i)} p_i}{l_i} | \mathcal{J}_i \right) \right) 1(P_i < \bar{p}) \end{aligned}$$

where $g_{i(1)}(\omega)$ is the density of the first order statistic of match-value and $\rho_{ib_i}(\mathcal{J}_i)$ is the choice probability of the bank chosen by borrower i.

Likelihood for Constrained Loans. When regulation is binding and a loan is approved, loan price equals the price cap and the lowest cost bank makes profits from the loan. In such a scenario, loan price is less than the optimal unconstrained price but higher than the cost of the lowest cost bank. The likelihood of a contract in such situation, signed at price $p_i = \bar{p}_i$ with bank b_i is:

$$\begin{aligned} P(P_i = \bar{p}_i, B_i = b_i|\mathcal{J}_i) &= P\left(P_i^u > \bar{p}, \frac{\varphi(T_i)}{E_\varepsilon[\varphi(S_i)]} (f_i - l_i \omega_{i(1)}) \leq \bar{p}_i, B_i = b_i | \mathcal{J}_i \right) \\ &= \left(G_{i(1)} \left(\frac{f_i - \frac{E_\varepsilon[\varphi(S_i)]}{\varphi(T_i)} \bar{p}_i}{l_i} | \mathcal{J}_{i\setminus j} \right) + (\rho_{ib_i}(\mathcal{J}_i) - 1) G_{i(1)} \left(\frac{f_i - \frac{E_\varepsilon[\varphi(S_i)]}{\varphi(T_i)} \bar{p}_i}{l_i} | \mathcal{J}_i \right) \right) \\ &\quad \times \left(1 - G_{i(1)} \left(\frac{f_i - \frac{E_\varepsilon[\varphi(S_i)]}{\varphi(T_i)} \bar{p}_i}{l_i} | B_i = b_i, \mathcal{J}_i \right) \right) \end{aligned}$$

where $G_{i(1)}(\omega)$ is the distribution of the first order statistic of match-value.

Likelihood for Rejected Loans. When a loan is rejected, the lowest cost bank does not make any profit out of it. Therefore, the cost of such bank is higher than the price cap on

the loan. The likelihood of a contract in such situation is:

$$\begin{aligned} P(P_i = \cdot, B_i = \cdot | \mathcal{J}_i, r_i) &= P\left(\frac{\varphi(T_i)}{E_\varepsilon[\varphi(S_i)]} (f_i - l_i \omega_{i(1)}) > \bar{p}_i | \mathcal{J}_i \right) \\ &= G_{i(1)} \left(\frac{f_i - \frac{E_\varepsilon[\varphi(S_i)]}{\varphi(T_i)} \bar{p}_i}{l_i} | \mathcal{J}_i \right) \end{aligned}$$

Likelihood Function. The likelihood of the data combines the individual likelihoods for these three cases. Let I_i^u, I_i^c and I_i^r indicate that the outcome for application by i was an unconstrained approved loan, a constrained approved loan or a rejected application respectively. The full log-likelihood function for the data is:

$$\begin{aligned} log\mathcal{L}^S &= \sum_{i \in \mathcal{A}} I_i^u \log P(P_i = p_i, P_i < \bar{p}, B_i = b_i | \mathcal{J}_i, x_i) \\ &\quad + I_i^c \log P(P_i = \bar{p}, B_i = b_i | \mathcal{J}_i, x_i) + I_i^r \log P(P_i = \cdot, B_i = \cdot | \mathcal{J}_i, x_i) \end{aligned}$$

We estimate the parameters related to banks' costs by maximizing this log-likelihood function.

A.2.4 Useful Properties of T1EV Distribution

In this Appendix, we summarize useful properties of the T1EV distribution, which we use in the derivation of the likelihood function. Proofs for these results are available in Froeb et al. (1998). First, it can be shown using the properties of extreme value distributions, that the cdf of the highest utility across banks for a borrower is given by:

$$G_{(1)}(\omega; \mathcal{J}_i) = G(\omega; (\omega_{i,max}, \sigma_\omega), \mathcal{J}_i)$$

where:

$$\omega_{i,max} = \sigma_\omega \log \sum_{j \in \mathcal{J}} \exp\left(\frac{\delta_{ij}}{\sigma_\omega} \right)$$

is the location parameter in the distribution.

Moreover, the probability that j is the bank with the lowest cost for i among those in

$\mathcal{J}_i$ is given by the usual logit formula:

$$\rho_{ij} \equiv P(u_{ij} = \max_{k \in \mathcal{J}_i} u_{ik}; \mathcal{J}_i) = \frac{\exp\left(\frac{\delta_{ij}}{\sigma_\omega}\right)}{\sum_{k \in \mathcal{J}} \exp\left(\frac{\delta_{ik}}{\sigma_\omega}\right)}$$

Finally, we can also derive an analytical expression for the distribution of the second order statistic of ω_{ij} in terms of that of the first order statistic. Conditional on j being the choice of borrower i, such distribution would be:

$$G_{(2)}(\omega | u_{ij} = \max_{k \in \mathcal{J}_i} u_{ik}; \mathcal{J}_i) = \frac{1}{\rho_{ij}} G_{(1)}(\omega; \mathcal{J}_{i \setminus j}) + \frac{\rho_{ij} - 1}{\rho_{ij}} G_{(1)}(\omega; \mathcal{J}_i)$$

which by integrating over j to recover the unconditional distribution of the second order statistic yields:

$$\begin{aligned} G_{(2)}(\omega; \mathcal{J}_i) &= \sum_{j \in \mathcal{J}_i} \rho_{ij} G_{(2)}(\omega | u_{ij} = \max_{k \in \mathcal{J}} u_{ik}; \mathcal{J}_i) \\ &= \sum_{j \in \mathcal{J}_i} G_{(1)}(\omega; \mathcal{J}_{i \setminus j}) + G_{(1)}(\omega; \mathcal{J}_i)(1 - |\mathcal{J}_i|) \end{aligned}$$

APPENDIX B

QUALITY REGULATION AND COMPETITION: EVIDENCE FROM PHARMACEUTICAL MARKETS

B.1 Model Simulation

B.1.1 Specification and Details

In order to simulate the model, we need to specify several of its elements. In this section, we introduce our assumptions. Moreover, we derive several outcomes of interest given those assumptions. In all cases, we focus on the symmetric equilibrium we discuss in the main text, which only depends on the innovator drug price, the common generic price and the number of generic firms, namely $\{p_I, p_G, n_G\}$.

Equilibrium Conditions. Formally, the symmetric equilibrium is defined by the conditions:

$$\begin{aligned}
\frac{\partial \pi_I}{\partial p_I}(p_I^*, p_G^*, n_G^*, \overline{\psi}(n_G^*; \underline{\psi})) &= 0, \\
\frac{\partial \pi_g}{\partial p_G}(p_I^*, p_G^*, n_G^*, \overline{\psi}(n_G^*; \underline{\psi})) &= 0 \quad \forall g, \text{and} \\
Ms_G(p_I^*, p_G^*, n_G^*, \overline{\psi}(n_G^*; \underline{\psi}))p_G^* &= C_G(\hat{\psi}(n_G^*; \underline{\psi})) + C_{QC}
\end{aligned}$$

where we use the fact that there is a one-to-one relationship between n_G on the one hand and $\hat{\psi}$ and $\overline{\psi}$ on the other, conditional on the minimum quality $\underline{\psi}$. The first two equations are the conditions for a Bertrand-Nash equilibrium for the innovator and generic producers respectively, whereas the third equation is the zero-profit entry condition for the marginal generic entrant.[1]

1. Note that we omit the condition for innovator participation. Allowing innovator exit is straightforward, though at the expense of added complexity in the equations describing the equilibrium and the model simulations. Since it is trivial to study when exit happens (lower innovator variable profits increases the likelihood of exit), and the qualitative effect of innovator exit is intuitive (positive effect on generic profits and entry), we exclude this aspect from the exposition.

Demand Side. First, we assume that ε_{iI} and ε_{ig} are drawn i.i.d. from an extreme value type I distribution. Second, we assume that τ_i is drawn i.i.d. from F_τ. In particular, we assume that $\tau_i \sim U[\underline{\tau}, \overline{\tau}]$. Furthermore, we normalize the quality of the innovator drug (ψ_I) to 1. These assumptions imply that market shares take the mixed logit form:

$$\begin{aligned} s_I &= \int s_I(\tau)\, \mathrm{d}F_\tau(\tau) = \frac{e^{\tau - p_I}}{1 + e^{\tau - p_I} + \sum_{k \in \mathcal{G}} e^{\tau \overline{\psi} - p_g}}\, \mathrm{d}F_\tau(\tau) \\ s_g &= \int s_g(\tau)\, \mathrm{d}F_\tau(\tau) = \int \frac{e^{\tau \overline{\psi} - p_g}}{1 + e^{\tau - p_I} + \sum_{k \in \mathcal{G}} e^{\tau \overline{\psi} - p_k}}\, \mathrm{d}F_\tau(\tau) \quad \forall g \end{aligned}$$

where $s_I(\tau)$ and $s_g(\tau)$ are choice probabilities conditional on τ, and $\mathcal{G}$ is the set of active generic producers. In particular, for a symmetric equilibrium with generic price as p_G and n_G active generic drugs, the market share of generic drugs is given by:

$$s_G = s_g\big|_{p_g = p_G \forall k \in \mathcal{G}} = \int \frac{e^{\tau \overline{\psi} - p_G}}{1 + e^{\tau - p_I} + n_G e^{\tau \overline{\psi} - p_G}}\, \mathrm{d}F_\tau(\tau)$$

Finally, Given the logit structure of the demand system, consumer surplus for a given set of parameters can be computed as:

$$CS = M \int \left(1 + e^{\tau - p_I} + n_G e^{\tau \overline{\psi} - p_G}\right) \mathrm{d}F_\tau(\tau)$$

where M measures market size.

Supply Side. We let the distribution of quality among potential generic producers be given by $\psi_g \sim U[0, 1]$, which implies that the quality of the n^{th} potential generic producer is $\frac{n}{N_G}$. Under this assumption, the marginal and average quality in the market (conditional on a minimum quality $\underline{\psi}$) are:

$$\begin{aligned} \hat{\psi}(n_G; \underline{\psi}) &= \frac{n_G}{N_G} + \underline{\psi} \\ \overline{\psi}(n_G; \underline{\psi}) &= E[\psi | \underline{\psi} < \psi < \hat{\psi}] = \frac{1}{2}\frac{n_G}{N_G} + \underline{\psi} \end{aligned}$$

Moreover, we assume that fixed manufacturing costs are given by $C_I = \kappa$ and $C_G(\psi) = \kappa\psi$ for the innovator and generic drugs respectively, where $\kappa \geq 0$ is a parameter governing the sensitivity of fixed costs to drug quality.

In the symmetric equilibrium we discuss, the profit the innovator drug is:

$$\pi_I = p_I s_I - C_I$$

while the profit of all active generic drugs is given by:

$$\begin{aligned} \int \pi_G(n)\,\mathrm{d}n &= \int_0^{n_G} \left[p_G s_G - C_G(\hat{\psi}(n)) - C_{QC}\right] \mathrm{d}n \\ &= n_G(p_G s_G - C_{QC}) - \int_0^{n_G} C_G(\hat{\psi}(n))\,\mathrm{d}n \end{aligned}$$

where total manufacturing fixed costs for generics are $\int_0^{n_G} C_G(\hat{\psi}(n))\,\mathrm{d}n = n_G\kappa\left(\frac{1}{2}\frac{n_G}{N_G} + \underline{\psi}\right) = n_G C_G(\overline{\psi})$ under the assumed functional form and distributions.

Total Welfare. Given this structure and assumptions, total welfare in the market is given by:

$$W = CS + \pi_I + n_G(p_G s_G - C_G(\overline{\psi}) - C_{QC})$$

such that it combines consumer surplus, profits for active producers and the cost of quality certification for generic drug producers.

Parametrization for Simulation The common parameters used when solving the model to produce the results in Figure 2.1 are listed below:

Parameter	Value
$(\underline{\tau}, \overline{\tau})$	$(0, 9)$
M	3
C_{QC}	0.5
κ	0.4
N_G	20

Finally, the minimum quality standard ($\underline{\psi}$) is set to 0.2 in scenario **a** of Figure 2.1, and to 0.6 for scenarios **b** and **c**. In **c**, the cost of quality certification is set to 0.5, while in **a** and **b** it is set to 0.

B.1.2 Additional Model Analysis

Relationship between Fixed Costs and Market Size. Consider the equation describing profits of the marginal generic entrant when compliance costs apply ($C_{QC} > 0$),

$$Ms_G p_G - C_G(\hat{\psi}) - C_{QC} = 0.$$

Let us consider how a change in C_{QC} will affect the quality of the marginal generic entrant, $\hat{\psi}$, keeping in mind that the number of active generics can be described as a function of $\hat{\psi}$ (conditional on $\underline{\psi}$). For this exercise, we will keep prices fixed, noting that the change in equilibrium prices will be determined by the change in $\hat{\psi}$. From the equation above, we get

$$\frac{\partial \hat{\psi}}{\partial C_{QC}} = \left[Mp_G \left(\frac{\partial s_G}{\partial n_G} \frac{\partial n_G}{\partial \hat{\psi}} + \frac{\partial s_G}{\partial \overline{\psi}} \frac{\partial \overline{\psi}}{\partial \hat{\psi}} \right) - C'_G(\hat{\psi}) \right]^{-1},$$

such that a higher M leads to a lower response to compliance costs on the quality of the marginal entrant (and thus on total entry) for any given minimum quality standard. It should be pointed out that this is conditional on the size of all other terms in the expression above.

Since one would generally expect markets of larger size to have a different equilibrium, a direct comparison is difficult. However, we consider the case of two markets with all parameters equal, except M and the addition of a fixed cost term FC, such that the equilibrium is equal,

$$\begin{aligned} 0 &= M_0 p^*_G s^*_G - C_G(\hat{\psi}^*) - C_{QC} \\ 0 &= M_1 p^*_G s^*_G - C_G(\hat{\psi}^*) - C_{QC} - FC, \end{aligned}$$

where $M_1 > M_0$, implying $FC = (M_1 - M_0) p^*_G s^*_G$. In this case, it is easy to see that the

response to changes in the compliance costs will be smaller in the larger market. This situation is illustrated in Figure B.3, where the left panels show the effects for a small market ($M_0 = 2$), while the right panels show the effects for a large market ($M_1 = 6$). Welfare and consumer surplus has been normalized by the market size (a per capita measure). Note that, for each outcome, point **a** coincides between the small and large market, except for variable profits which are less sensible to compare between these scenarios. Horizontal lines are added to indicate the level of post-equilibrium outcomes with costly compliance (points **c**) for the small market.

Quality Regulation with Desirable Competitive Effects. There are several factors in our model that can improve the welfare effect from quality regulation. The most obvious and direct one is lower compliance costs, which yields less exit/more entry on the high-quality margin, thereby both increasing the average quality and strengthening price competition compared to a scenario with higher compliance costs. Another is high overall willingness to pay for quality, which tends to both increase the viability of high-quality entry and increase the impact on (consumer) welfare from higher average quality in the market. This latter situation is illustrated in Figure B.4, where we have set $(\underline{\tau}, \overline{\tau}) = (5, 9)$, which makes consumer surplus increase quicker along the minimum quality (primarily driven by higher average quality).

Furthermore, if $C_G(\psi)$ is relatively flat, this will increase the effect of stronger quality regulation on marginal quality (and thus average quality). Particularly, in markets where there is entry on the high-quality margin, this entry will be larger in markets with a lower $C'_G(\psi)$.

The Role of a Loyal Segment. We consider the effect of allowing brand loyalty above quality differences. In our model, we can add brand loyalty as an extra term ν in u_{iI} for a fraction ϕ of consumers, capturing additional utility from purchasing the innovator drug.[2] The existence of a brand-loyal segment can help rationalize certain price strategies by the innovator, such as increasing the price when competition from generics increases (i.e., the

2. For simplicity, we let ν be a constant among the brand-loyal consumers.

"Generic Paradox"). This situation is illustrated in Figure B.5.

The presence of a loyal segment generally dampens price-responses of the innovator firm, and might make the innovator's price response to stronger quality regulation non-monotonic, as the innovator may decide to set prices targeting either mainly the loyal segment or a larger share of the market.

B.2 Event Study Evidence of Policy Effects

The empirical strategy we propose in Section 2.6.1 exploits the staggered roll-out of the regulation across molecules as a useful source of identifying variation, which we complement with within market variation in drug license renewal dates. As a complement to estimates of policy effects using that strategy, we implement an event study analysis. The event study serves two purposes: (i) assessing the assumption of parallel trends across groups of molecules treated by the policy at different moments; and (ii) providing transparent visual evidence of the effects of bioequivalence on relevant market outcomes.

We implement an event study by replacing the treatment variable T_{mt} in equation (2.3) by a set of event-time dummies that capture the policy effect for each month around the policy event. Concretely, we estimate the following variant of equation (2.3):

$$y_{mt} = \sum_{\tau} \beta_\tau D_{mt,\tau} + \theta_m + \delta_t + \varepsilon_{mt}$$

where we have replaced T_{mt} in equation (2.3) for indicators $D_{mt,\tau}$ of the time period where the policy event occurred exactly τ periods before. Formally, if the policy for market m occurred in period t_{0m}, then:

$$D_{mt,\tau} \equiv \mathbb{1}(t - t_{0m} = \tau).$$

In practice, we consider the first policy deadline as the event that defines t_{0m}. Although decrees were extended, we cannot rule out that extensions were unexpected. This choice allows us to remain agnostic about potential reactions to the announcement of the first

decree. We also place the following end-point restrictions:[3]

$$\beta_\tau = \begin{cases} \bar{\beta} & \text{if } \tau > 24 \\ \underline{\beta} & \text{if } \tau < -24 \end{cases}$$

Finally, we normalize the coefficient $\beta_{\tau=-1} = 0$. Therefore, all effects are interpreted as relative to the month before the first deadline. Finally, we include the same sets of fixed effects as in equation (2.3).

Figure B.6 plots estimates with their corresponding 95% cluster-robust confidence intervals. The first row displays results for the number of drugs across drug types. Our estimates show a slight decrease in the number of drugs overall, which seems to be driven by non-bioequivalent generics. As expected from the policy, our estimates show a large increase in the number of bioequivalent generics. The second row displays results for drug prices. We find no clear price effects overall, though the price of innovator drugs and unbranded generics show signs of increase in the second year after the policy event, while there might be a small decrease in the price of branded generics. Finally, the third row displays the estimated effects on market shares. Our results show substitution from non-bioequivalent to bioequivalent branded generics, while unbranded generics possibly decrease and innovator drugs possibly increase their market shares. We provide a detailed discussion of effects on all these and other margins in our main analysis in Section 2.6.

Overall, trends in outcomes before the first deadline appear to be well behaved: most of the estimated coefficients are close to zero. This fact is reassuring for using the differential timing of bioequivalence requirements across markets as identifying variation in estimating the effects of quality regulation on market outcomes in our setting.

3. Note that for some markets, our data covers as much as seven years of data after the policy event, such that this window will not show effects for all the period after the policy that we observe. Results in Section 2.6 do consider the full period after the policy implementation that we observe in our data.

B.3 Description of Consumer Survey

B.3.1 Methodology and Results

In order to inform potential explanation for the results from our main analysis, we collect additional survey data in which we interview consumers and gather information on perceived quality, perceived price differences, relationship between physician prescription behavior and consumer choices and some additional characterization variables. The questionnaire is displayed in Section B.3.2 below.

A surveying team composed by 6 members conducted surveys in 4 counties in the city of Santiago, namely Ñuñoa, Providencia, Puente Alto and Santiago. Within such counties, surveyors recruited consumers for the study outside pharmacies, where consumers were purchasing drugs. This recruiting strategy aimed at constructing a sample of consumers familiar with the pharmaceutical market. Recruited participants were asked to participate in a survey with a duration of between 5 and 10 minutes, and were offered no compensation for it.

In order to collect data on perceived quality and price differences, we focus on a particular market, Atorvastatin, a molecule commonly prescribed as a treatment to cholesterol. Within that market, we focus on 4 drugs that are relevant products in this market. In particular, we work with (i) a popular innovator drug called Lipitor, which is produced by Pfizer, (ii) a bioequivalent branded generic called Lipoten, produced by Pharmavita, (iii) a bioequivalent unbranded generic called simply Atorvastatina, produced by Mintlab, and (iv) and a non-bioequivalent unbranded generic also called Atorvastatina and produced by Mintlab. For reference, the prices of these drugs in the market are around $50,000 CLP, $10,000 CLP, $2,500 CLP and $2,500 CLP respectively ($77.5, $15.5 and $7.8 U.S. dollars respectively). Perceived quality and price differences are elicited using a paper sheet that showed the 4 drugs, which is displayed in Figure B.8.

The final sample includes $N = 401$ consumers. Table B.7 provides summary statistics for the main variables in the survey. Among consumers in the sample, 62% report having a household member with a chronic disease, and 36% report purchasing Atorvastatin for a household member. In terms of purchase behavior, 41% often purchases innovator drugs,

21% often purchases branded generics, and the remainder 38% often purchases unbranded generics. The main results of the survey and their relationship to the results in our main analysis are discussed in Section 2.7. We code observations in which a consumer answered "I don't know" or "I don't recall" as missing. Finally, the questions regarding physicians' prescription behavior have less observations because they were added to the survey with a lag and are therefore not available for a around a fourth of the sample.

B.3.2 Questionnaire

We are conducting a survey about the quality perception of drugs sold in pharmacies. We will ask you a few questions regarding the quality and prices of drugs. In all examples, we will focus in a drug called Atorvastatin, which is commonly used to control cholesterol levels. While we understand that it may be that no one in your household takes Atorvastatin, we ask that you consider it as an example and think as if you had to acquire it for a family member.

1. [Show pictures of four drugs] Consider a scale of 1 to 7, where 1 is a drug of the minimum quality and that does not have the desired therapeutic effects and 7 is a drug of the highest quality that has exactly the expected therapeutic effects. What level of quality do you think the following drug has?

    - Innovator
    - Bioequivalent unbranded generic
    - Unbranded generic
    - Bioequivalent branded generic

2. [Show pictures of 4 drugs] If the price of the innovator drug is $50,000. What price do you think each of these drugs has?

    - Bioequivalent unbranded generic
    - Unbranded generic
    - Bioequivalent branded generic

3. [Show pictures of innovator and bioequivalent unbranded generic] If you were buying a box of Atorvastatin and were offered these two drugs. The innovator is priced at $50,000 in pharmacies. What do you think is the price of this generic?

4. [Show pictures of innovator and unbranded generic] If you were buying a box of Atorvastatin and were offered these two drugs. The innovator is priced at $50,000 in pharmacies. What do you think is the price of this generic?

5. [Show pictures of innovator and bioequivalent branded generic] If you were buying a box of Atorvastatin and were offered these two drugs. The innovator is priced at $50,000 in pharmacies. What do you think is the price of this generic?

6. [Show bioequivalence label] Have you ever seen this label on a drug before this survey?

    - Yes
    - No

7. [Do not read, use the following scale] Do you know what it means for a generic drug to be bioequivalent?

    - Very good response: Bioequivalence implies that two drugs have exactly the same therapeutic effects as the original
    - Good response: The generic is the same as the innovator
    - Regular response: A vague answer in terms of the quality of both drugs
    - Bad response: They are part of the same group of medications (e.g. both are Atorvastatin)
    - He has no idea: He does not know, he has no idea, he has not heard

8. When doctors deliver prescriptions, do they generally prescribe drugs by specifying a particular brand or without specifying a brand?

    - Prescribe drug without a specific brand

- Prescribe drug with a specific brand
- Does not know

9. When buying a prescription drug at a pharmacy, how much does your doctor, the pharmacist who serves you, and yourself weight in deciding which version of the medication to buy? In particular, on a scale of 1 to 5, where 1 is no power and 5 is a lot of power, how much power they have:

    - Doctor
    - Pharmacist
    - Customer

10. What type of drug did you buy the last time you needed one?

    - Innovator
    - Bioequivalent unbranded generic
    - Unbranded generic
    - Bioequivalent branded generic
    - Do not remember
    - Never purchased

11. Do you or anyone in your home take any drug for a chronic illness?

    - Yes
    - No

12. Do you or anyone in your household take any drug to control cholesterol?

    - Yes
    - No

13. What type of drug do you choose when you buy this medication for cholesterol control?

- Innovator
- Bioequivalent unbranded generic
- Unbranded generic
- Bioequivalent branded generic
- Do not remember
- Never purchased

APPENDIX C
VERTICAL INTEGRATION BETWEEN HOSPITALS AND INSURERS

C.1 Data Appendix

C.1.1 Construction of Admissions Dataset

Denote plans data by $\mathcal{P}$, claims data by C. The estimating dataset is constructed following steps given by:

1. Keep all plans in $\mathcal{P}$ in 2013 and 2014.
2. Recover preferential hospitals for each plan from C, using years 2008 to 2016. We keep the three most relevant preferential hospitals of each plan.
3. Merge preferential hospitals from C by plan name to plans in $\mathcal{P}$. Only 6% of the plans in $\mathcal{P}$ are not in C, equivalent to 0.1% of the claims in C. We drop them.
4. Construct plan identifiers by collecting plans with the same insurer, inpatient and outpatient coinsurance rate, whether it has a coverage cap or not, in the same base price decile, and with the same preferential hospitals. From now on, this is the definition of plans.
5. Merge plans identifiers in $\mathcal{P}$ to each claim in C for 2013 to 2016.
6. Construct events as a collection of claims.
7. Define main hospital as one of 12 main hospitals (Alemana, Avansalud, Bicentenario, Dávila, Indisa, Las Condes, Santa María, Tabancura, UC, UC San Carlos, Vespucio, UChile). These hospital account for 76% of events in C. Collect all other hospitals in another category, "other".
8. Assign each event to a main hospital.
9. Collapse claims in each event to a single, event-level, observation. We construct price paid and full price for each event.

10. Recover effective coinsurance rate by plan, for preferential and non-preferential hospitals.

11. Merge consumer covariates. Drop if no consumer information.

12. Select estimating data. Keep only plans with more that 100 policyholders and claims for more than 10 diagnosis.

13. Define markets as the combination of year, plan and diagnosis. Drop markets with claims from 3 main hospitals or less in C.

C.1.2 Estimation of Negotiated Prices and Resource Intensity Weights

Equation (3.13) in the main text shows the generic decomposition of observed price into a negotiated price and resource intensity weights. The assumptions made in that section imply that our estimating equation takes the form:

$$\rho_{ihmdt} = p_{hmt}\omega_{id}$$

which separates observed prices into a negotiated component and a common utilization based component. The econometric challenge is that ω_{id} varies by potentially unobserved attributes of consumer i, and as not all consumer get ill of all conditions, we can not recover these values directly from the data. Our approach to solving this issue leverages our data on the public system prices and the fact that we observe the itemized claims of each admissions. This allows us to construct the exact price that the admissions would have cost in the public hospital system, which we denote p_{id}^{pub}. The public price is unrelated to the negotiation between h and m, as neither are affected by the public system's prices. However, this price is a clear metric of consumer utilization and the cost of providing treatment for the consumer. Using this we proceed by estimating the equation:

$$\log(\rho_{ihmdt}) = \log(p_{hmt})\iota_{hmt} + \alpha_h p_{id}^{pub} + \epsilon_{ihmdt}$$

where ι_{hmt} is an indicator for hospital h, insurer m and time t. This regressions identifies the negotiated prices as the coefficient on the indicators. Importantly, we allow for the connection between the public system price to scale differently for different hospitals, as captured by α_h. We then reduce the dimensionality of the utilization component to consumer types κ defined by gender and age (binned in 25-year age brackets) by:

$$\omega_{\kappa d} = \frac{1}{|I_{\kappa,d}|} \sum_{i \in I_{\kappa,d}} \hat{\alpha}_h p_{id}^{pub} \quad \forall \kappa$$

where $I_{\kappa,d}$ is the set of observations for the consumer group κ and diagnosis d.

Table C.2 provides details regarding the distribution of the prediction error for four different methods of estimation. The first column shows the error distribution from the first stage. This stage is used to recover the negotiated prices. The second columns shows the loss of prediction that we incur by reducing the dimensionality of ω to tractable levels. The following two columns correspond to estimating the resource intensity weights using fixed effects. The first shows the error from using only a diagnosis fixed effect, while the second uses a diagnosis and consumer attribute interaction fixed effect. By construction, the last column provides a better fit, however it drastically trades off precision in the estimated parameters, crucially so for the negotiated prices which play a central role in our structural estimation. Finally, table C.3 shows the estimated resource intensity weights [1] and figure C.3 shows the prediction fit averaged over insurer-hospital-year.

C.1.3 Construction of Insurance Plans Choice Sets

The construction of the plan demand estimation panel builds upon the hospital demand panel and the associated estimates. The main issue this code has to tackle is the overwhelming computation cost of calculating network expected utilities for each consumer and their dependents for each plan in each year, i.e computing equation (3.15). The algorithm proceeds as follows:

1. Load the hospital demand panel and filter the columns relevant for either equation

1. We do not include the disaggregated estimated negotiated price for confidentiality reasons.

(3.15) or (3.16). Split income into deciles to create a large yet finite number of consumer types. Consumer types will determine groups that share the same expected utility of networks as they agree over all hospital and plan utility dimensions.

2. Load the payer and dependent panels for the year 2013-2017. Merge and reduce to the consumers that belong to plans for which we have sufficient information to compute conditional hospital demands. This is the same filter applied in the hospital demand panel formation.

3. Define plan demand markets as combinations of year, gender, age group and whether the consumer has dependents. Split plans into independent plans over markets and compute their market share. For each insurer-market keep the 5 plans with the largest market shares. Expand the consumer and dependent data such that each individual now has all options available in his market.

4. Operating in batches of consumers, add for each plan all available hospitals. Expand to include all diagnoses. Using the estimated medical risk, resource intensity weights and negotiated price, compute equation (3.15) for all possible combinations of consumer-plan-hospital-diagnosis. Collapse over payer-plans (i.e, sum dependents expected utility if necessary) and update consumers that share the same utility type as the ones just computed to reduce computation time.

5. Finally, for each market restrict the choice set of consumer to only include their current plan and plans currently being commercialized. This removes less than 2% of alternatives and leaves no consumer with less than 5 alternatives.

C.2 Details about Health Insurance in Chile

C.2.1 How do Public and Private Sectors Interact?

The public and private health systems seem to operate in practice in a remarkably isolated fashion. For instance, most of the consumers that purchase insurance in the private sector are also provided health care services mostly by private sector hospitals. A substantial 97%

of all payments by private insurers are collected by private hospitals, while only 3% are collected by public hospitals (Galetovic and Sanhueza, 2013). Research on sorting across sectors points towards the remarkable differences in premium structures across sectors as the most relevant determinant of consumers' choice between public and private insurance (Pardo and Schott, 2012, 2013).

In terms of the evolution of their market shares, Figure C.8 shows that through the period of study there has been a slight increase in the market share of public insurance, from 66% to 76%, while the market share of private insurers has remained almost unchanged at around 18%. The increase in the public insurance market share originates mostly from a reduction in the share of consumers with either no insurance or other forms of insurance. An interesting margin of study in this market is that of switching across the private and public sector. Data availability only allows for looking at switching out of the private sector. Duarte (2011) provides preliminary evidence showing that (i) the amount of switching across sectors is low, and that (ii) the public sector operates as a safety net, as one of the major determinants of a consumer's decision to switch is job loss.

There are some aspects that are worth studying in further detail in term of the relationship between these two sectors. First, there are some remarkable differences and interactions in terms of regulation. Second, additional policies and regulations have been enacted during the period of study of this paper.

Constraints on plan design. Private insurers are mandated to offer coverage caps that are at least as large as those offered by the public insurer, FONASA. Therefore, private insurers' coverage caps are updated annually following the the public insurer updates, which are implemented every April. Presumably, private insurers optimally adjust premiums as well as a response to this change in coverage caps induced by the public insurer.

Differences in risk pricing. A notable feature that distinguishes the public and private system in this market is the differential ability of the latter to implement risk pricing or risk selection. As mentioned above, FONASA's only distinction across consumers is based on income and, to a second order, family size. However, they do not offer different plans across

other dimensions. On the other side, while regulation limits the extent of risk pricing by private insurers, they can still price differently across age and gender. Moreover, private insurers are able to reject applications from consumers based on pre-existing conditions. Finally, the large number of plans available in the market suggest that such variety could be a vehicle through which private insurers implement some form of risk pricing. The result of these differences is cream skimming: the concentration of risky consumers is lower in the private than in the public sector.

***Ley Larga* de Isapres.** Through law 20,015, enacted in May, 2005, the government introduced a number of regulations to the private insurance sector. The focus of these was to reduce the extent for risk pricing by private insurers. Two relevant constraints on pricing that were introduced by this law were already described above: (i) the number of risk-rating functions (i.e. f in section 3.2.1) was limited to 2 per insurer, and (ii) the extent to which premiums could be adjusted through time was limited to 1.3 of the average premium change, to reduce the extent for risk reclassification. Additionally, this law arguably increased the cost of vertical integration. This, as it explicitly established that insurers are not allowed to participate in the provision of health care services. This is the reason why vertical integration in this market is organized through common ownership of insurers and hospitals by *holdings*, rather than through direct ownership of hospitals by insurers.

AUGE-GES plan. Through law 19,966, enacted in September, 2004, the government made mandatory the coverage of a list of health conditions dictated by the Ministry of Health. This regulation implied that since June, 2005, both public and private insurers are required to provide adequate treatment and insurance for consumers under conditions included in the list. The four elements considered by the law were (i) *access* to adequate treatment, (ii) certification of the *quality* of treatment by hospitals, (iii) *financial protection* of consumers through the imposition of thresholds below which there is a 20% coinsurance rate and beyond which such rate is set to 0%, and (iv) *opportunity*, by imposing maximum wait times for consumers to be treated by the system. The list started by including 25 conditions since July, 2005, and then was extended to 40 and 56 by July, 2006 and July, 2007 respectively.

C.2.2 *Structure of Private Insurance Plans*

Private insurers are allowed to engage in risk selection by rejecting applications based on pre-existing medical conditions. In contrast, the public insurer cannot deny coverage, which has led to a relative concentration of risky consumers in the public sector. However, in the private sector there is guaranteed renewability, by which contracts are automatically renewed for current enrollees under the original agreement terms, regardless of changes in health status.

Contracts offered in the private sector are regulated and are composed of the following elements. First, they have a monthly premium P which is a combination of a base price P_B and a risk-rating factor f, so that $P = P_B \times f$, where f is a gender-specific step function of age. Second, insurers choose a base price for each plan, which can be adjusted yearly.[2] Third, each plan has separate coinsurance rates for inpatient and outpatient care. Coinsurance rates are constant across claims for the same service. Fourth, plans may specify a coverage cap for each service, which is equivalent to having the coinsurance rate becoming one beyond that expenditure level. This maximum payment is constant across claims for the same service.[3]

Payments from insurers to hospitals operate in a fee-for-service system. Copayments that policyholders pay for a given service are a function of plan attributes as follows:

$$\text{copayment} = \text{price} - \min\{\text{price} \times (1 - \text{coinsurance}), \text{cap}\}$$

such that the marginal price increases after the coverage cap is reached.

Regarding hospital networks, plans offer either unrestricted open networks or tiered

2. Risk-based pricing is allowed in the private market but regulated since 2005. First, base-prices are chosen at the plan and not the individual level. Second, each insurer may use only two f functions. Third, the increase in a plan price cannot be higher than 1.3 times the average increase in plan base prices across all plans offered by an insurer. However, plan proliferation is evident from the data, as around 40% of insurance plans in the market serve only one consumer, and the average number of consumers per plan is 28 (Atal, 2015). This proliferation suggests that insurers possibly implement some form risk pricing through that mechanism.

3. Although private plans may impose coverage caps for some services, in our application we use the ex-post empirical coverage for each claim, and thus, our results are robust to the face value of these caps.

networks.[4] Unrestricted network plans provide the same coverage for all hospitals. Tiered networks offer differentiated coverage across different sets of private hospitals, similar to PPO plans in the U.S.. Hospitals cannot deny health care to patients, and thus all consumers have access to all hospitals, although they may have zero coverage from their plan. Overall, private insurers provide better coverage in private hospitals, which are generally perceived as being of higher quality than public hospitals in terms of waiting time, medical resources, and medical outcomes.

C.3 Model Appendix

C.3.1 GMM Estimation Algorithm

The GMM estimation algorithm builds upon equation (3.17). The general procedure of the estimation was described in section 3.4.4. In this Appendix, we provide additional details regarding our implementation.

We start by describing the structure of the estimation algorithm, which comprises the following iterative steps:

$t = 0$ - Initialize variables and load data

$t \geq 1$ - Recover a guess of bargaining weights from a non-linear solver

- t_1 - Compute the GMM objective function
  - $t_1^{'}$ - Evaluate $\boldsymbol{c}' = C(\boldsymbol{\phi}^*(\boldsymbol{c}), \lambda)$
    - $t_1^{''}$ - Evaluate $\boldsymbol{\phi}' = \Phi^*(\boldsymbol{\phi}, \boldsymbol{p}, \boldsymbol{c})$
    - $t_2^{''}$ - If $\|\boldsymbol{\phi}' - \boldsymbol{\phi}\|_2 \leq \epsilon_\phi$ break loop, otherwise set $\boldsymbol{\phi} = \boldsymbol{\phi}'$ and return to $t_1^{''}$
  - $t_2^{'}$ - If $\|\boldsymbol{c}' - \boldsymbol{c}\|_2 \leq \epsilon_c$ break loop, otherwise set $\boldsymbol{c} = \boldsymbol{c}'$ and return to $t_1^{'}$.
- t_2 - Assess if the change in the GMM objective function is below tolerance. If so break the loop, otherwise update solver and return to t_1 with $t = t + 1$.

4. Few plans offer restricted networks, similar to HMO plans in the U.S. They are rarely observed in the data and not offered publicly. We do not consider them in our analysis.

There are two important implementation details that are worth mentioning. First, this code needs to recurrently access multiple data sets to compute the different steps. Furthermore, often datasets need to be accessed in different orders or specific values need to be found. For example, the bargaining first order conditions requires computing the derivative of premiums with respect to negotiated prices, this implies iterating over premiums and looking up whether they belong to integrated insurer and if so to which hospital system. As our code builds upon nested fixed points which need to be evaluated often tens of thousand of times, these operations need to be extremely fast. To tackle this problem we rely heavily on pointer-based operations and hash-table lookups. For this purpose, we code our GMM in C and use highly optimized linear algebra routines whenever available.

Second, our implementation of the equilibrium premium is substantially more detailed than the illustrative FOC of equation (3.12). To present the exact premium formula we need to further extend our notation.

Let I denote the set of markets and define $\mathcal{J}_m^i$ the set of plans insurer m offers in some market $i \in I$.[5] Also, denote $\mathcal{J}^i$ the complete set of plans offered in market i, i.e $\mathcal{J}^i = \cup_{m \in M} \mathcal{J}_m^i$. Furthermore, $\sigma_{j|k}^M(\boldsymbol{\phi}, \boldsymbol{p})$ denotes the share of plan j if plan k were removed from the market, keeping all else constant. As we assume that insurers optimize at a mean consumer level in each market, we can identify each plan with its relevant consumer. Denote α_j^M the mean premium sensitivity of consumers in the market in which plan j is offered. Furthermore, we denote $\delta_k^M = \alpha_f^M \phi_{fk} + \beta_f \sum_{i \in f} EU_{ik}^H + \delta_{m(k)\kappa(f)}^M$ for the mean consumer f of plan k.

Using this, it can be shown that the equilibrium premium of a plan j being offered in market i by insurer m can be written as:

$$\phi_j^* = \pi_{m|j}^M + \mathbb{1}\{m \in \mathcal{V}\}\tilde{\pi}_{s(m)|j,i}^H + c_j^M - \frac{1}{\alpha_j^M}(1 + W(\tilde{\lambda}_j)) \tag{C.1}$$

where $\mathbb{1}\{m \in \mathcal{V}\}$ indicates if insurer m is vertically integrated with some system $s(m)$ and c_j^M is the expected cost of plan j. $\pi_{m|j}^M$ corresponds to the profit of insurer m if it were to

5. In this appendix, we suppress the time subscript t for simplicity.

remove plan j from the market:

$$\pi^M_{m|j} = \sum_{r \in \mathcal{J}_m} \sigma^M_{r|j}(\phi_r - c^M_r)$$

Moreover, $W(\cdot)$ in equation (C.1) is the Lambert W function and $\tilde{\lambda}_j$ is:

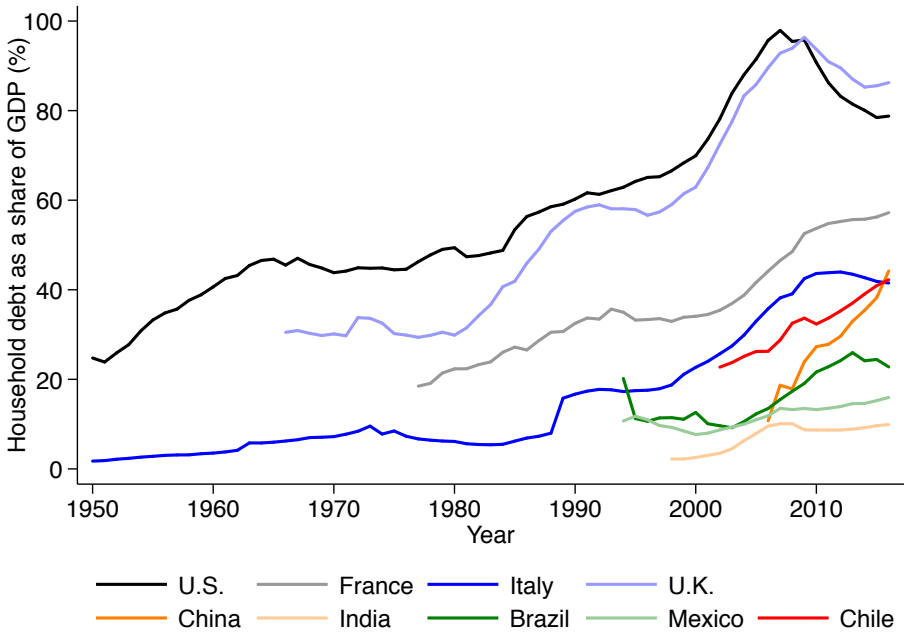
$$\bar{\lambda}_j = \frac{\exp(\alpha^M_j \pi^M_{m|j} + \alpha^M_j c_j - 1 + \delta^M_j - \alpha^M_j \phi_j + \mathbb{1}\{m \in \mathcal{V}\}\alpha^M_j \tilde{\pi}^H_{s(m)|j,i})}{\sum_{k \in \mathcal{J}^i \setminus \{j\}} \exp(\delta^M_k)}$$

Finally, the vertical integration effect is given by:

$$\tilde{\pi}^H_{s(m)|j,i} = \sum_{l \in M} \sum_{h \in H_s} \left(\sum_{k \in \mathcal{J}^i_l} \sigma^M_{k|j} \sum_{d \in D} \gamma_{di} \omega_{di} \sigma^H(ikh|d) \right) (p_{lh} - c^H_{lh}) - \sum_{h \in H_s} \sum_{d \in D} \gamma_{di} \omega_{di} \sigma^H_{ijh|d} (p_{hj} - c^H_{hj})$$

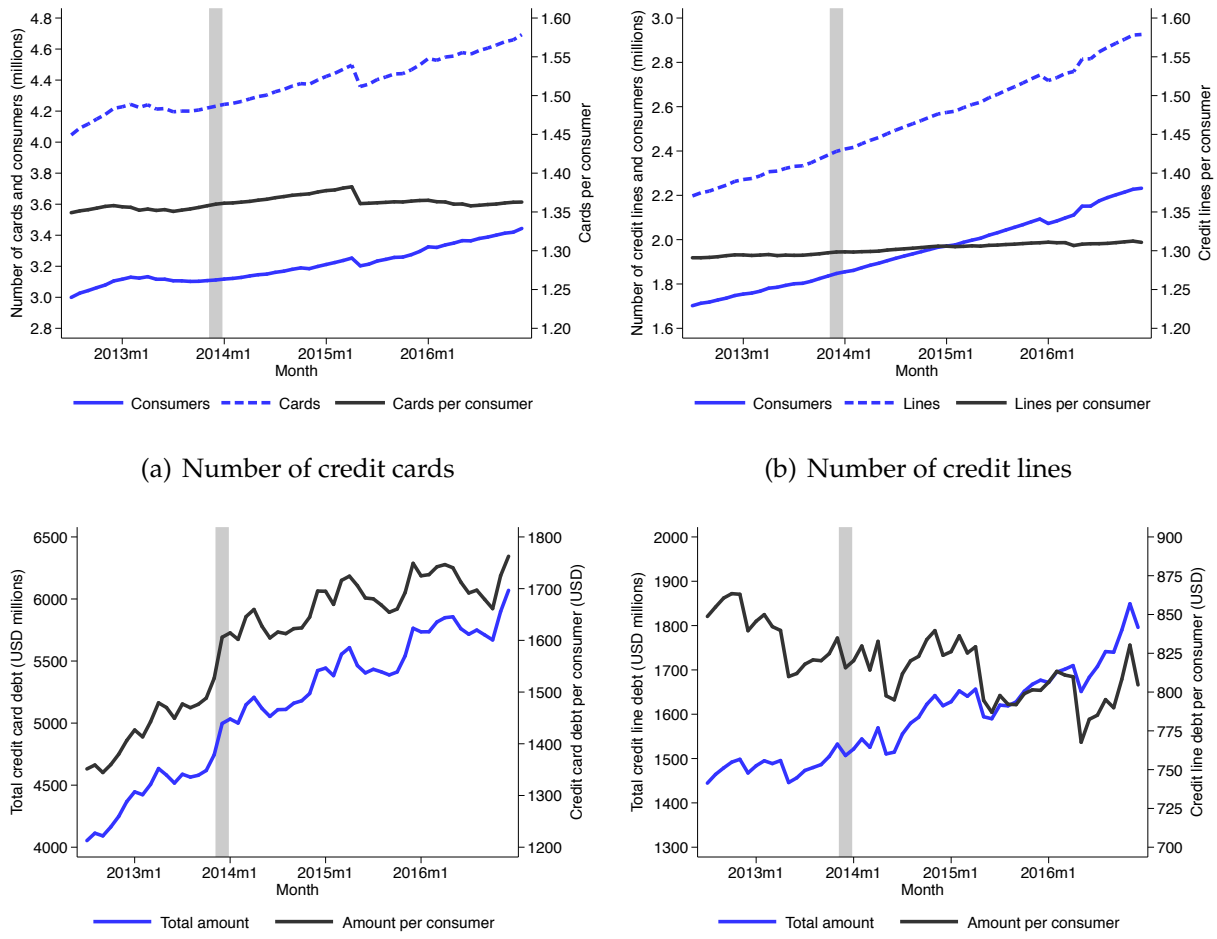
The benefit of the reformulation presented in equation (C.1) is that ϕ_j only appears on the left hand side. This helps the convergence of the fixed point equation and allows easier computation of the derivatives of premiums with respect to other premiums and prices.

Figure A.1: Evolution of household debt as a share of GDP across time and countries



Notes: This figure displays the evolution of household debt as a share of GDP for a sample of countries. Authors' calculation based on based on data from the Global Debt Database by the International Monetary Fund (Mbaye et al., 2018). The length of each series is determined by the availability of data for each country. There is substantial cross sectional variation. For 2016, household debt as a share of GDP ranges from 0.6% for Afghanistan to 126.3% for Switzerland, with an average of 39.7%.

Figure A.2: Evolution of credit card and credit line debt



(a) Number of credit cards

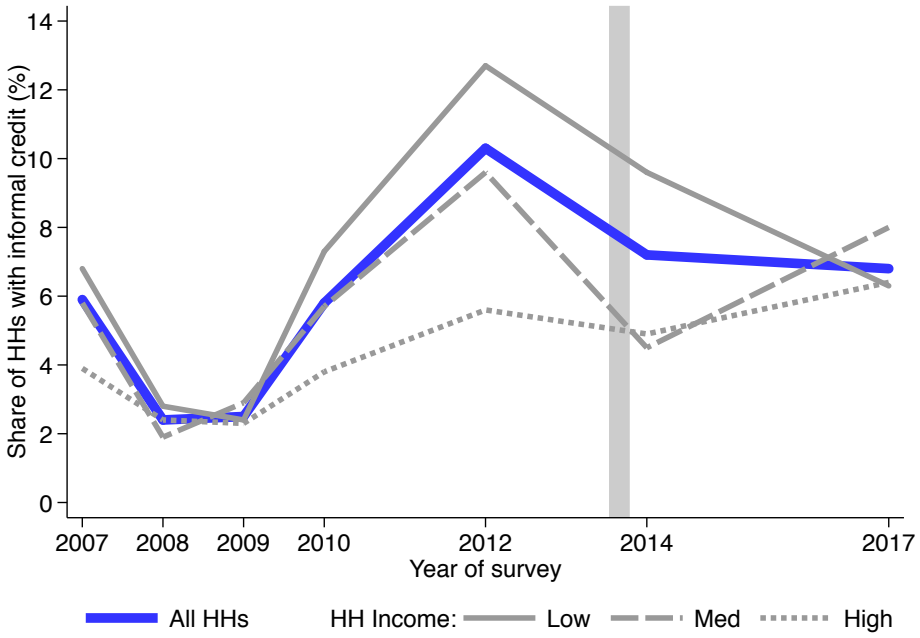
(b) Number of credit lines

(c) Amount of credit card debt

(d) Amount of credit line debt

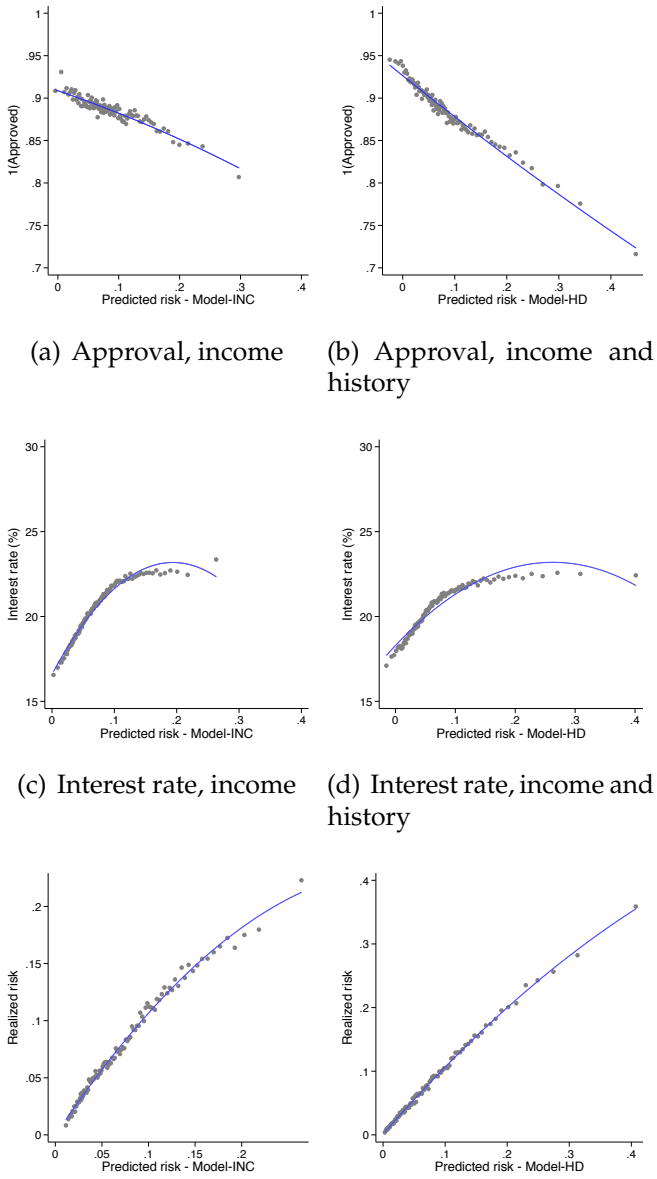
Notes: This figure displays the evolution of the the markets for credit cards and credit lines in the Chilean market using administrative data from SBIF. Panels (a) and (b) display the number of consumers and cards/lines (blue) as well as the number of cards/lines per consumer in the market (black). Panels (c) and (d) display the total amount of credit card/line debt in the market (blue) and per consumer in the market (black). The vertical gray line indicates the policy change.

Figure A.3: Evolution of informal debt penetration



Notes: This figure displays the evolution of the share of households in the Chilean market that hold some kind of informal debt, as measured by the nationally representative Survey of Consumer Finance (EFH, 2018). This statistic covers several sources of informal credit, including family and friends, informal lenders, pawn shops, among others. Data is only available for selected years displayed in the x-axis, which are the ones in which the survey has been implemented. The blue line measures the overall share, whereas gray lines measure shares by terciles of household income. The vertical gray line indicates the policy change.

Figure A.4: Predicted and realized default



(a) Approval, income

(b) Approval, income and history

(c) Interest rate, income

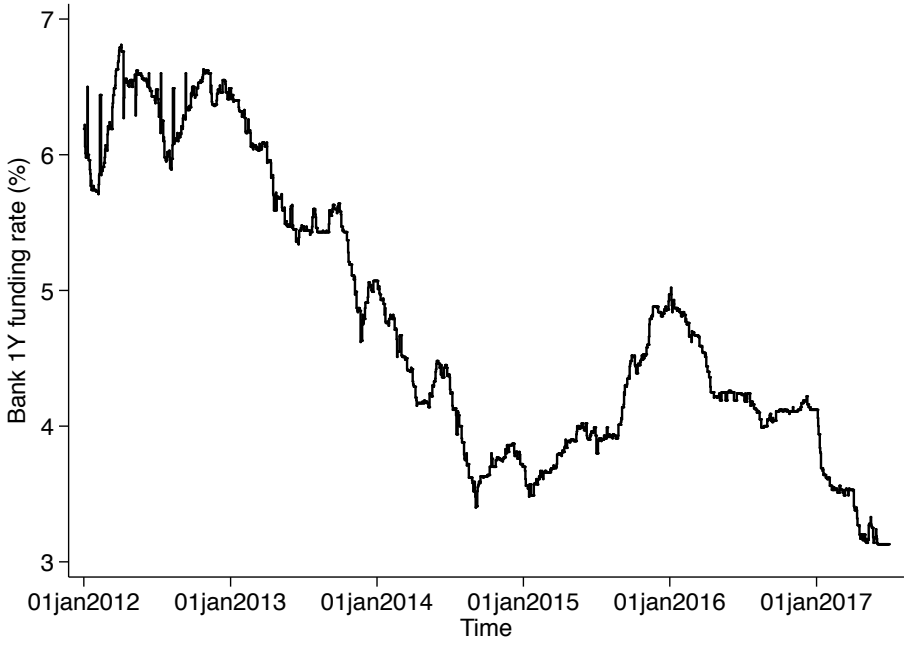
(d) Interest rate, income and history

(e) Realized default, income

(f) Realized default, income and history

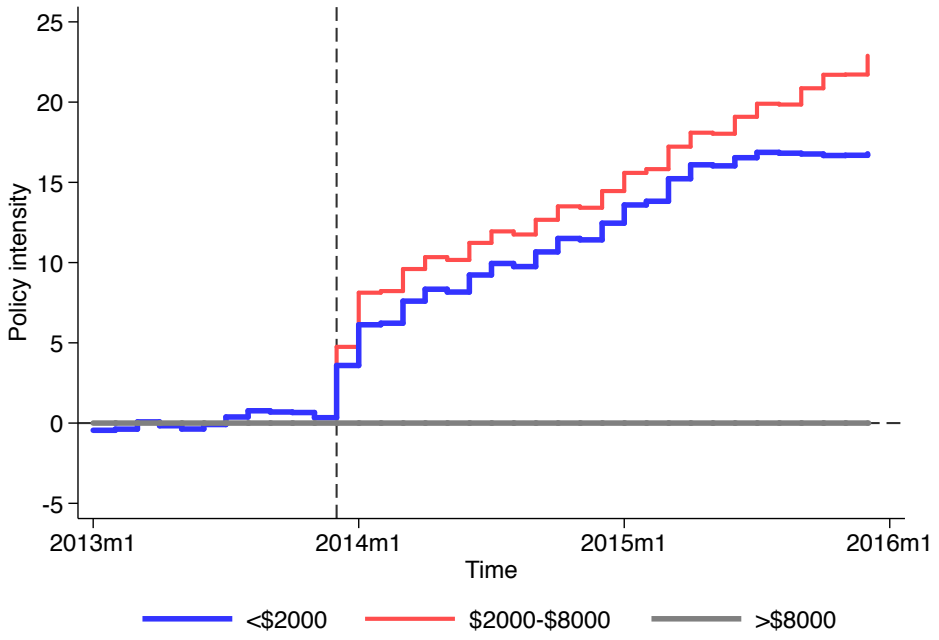
Notes: This figure displays binned scatterplot of predicted loan default probability as constructed using the model described in Section 2.4.1 and realized outcomes. The left column displays results using borrower income and loan to income ratios as main predictors of default, while the right columns adds a long vector of credit history covariates. Panels (a) and (b) display the relationship between predicted default and loan application approval; Panels (c) and (d) display the relationship between predicted default and interest rate; while Panels (e) and (f) display the relationship between predicted default and realized default,. Each dot measures average realized default for loans in each of 100 quantiles of predicted default. The blue line is a quadratic fit of the relationship between both variables.

Figure A.5: Evolution of the funding cost of banks



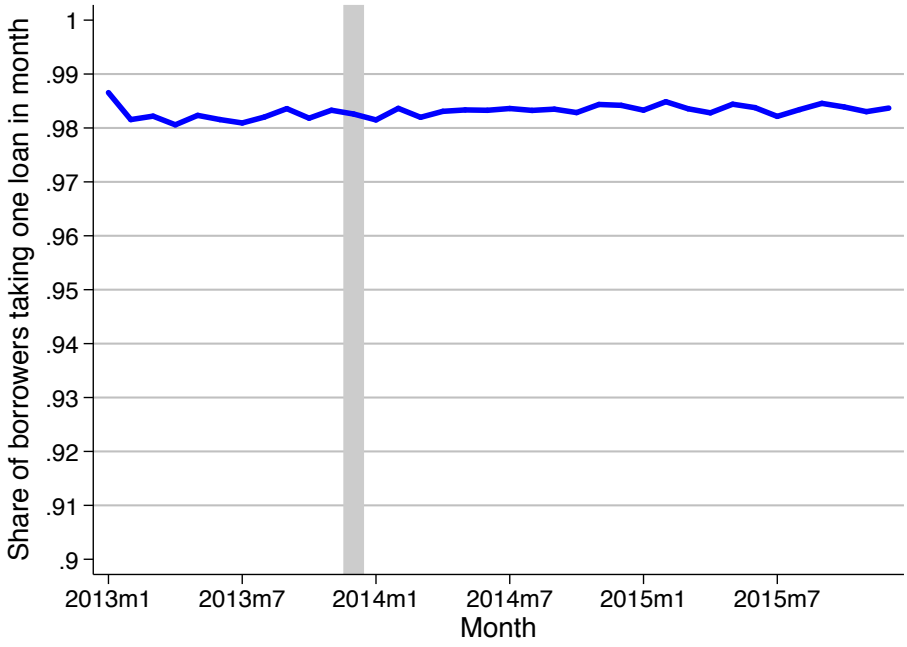
Notes: This figure displays the evolution of the funding cost of banks. This funding cost is calculated as a weighted average of banks deposit rates.

Figure A.6: Treatment intensity variable



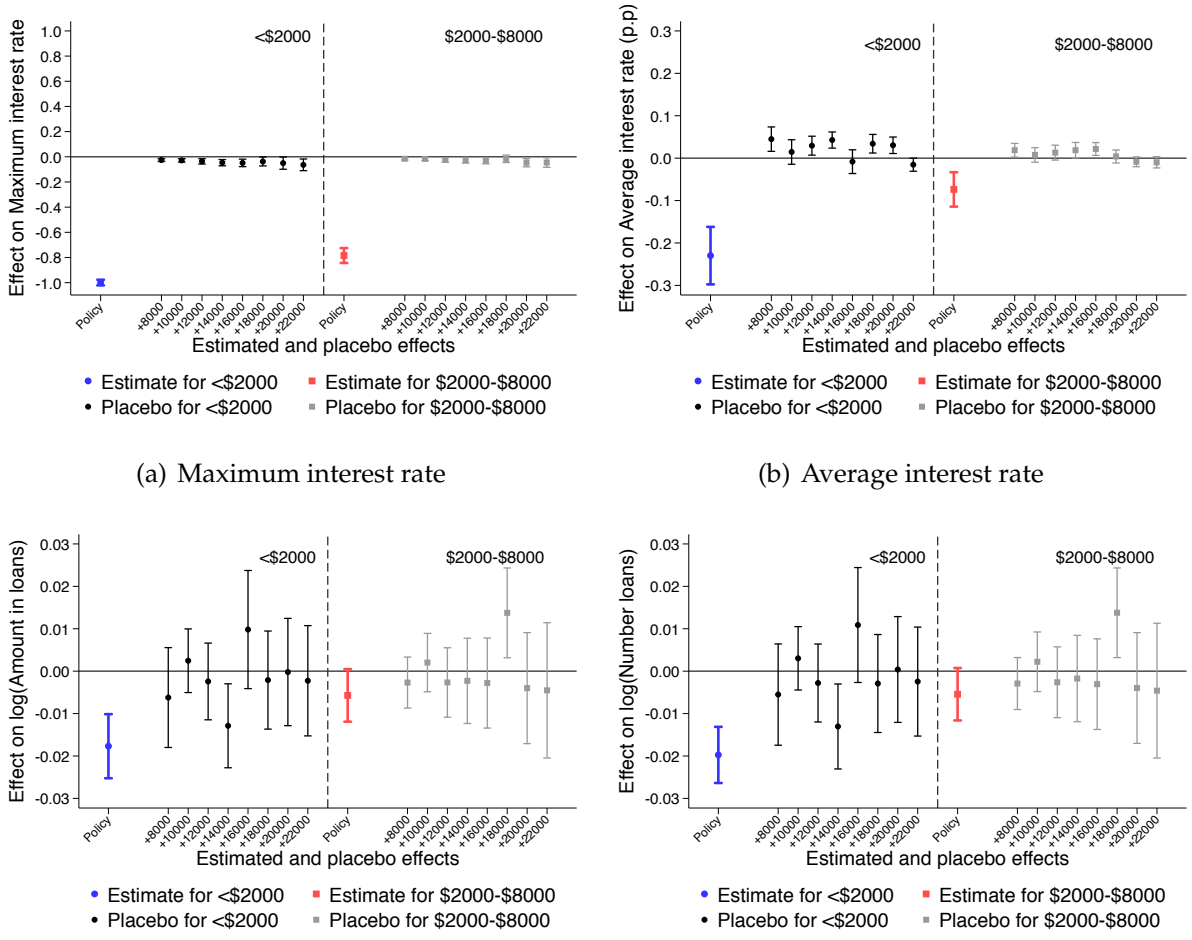
Notes: This figure displays the evolution of the treatment intensity variable defined in Section 1.3.2. This variable is defined as $\Delta^{\bar{\iota}}_{\ell,t} \equiv (\bar{\iota}_{\ell,0} - \bar{\iota}_{\ell,t}) - (\bar{\iota}_{>8000,0} - \bar{\iota}_{>8000,t})$ and measures the decrease in the interest rate cap for a treated loan-size bracket net of the decrease in the interest rate cap for the untreated loan-size bracket of loans in \$0-\$8,000.

Figure A.7: Number of loans per borrower and month, conditional on borrower



Notes: This figure displays the evolution of the share of borrowers taking only one loan in a month in which they borrow. The gray vertical line indicates the policy change.

Figure A.8: Differences-in-differences effects of placebo policies



(a) Maximum interest rate

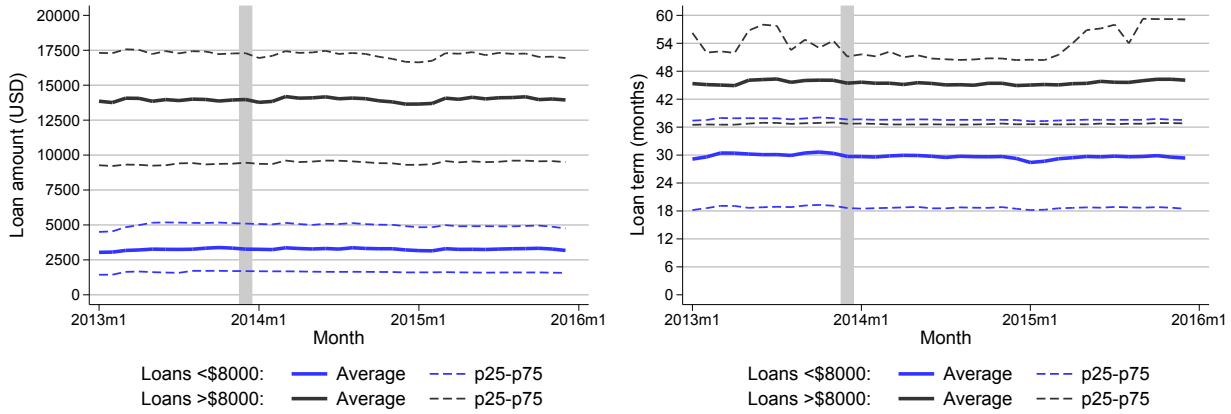
(b) Average interest rate

(c) Amount in loans

(d) Number of loans

Notes: This figure displays the contrast between our estimates from Table 1.3 and Table 1.4 (blue and red) with estimates for a range of placebo policies (black and gray). In each figure, the left panel displays results for loans of \$0-\$2,000 and the left panel displays results for loans of \$2,000-\$8,000. Placebo policies are constructed by using the same policy intensity variables to estimate effects of interest rate regulation on different parts of the loan size distribution. The first placebo policy adds \$8,000 to the actual policy definition, and subsequent placebo policies subsequently add \$2,000. Each dot indicates the estimated coefficient, while spikes indicate 95% confidence intervals clustered at the risk bin-product bin level. All regressions are weighted by the number of loans in the product type bin-risk bin before the policy was implemented.

Figure A.9: Evolution of distribution of application loan amount and term

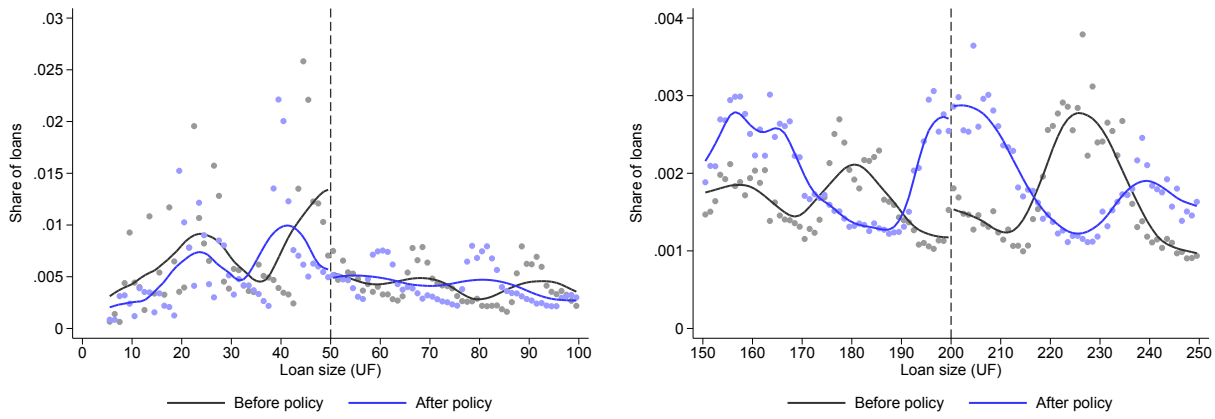


(a) Application loan amount

(b) Application loan term

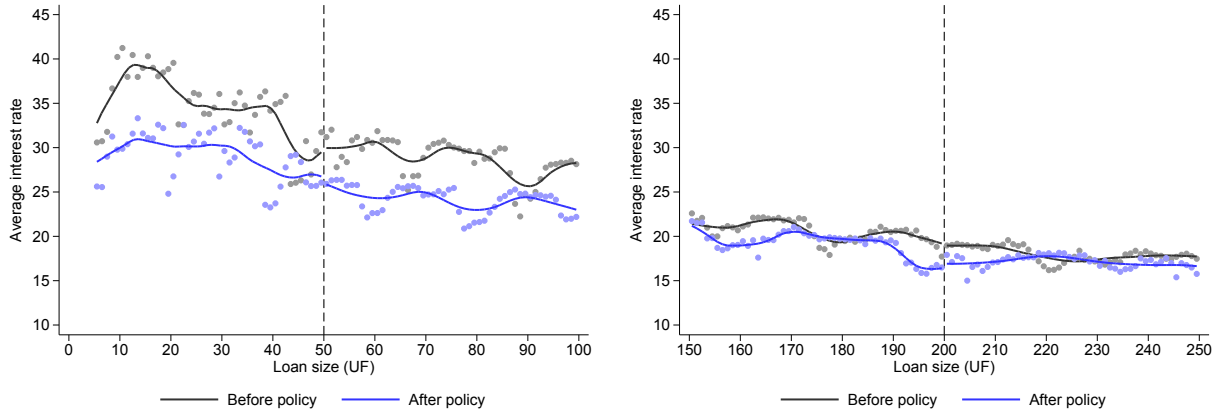
Notes: This figure displays the evolution of the distribution of application loan amount and term separately for loans of \$0-\$8,000 and loans of \$8,000-\$20,000. In particular, each panel displays the average and the 25th and 75th percentiles of each variable for each month.

Figure A.10: Distribution of loan size around policy thresholds



(a) Loan size around $2,000 threshold, before and after

(b) Loan size around $8,000 threshold, before and after

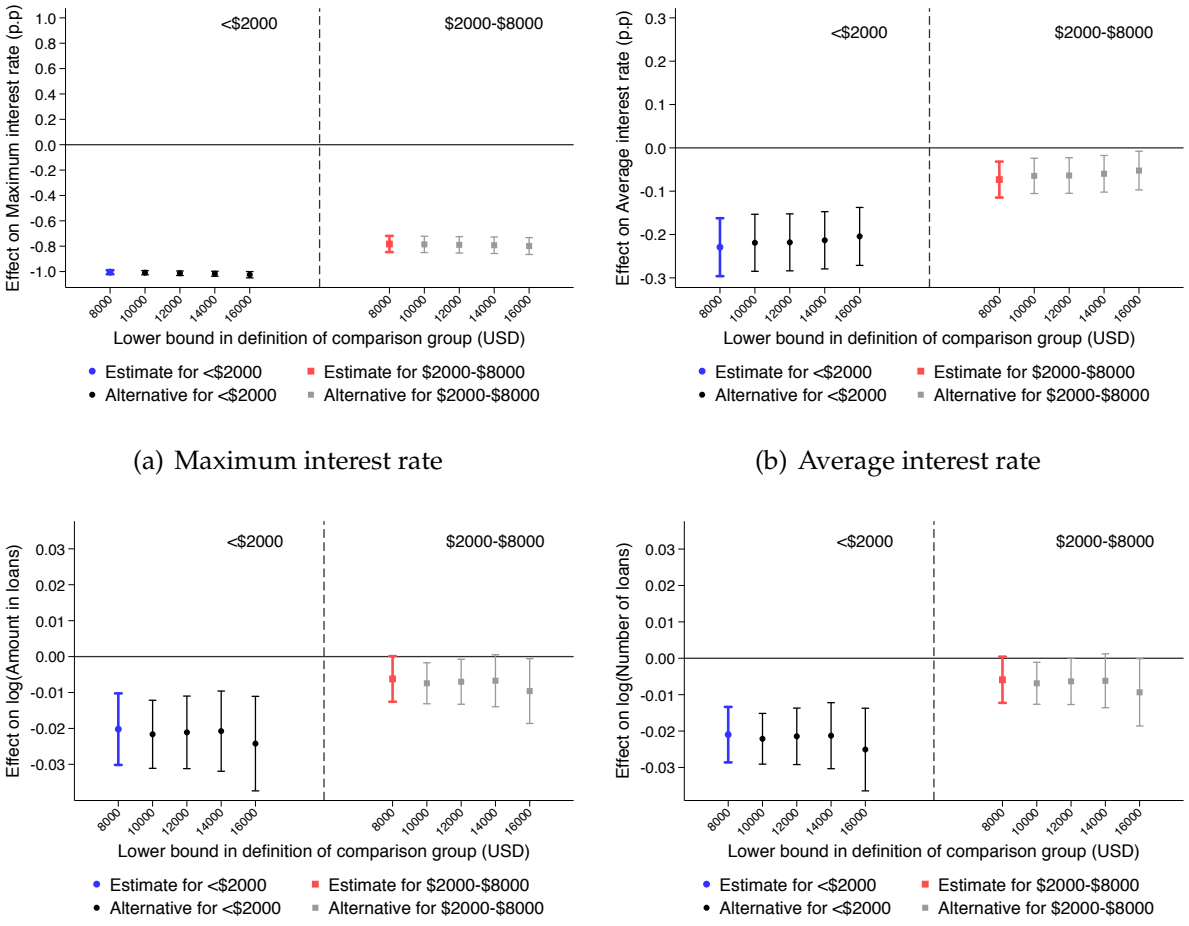


(c) Rates around $2,000 threshold, before and after

(d) Rates around $8,000 threshold, before and after

Notes: This figure displays shares of loans and average interest rates by loan size around policy size thresholds at 50UF ($2,000) and 200UF ($8,000). The data is binned in bins of 1UF ($40). For each bin, dots indicate the share of loans originated and average interest rates. Shares are computed across the $0-$20,000 interval. Gray dots indicate loan originations in the semester before the policy was implemented, between January 2013 and November 2013. Blue dots indicate loan originations in the last semester before the policy was fully in place, between January 2016 and November 2016. Gray and blue lines are local polynomial fits of the relationship between number of loans and loan size in Panels (a) and (b) and between interest rates and loan size in Panels (c) and (d), allowed to differ at both sides of the relevant policy threshold.

Figure A.11: Differences-in-differences effects under alternative comparison groups



(a) Maximum interest rate

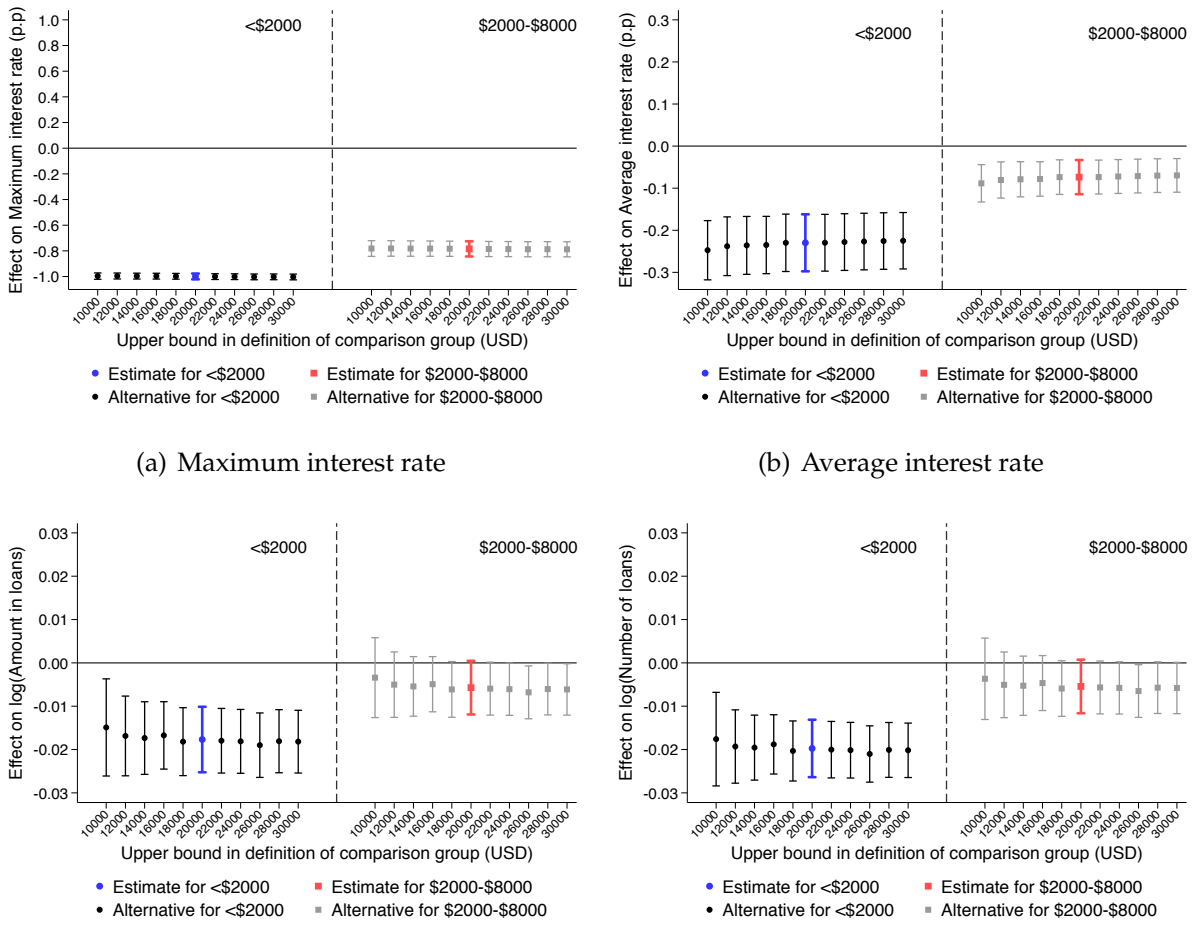
(b) Average interest rate

(c) Amount in loans

(d) Number of loans

Notes: This figure displays the contrast between our estimates from Table 1.3 and Table 1.4 (blue and red) with estimates for a range of alternative comparison groups (black and gray). In each figure, the left panel displays results for loans of \$0-\$2,000 and the left panel displays results for loans of \$2,000-\$8,000. Alternative comparison groups are constructed by shifting the lower bound in the definition of the comparison group, so as to vary the inclusion criterion in terms of the distance to the cutoff set by the policy at \$8,000. The first comparison group sets such upper bound at \$8,000 as in our baseline results, and subsequent alternative comparison groups include loans \$2,000 larger in size. Each dot indicates the estimated coefficient, while spikes indicate 95% confidence intervals clustered at the risk bin-product bin level. All regressions are weighted by the number of loans in the product type bin-risk bin before the policy was implemented.

Figure A.12: Differences-in-differences effects under alternative comparison group size



(a) Maximum interest rate

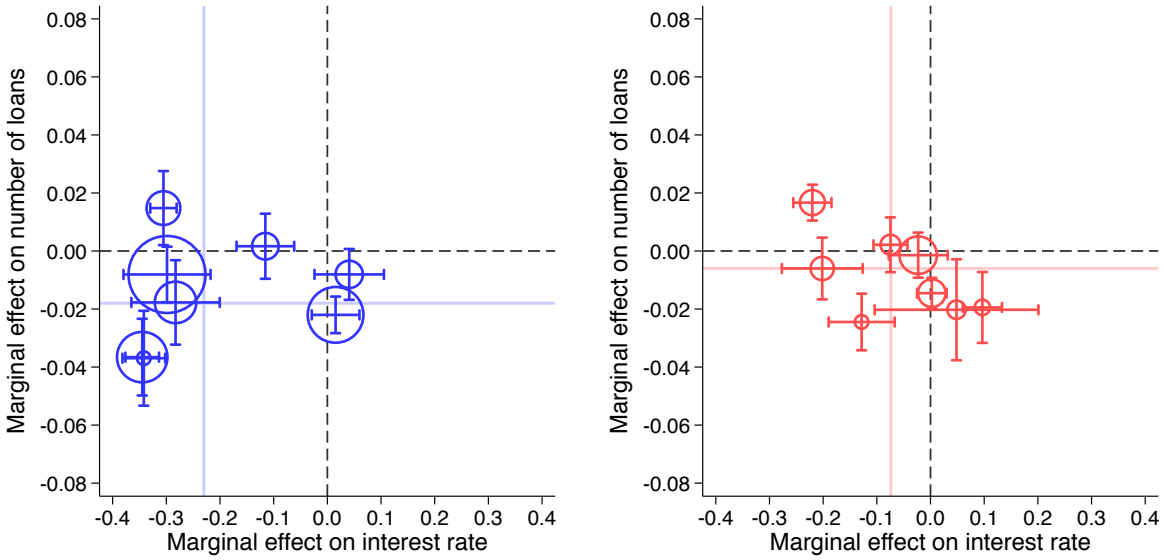
(b) Average interest rate

(c) Amount in loans

(d) Number of loans

Notes: This figure displays the contrast between our estimates from Table 1.3 and Table 1.4 (blue and red) with estimates for a range of alternative comparison groups (black and gray). In each figure, the left panel displays results for loans of \$0-\$2,000 and the left panel displays results for loans of \$2,000-\$8,000. Alternative comparison groups are constructed by shifting the upper bound in the definition of the comparison group, so as to vary the inclusion criterion in terms of the distance to the cutoff set by the policy at \$8,000. The first comparison group sets such upper bound at \$10,000, and subsequent alternative comparison groups include loans \$2,000 larger in size. Each dot indicates the estimated coefficient, while spikes indicate 95% confidence intervals clustered at the risk bin-product bin level. All regressions are weighted by the number of loans in the product type bin-risk bin before the policy was implemented.

Figure A.13: Heterogeneity in effects across banks

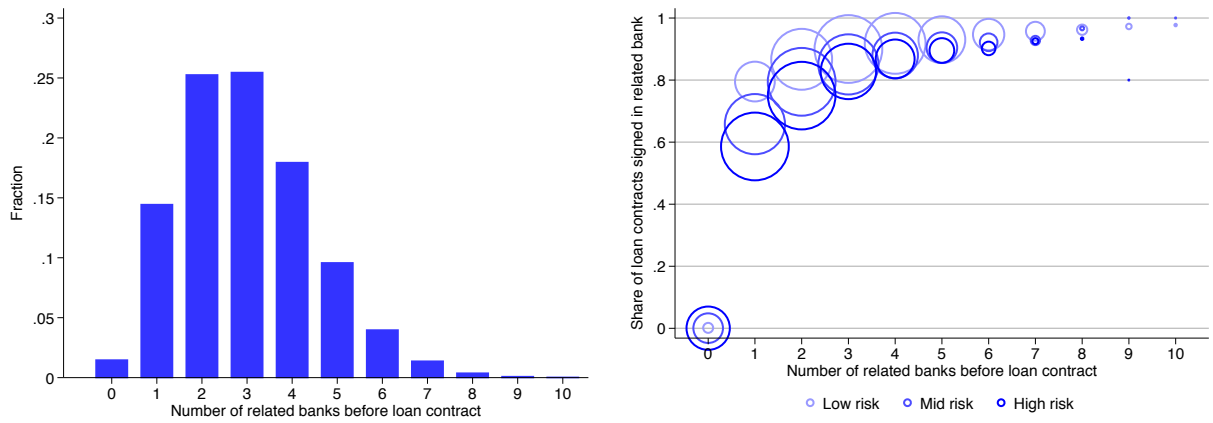


(a) Loans of \$0-\$2,000

(b) Loans of \$2,000-\$8,000

Notes: These figures display marginal effects of interest rate regulation on both number of loans and prices across banks. In each panel, circles indicate estimates for effect on prices on the x-axis and on number of loans on the y-axis the size of the circle is given by the market share of the bank; and spikes indicate standard errors clustered at the risk bin-product bin level. All regressions are weighted by the number of loans in the product type bin-risk bin before the policy was implemented. Solid lines indicate marginal effects estimated across banks, as displayed in Table 1.3 and Table 1.4.

Figure A.14: Relationship between borrowers and banks

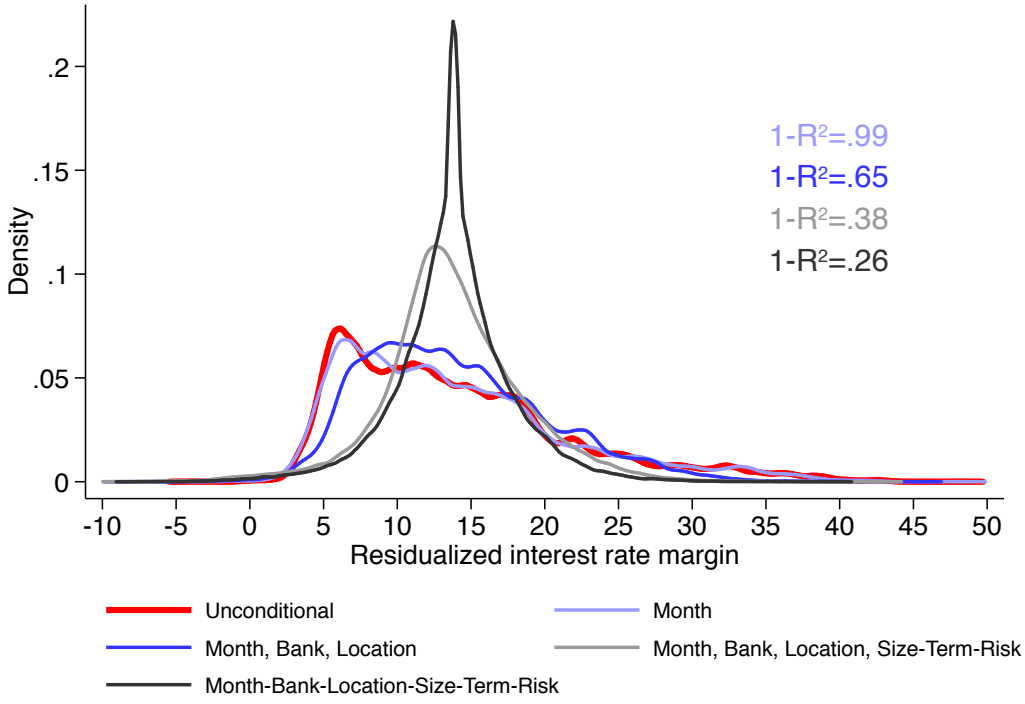


(a) Number of related banks

(b) Loans at related banks

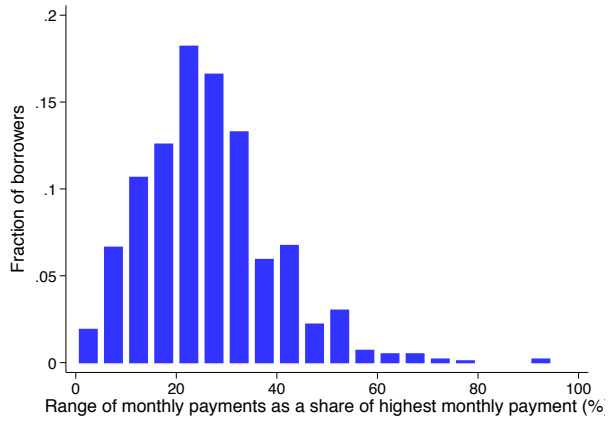
Notes: Panel (a) describes the share of previously related banks for each borrower in the dataset. Panel (b) describes the share of loan contracts signed with a previously related bank for each tercile of borrower risk and each number of previously related banks.

Figure A.15: Price dispersion in consumer loan contracts

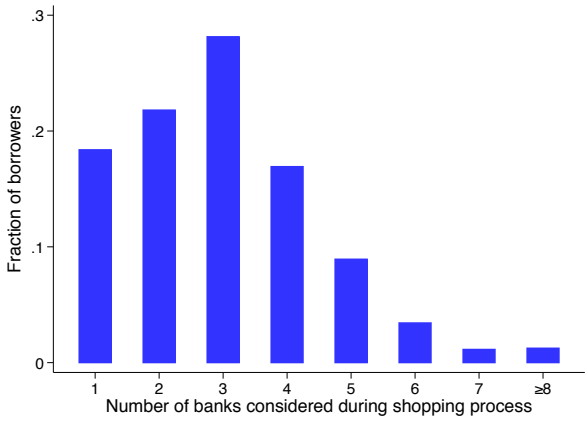


Notes: This figure displays interest rate margins. The red line displays the density of raw interest rate margins in the data. Each additional density displays margins residualized by a increasingly richer sets of covariates, from month FEs to month-bank-size-term-risk FEs.

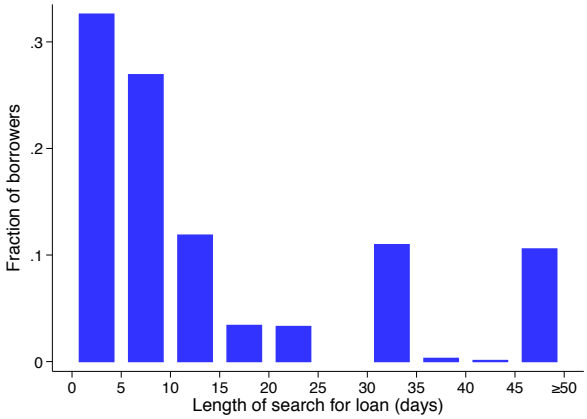
Figure A.16: Survey evidence supporting modeling choices



(a) Perceived price dispersion



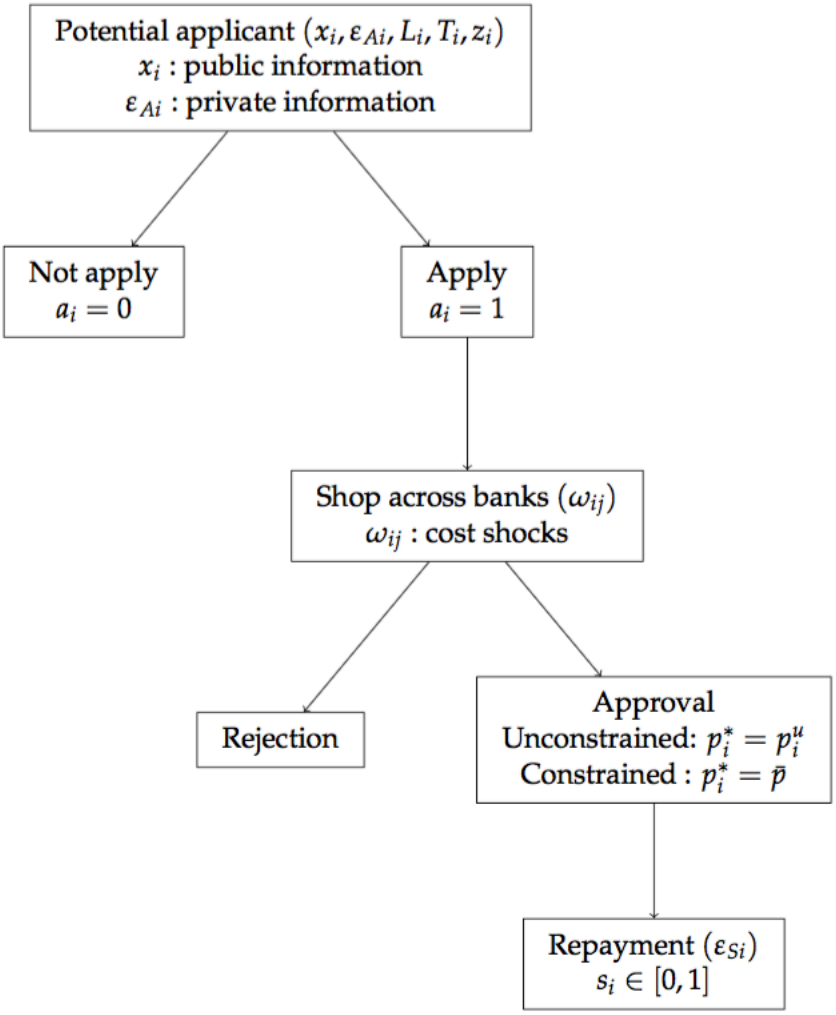
(b) Number of banks considered in shopping process



(c) Length of shopping process

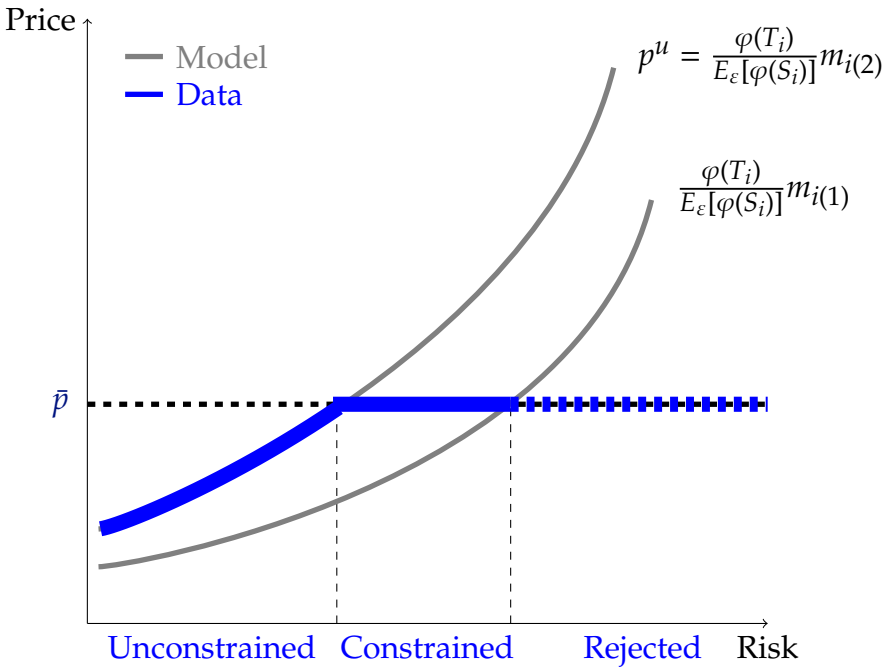
Notes: These figures display survey results related to modeling choices. Panel (a) shows a histogram of the number of banks that borrowers reported to consider during the shopping process for loans. Panel (b) shows a histogram of the length of the search process measured in days between beginning of their search to the end of it, regardless of the approval or rejection of their applications. Panel (c) shows a histogram of perceived price dispersion in the market, as measured by the ratio of the range between the lowest and the highest monthly payment in the market to the highest monthly payment in the market, $\frac{p_H - p_L}{p_H}$.

Figure A.17: Timing of the model



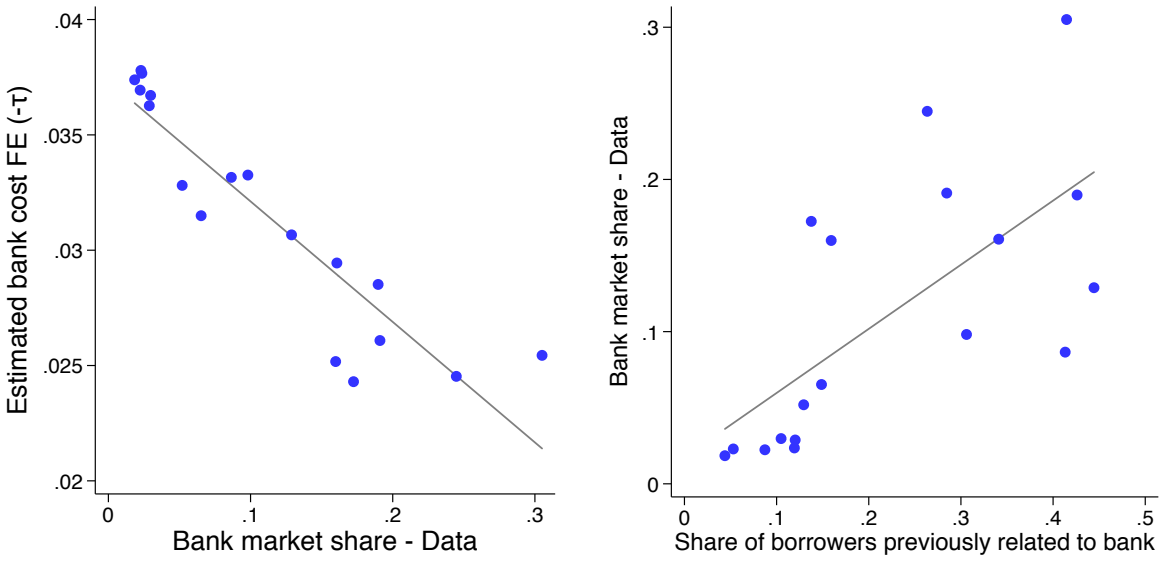
Notes: This figure displays the timing and structure of the model. Exogenous observables characterizing the consumer are covariates x_i, loan amount and term (L_i, T_i), and application cost shifters z_i. Consumer unobservables are $(\varepsilon_{Ai}, \varepsilon_{Si})$, whereas bank unobservables are cost shocks ω_{ij}. Finally, endogenous variables are application choices a_i, approval and pricing choices p_i, and repayment s_i.

Figure A.18: Intuition for identification of bank cost



Notes: This figure provides an illustration of how observed outcomes inform the identification of bank cost. The blue line combines observed application outcomes with observed prices, while the gray lines represent the first order statistic of cost and optimal price for the bank with the lowest cost in the market.

Figure A.19: Relationship between cost estimates and data

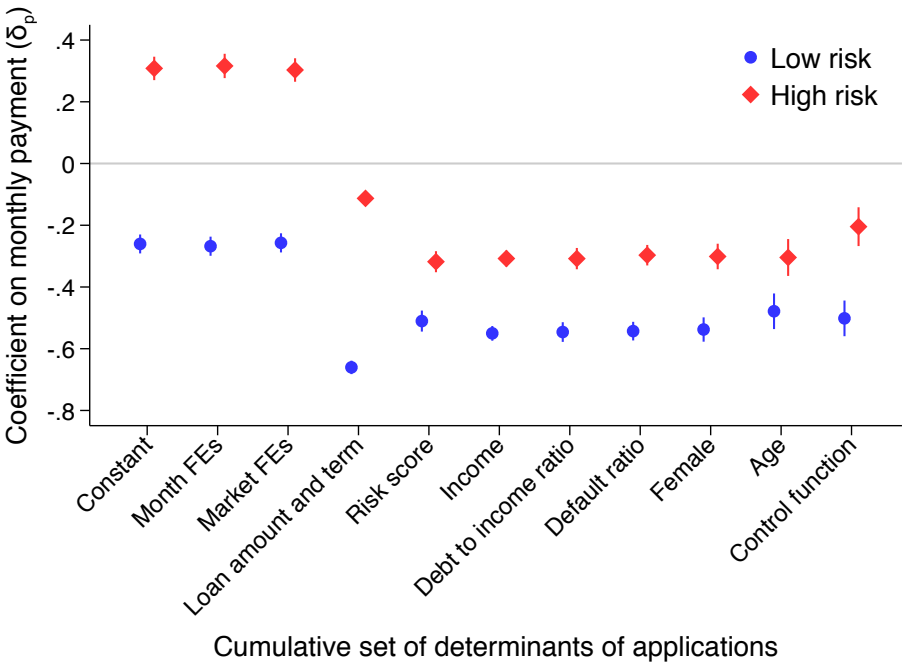


(a) Market shares and cost FEs

(b) Relationships and shares

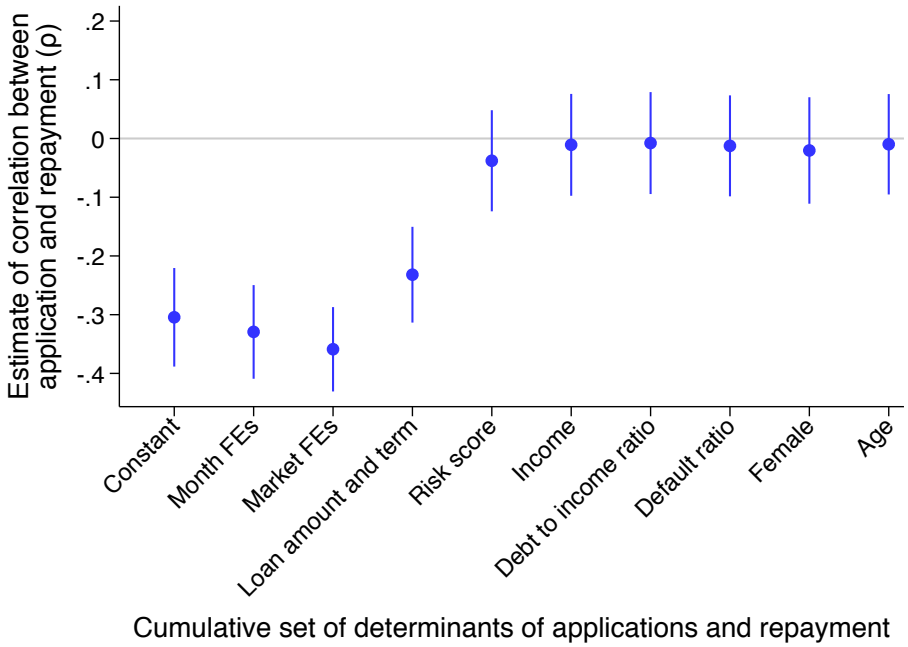
Notes: Panel (a) in this figure displays observed market shares and estimates for bank fixed effects τ_j in ω_{ij}. Panel (b) in this figure displays the correlation between observed shares of previously related borrowers and observed bank market shares.

Figure A.20: Price sensitivity estimates under different specifications



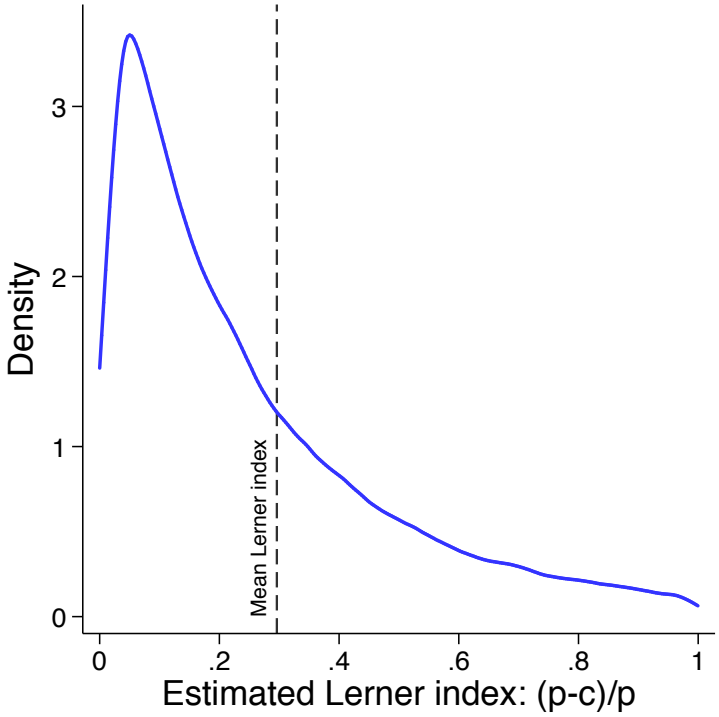
Notes: This figure displays estimates for the coefficient on expected monthly payment in the application equation (δ_p) under different specifications. In particular, we estimate that equation using an increasingly rich set of borrower covariates to assess the role of unobservables related to both applications and bank pricing in terms of driving our estimates of price sensitivity, in line with Altonji et al. (2005). The last estimates include a control function as an additional covariate in estimation, following Petrin and Train (2010). The figure displays estimates for low-risk (blue) and high-risk (red) borrowers. Dots indicate estimates of δ_p. Lines indicate 95% confidence intervals.The first specification we consider includes a constant, and subsequent specifications add more covariates sequentially. Our preferred specification for the analysis in the paper is that with the full vector of covariates.

Figure A.21: Selection estimate under different specifications



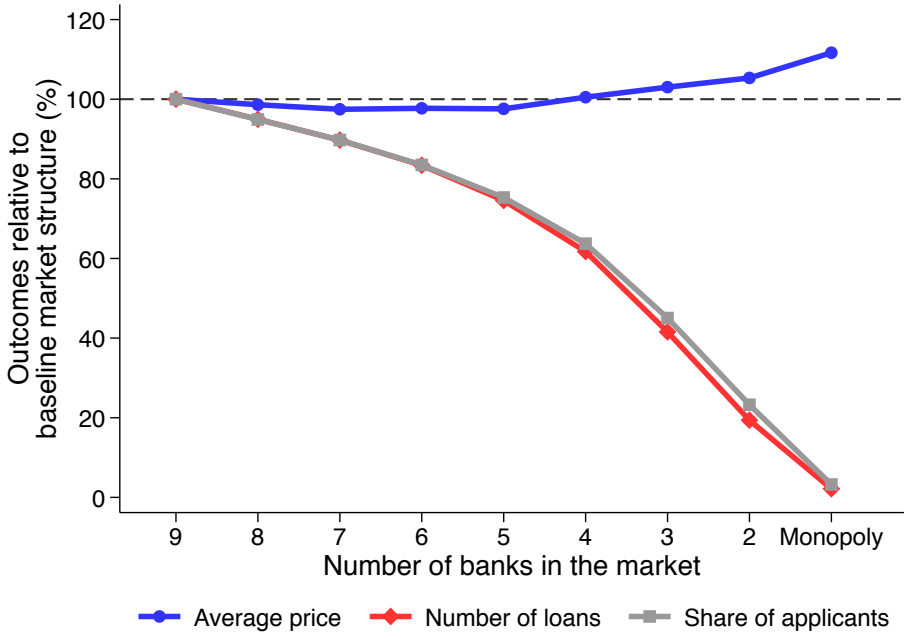
Notes: This figure displays estimates for the coefficient measuring the correlation between shocks to application and repayment (ρ) under different specifications. In particular, we estimate the application and repayment equations using an increasingly rich set of borrower covariates for *both* of them to assess the extent to which available observables are able to capture patterns of risk selection into the market. Dots indicate estimates of ρ. Lines indicate 95% confidence intervals. The first specification we consider includes a constant, and subsequent specifications add more covariates sequentially. Our preferred specification for the analysis in the paper is that with the full vector of covariates.

Figure A.22: Simulated bank profit margins



Notes: This figure displays results for the distribution of the predicted Lerner index using model estimates.

Figure A.23: The effects of interest rate regulation under different market structures: baseline outcomes



Notes: This figure display baseline outcomes under baseline interest rate regulation under different market structures. We start with the baseline market structure of 9 banks, and sequentially merge banks until a scenario in which the market is served by a monopoly.

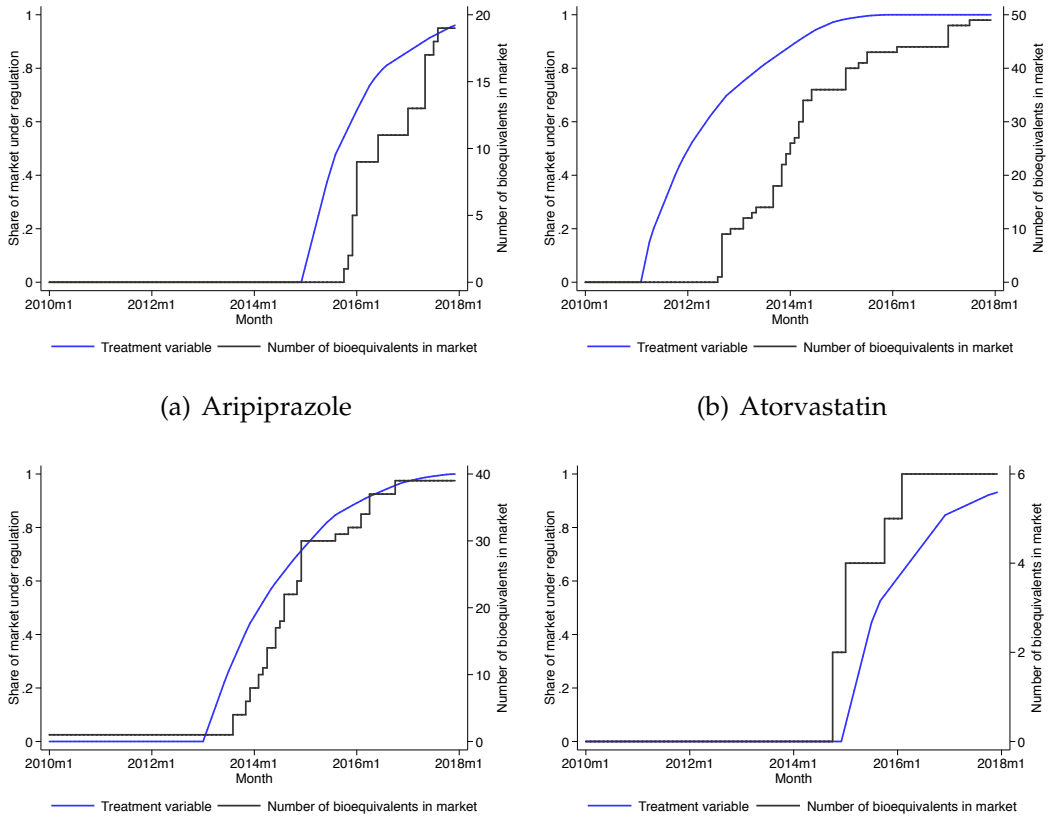
Figure B.1: Labeling of Bioequivalent Drugs

(a) Instructions for bioequivalent drugs labeling

(b) Examples of labeled bioequivalent drugs

Notes: This figures display both instructions and examples of required labeling of bioequivalent drugs. The objective of this labeling was to highlight drugs with BE approval.

Figure B.2: Policy Variation induced by Bioequivalence Requirements



(a) Aripiprazole

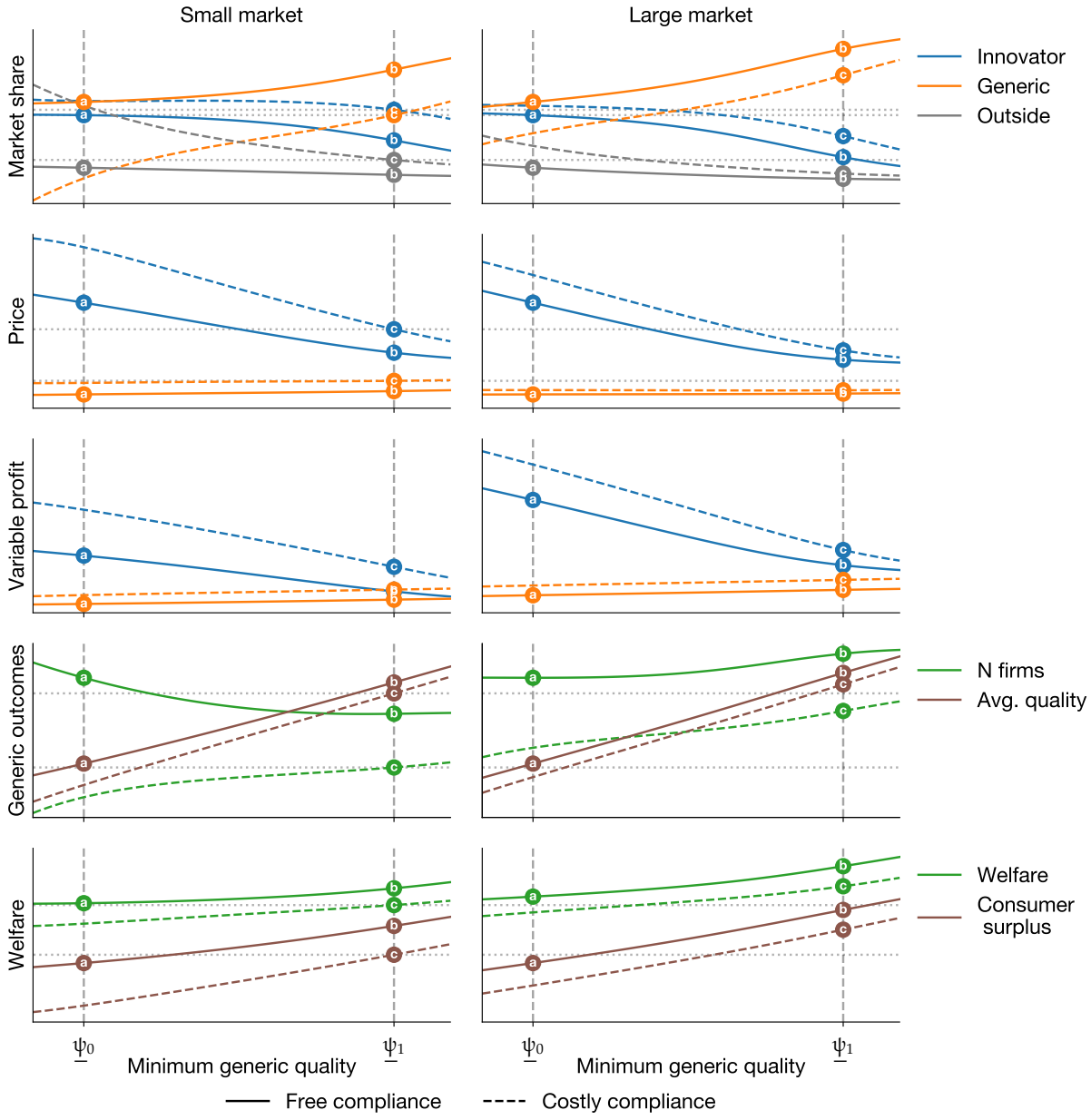
(b) Atorvastatin

(c) Citalopram

(d) Deflazacort

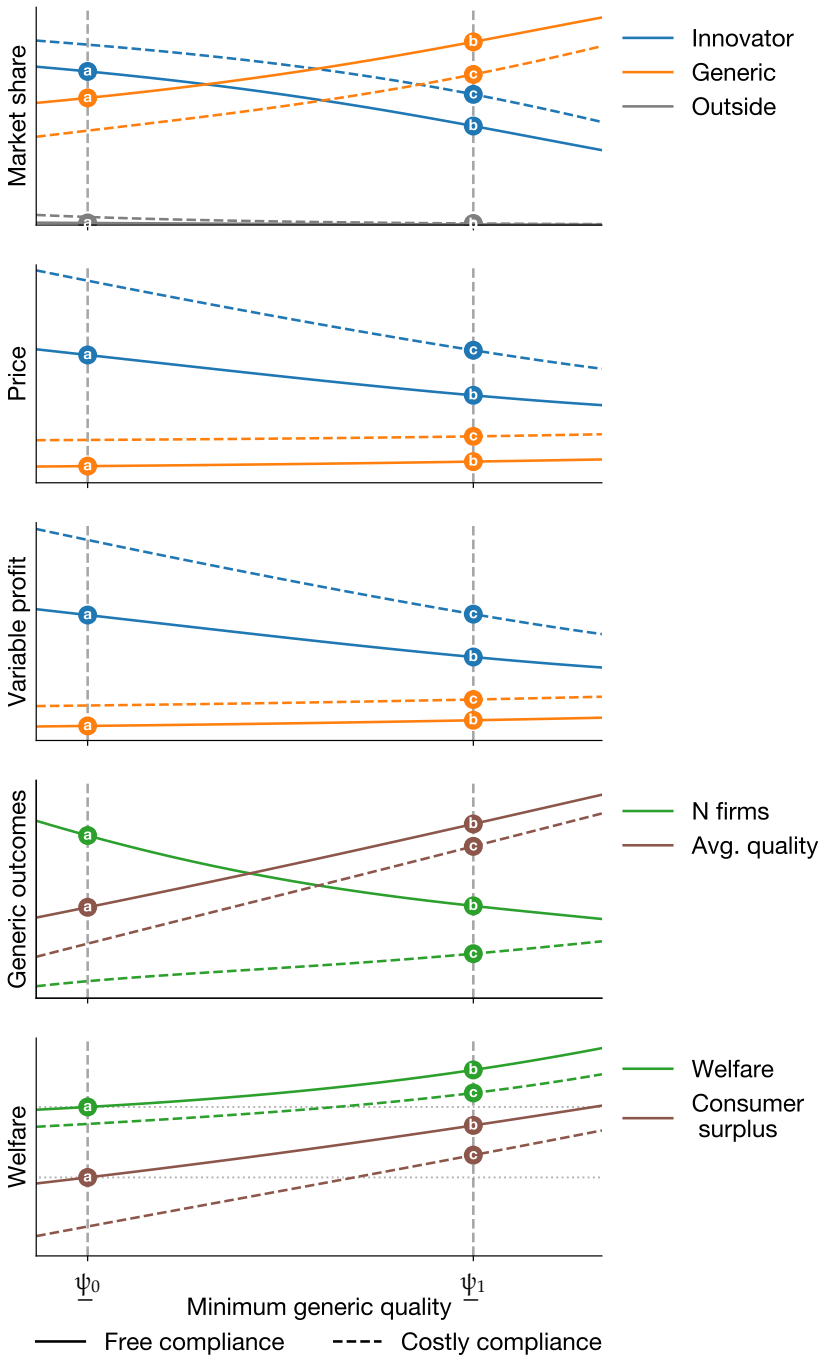
Notes: Each figure displays the values of the treatment variable and the number of BEs in a different market. This version of the treatment variable uses the first deadline as the relevant date. The instrument is displayed in blue, and takes a value of 0 before the first decree, and then increases as renewal dates of drugs in the molecule approach. The number of BE drugs in the molecule is displayed in gray. These four examples are plotted along all other markets in our sample in Figure A.17-b.

Figure B.3: Effects of Quality Regulation, Small versus Large markets



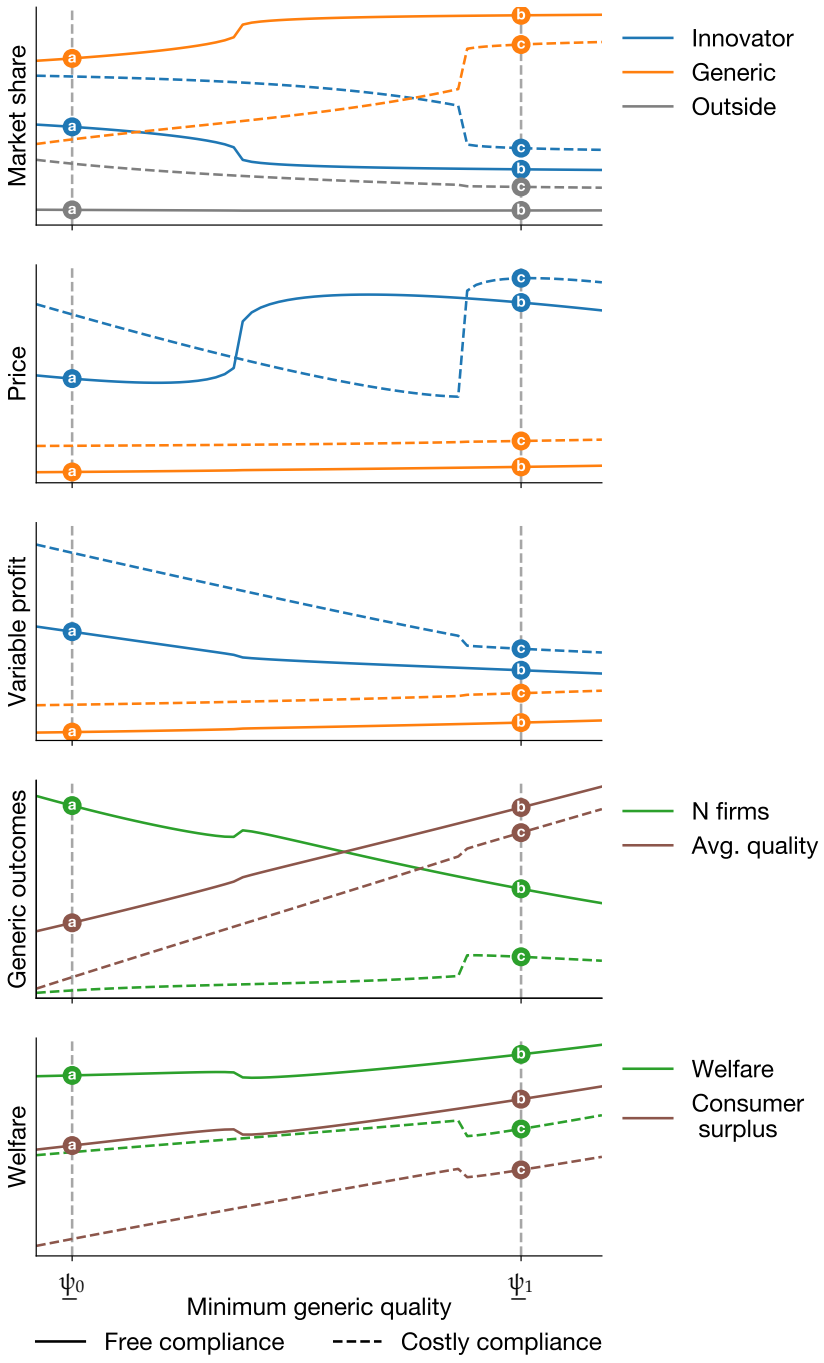
Notes: Market outcomes for different levels of minimum quality in a small market (left column) and large market (right column). The dashed (solid) lines represent a situation with (no) compliance costs. Example minimum qualities before and after regulation are indicated by $\underline{\psi}_0$ and $\underline{\psi}_1$, where point **a** indicates pre-reform outcomes, **b** indicates post-reform outcomes if compliance was free, while **c** indicates post-reform outcomes with costly compliance. Dotted horizontal lines indicating post-reform outcomes with costly compliance in small markets.

Figure B.4: Effects of Quality Regulation, welfare enhancing



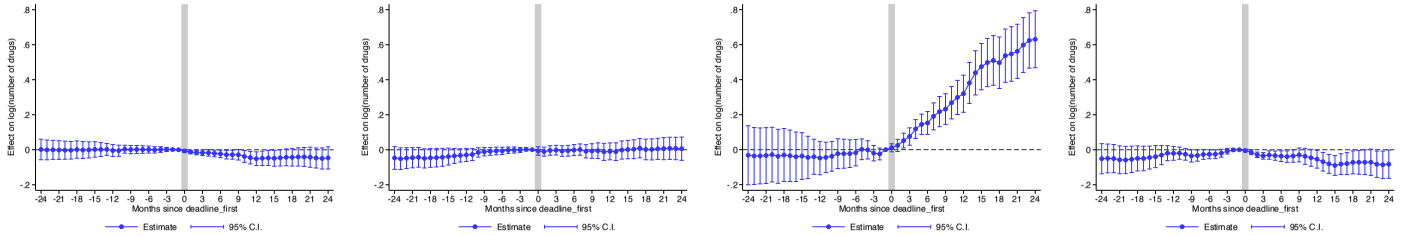
Notes: Market outcomes for different levels of minimum quality in the market. The dashed (solid) lines represent a situation with (no) compliance costs. Example minimum qualities before and after regulation are indicated by $\underline{\psi}_0$ and $\underline{\psi}_1$, where point **a** indicates pre-reform outcomes, **b** indicates post-reform outcomes if compliance was free, while **c** indicates post-reform outcomes with costly compliance.

Figure B.5: Effects of Quality Regulation with a loyal segment

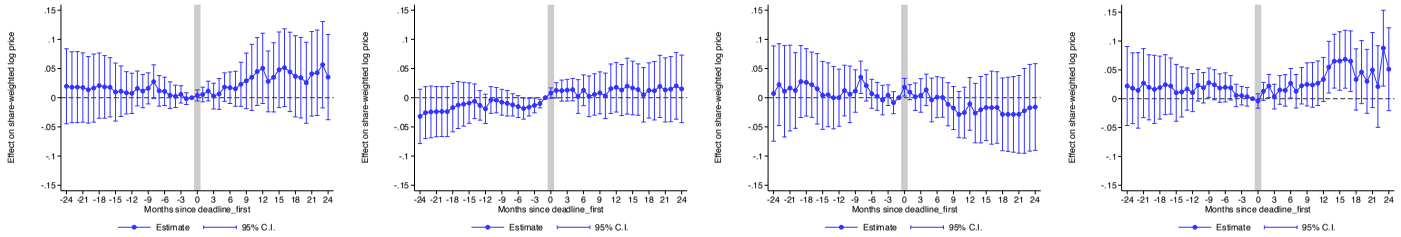


Notes: Market outcomes for different levels of minimum quality in the market. The dashed (solid) lines represent a situation with (no) compliance costs. Example minimum qualities before and after regulation are indicated by $\underline{\psi}_0$ and $\underline{\psi}_1$, where point **a** indicates pre-reform outcomes, **b** indicates post-reform outcomes if compliance was free, while **c** indicates post-reform outcomes with costly compliance.

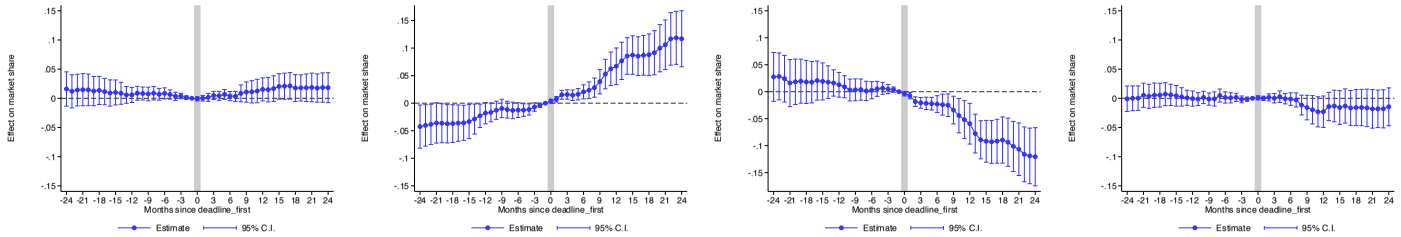
Figure B.6: Policy Effects using an Event Study Approach



(a) Number of drugs, all drugs (b) Number of drugs, innovator (c) Number of drugs, BE generics (d) Number of drugs, non-BE generics



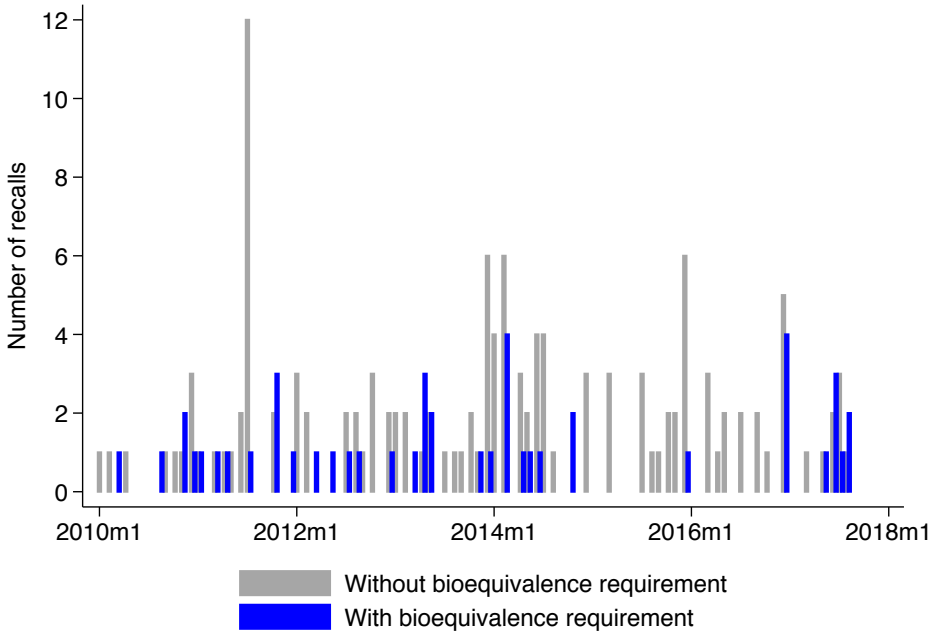
(e) Prices, all drugs (f) Prices, innovator (g) Prices, branded (h) Prices, unbranded



(i) Shares, innovator (j) Shares, branded BE (k) Shares, branded non-BE (l) Shares, unbranded

Notes: This figure displays results from event study specifications described in Section B.2, using the first bioequivalence deadline as policy event. Dots indicate point estimates and lines indicate 95% confidence intervals based on standard errors clustered at the market level. Coefficients are displayed for 24 months before and 24 months after the policy event. The coefficient on the month previous to the event is normalized to zero. The first row displays results for the number of drugs in the market, the second row displays results for the price index defined in equation (2.4), and the third row displays results for drug market shares.

Figure B.7: Number of Recalls per Month

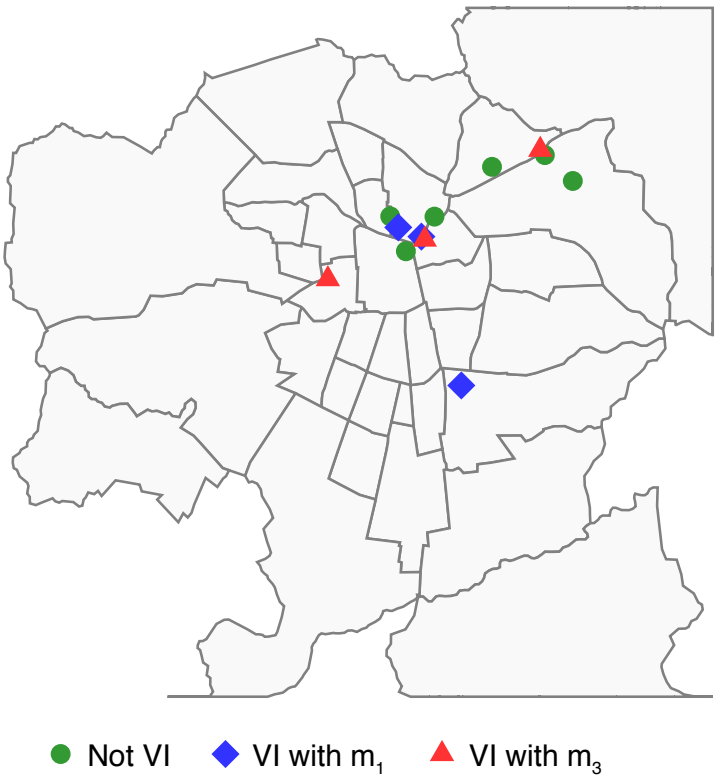


Notes: The figure shows the number of product recalls over time split into markets with bioequivalence requirements and markets without bioequivalence requirements.

Figure B.8: Consumer Survey: Elicitation of Perceived Quality and Price

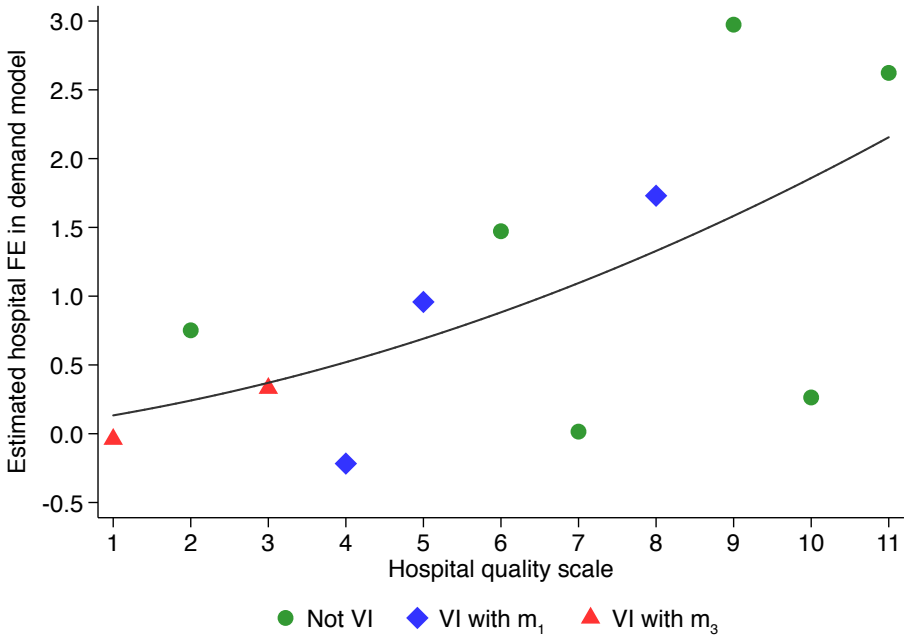
Notes: This figure displays the sheet surveyors provided consumers in our survey sample. This sheet displays the 4 drugs we used as an example to elicit perceived quality and price differences. While observing this sheet, surveyors asked consumers first to assign a score in a 1-7 scale to each drug regarding their quality, and then to estimate the price of each drug given that the innovator had a price of $50,000 CLP ($77.5 U.S. dollars).

Figure C.1: Location of Hospitals in the Market



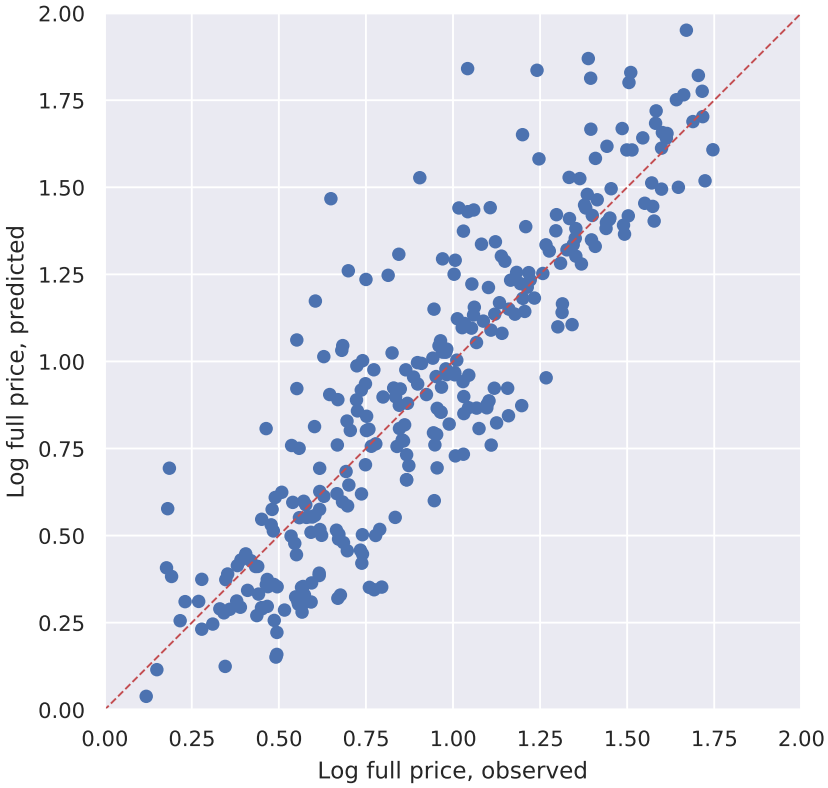
Notes: This figure shows the location of hospitals in the market. The map covers most of urban Santiago, out market of interest. Green circles indicate independent hospitals, blue diamonds indicate hospitals that are vertically integrated with insurer m_1, and red triangles indicate hospitals vertically integrated with insurer m_3.

Figure C.2: Unobserved preferences for hospitals and observable hospital attributes



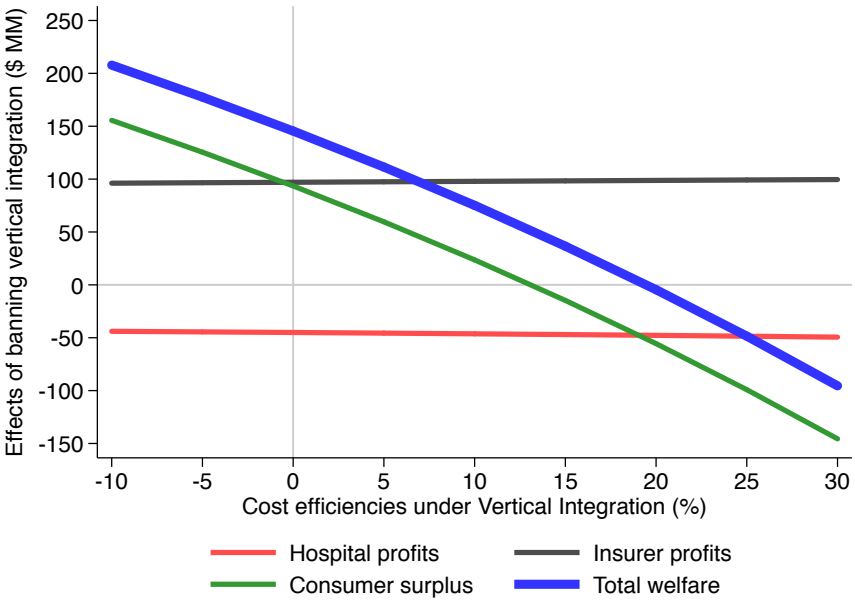
Notes: This figure shows the relationship between estimated hospital fixed effects in Column (2) of Table 3.5 and an objective measure of quality, which comes from the position of the hospital in the Webometrics Ranking of World Hospitals developed by Cybermetrics Lab (Cybermetrics Lab, 2016). h_3 is not considered in the ranking, and therefore not included in the figure. Green circles indicate independent hospitals, blue diamonds indicate hospitals that are vertically integrated with insurer m_1, and red triangles indicate hospitals vertically integrated with insurer m_3. The black line is a quadratic fit for the relationship between both variables.

Figure C.3: Observed and Predicted Hospital Prices

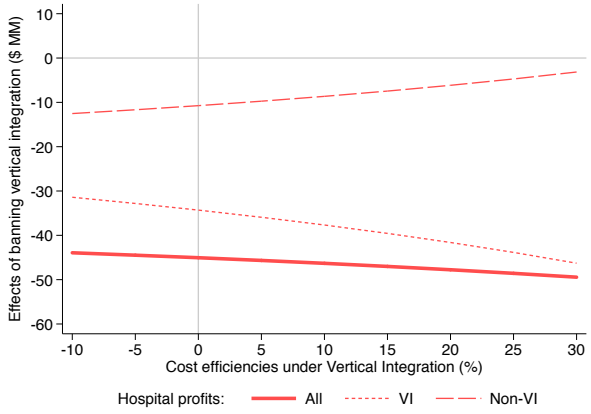


Notes: This figure shows the comparison between predicted and observed mean prices for each combination of insurer-hospital-year. Recall that predicted prices are constructed using estimates from equation (3.13).

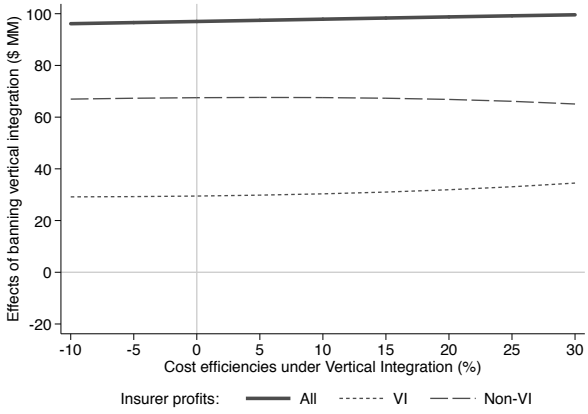
Figure C.4: Welfare Effects of Banning Vertical Integration for Shared Cost Efficiencies: Chilean Market



(a) Aggregate effects



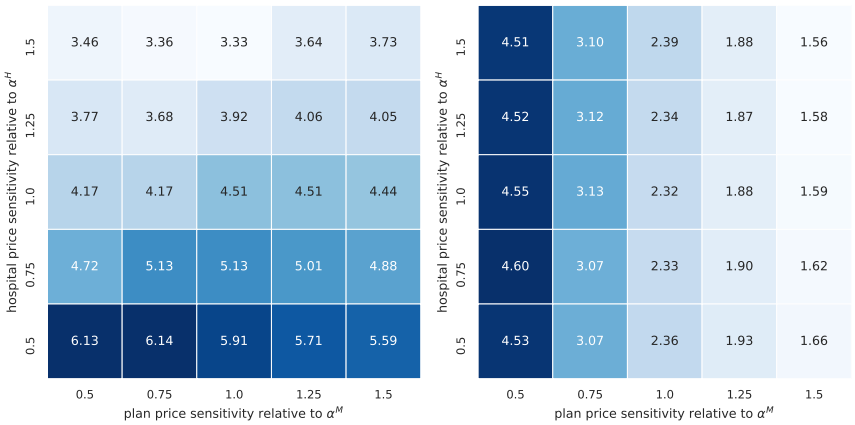
(b) Effects on hospitals, by vertical integration



(c) Effects on insurers, by vertical integration

Notes: This figure shows the effect of banning vertical integration on equilibrium welfare outcomes for different levels of cost efficiencies, for the Chilean market. In this case, vertical integration efficiency reduce the cost for all insurers at integrated hospitals. Panel (a) displays aggregate effects, Panels (b) and (c) respectively decompose effects on hospitals and insurers profits by vertical integration at baseline. The x-axis in each graph measure cost efficiencies induced by vertical integration on integrated hospitals for all insurers. Blue lines show overall welfare effects, green lines show effects on consumer surplus, red lines show effects on hospital profits, and black lines show effects on insurer profits.

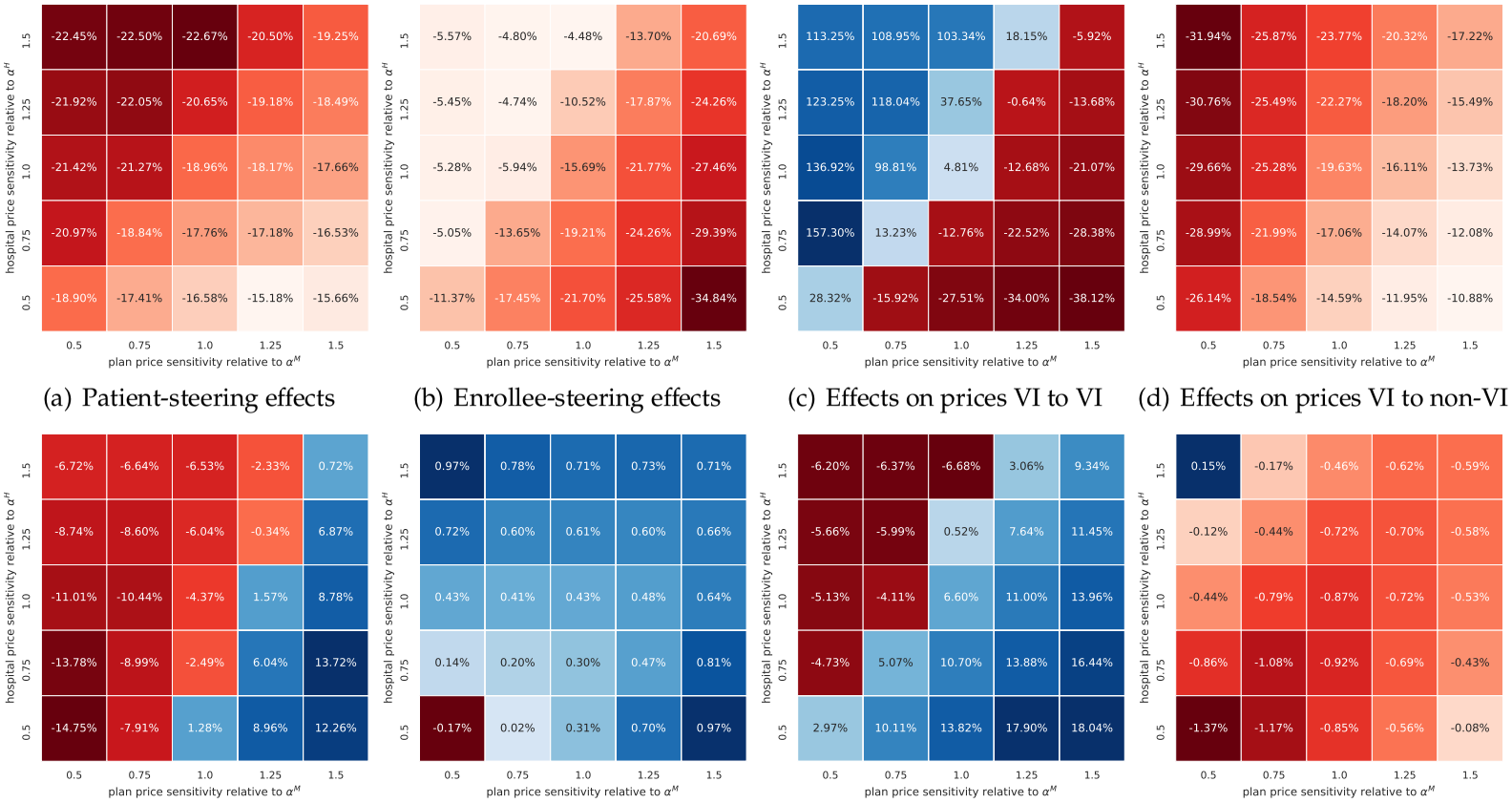
Figure C.5: The Role of Price Sensitivity for the Effects of Banning Vertical Integration: Baseline Prices



(a) Baseline hospital prices (b) Baseline plan premiums

Notes: This figure shows simulated baseline hospital prices and plan premiums under the baseline market structure for a grid of consumer price sensitivity. For each plot, we show results for a 5×5 grid of hospital and plan demand sensitivity defined by $(\tau^M \times \alpha_i^M, \tau^H \times \alpha_f^H)$ for $\tau = \{0.5, 0.75, 1, 1.25, 1.5\}$. Darker blue indicates a higher price or premium respectively.

Figure C.6: The Role of Price Sensitivity for the Effects of Banning Vertical Integration with 20% Cost Efficiency: Outcomes

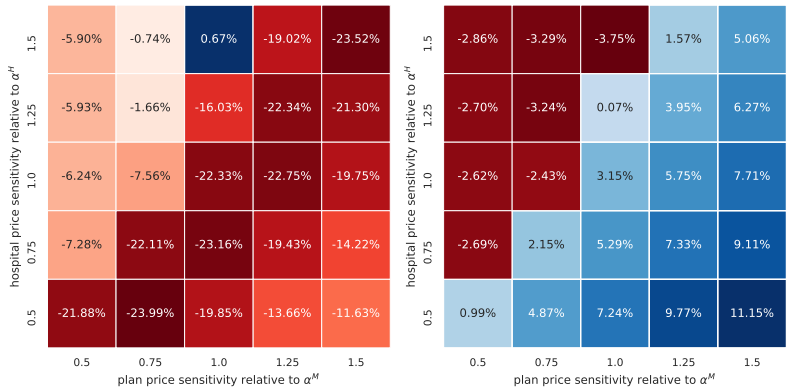


(a) Patient-steering effects (b) Enrollee-steering effects (c) Effects on prices VI to VI (d) Effects on prices VI to non-VI

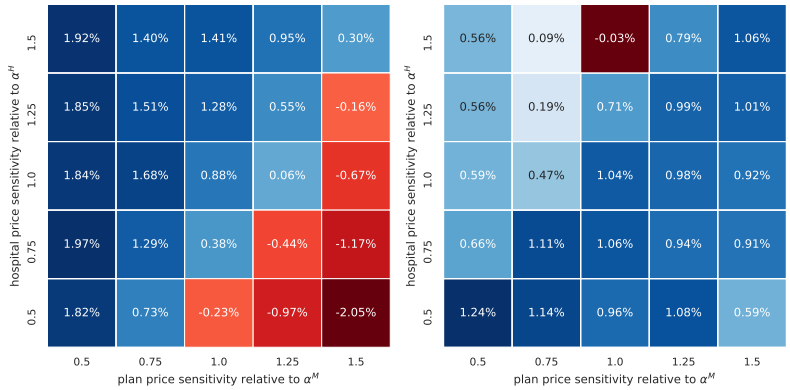
(e) Effects on prices non-VI to VI (f) Effects on prices non-VI to non-VI (g) Effects on VI plan premiums (h) Effects on non-VI plan premiums

Notes: This figure shows the effect of banning vertical integration on a variety of outcomes for a grid of consumer price sensitivity. For each plot, we show results for a 5×5 grid of hospital and plan demand sensitivity defined by $(\tau^M \times \alpha_i^M, \tau^H \times \alpha_f^H)$ for $\tau = \{0.5, 0.75, 1, 1.25, 1.5\}$. Panels (a) and (b) quantify patient- and enrollee-steering effects respectively in the baseline scenario, as described in Section 3.4.5. Panels (c) through (f) display the effect of banning vertical integration on quantity-weighted average hospital prices from a VI/non-VI hospital to a VI/non-VI insurer, and panels (g) and (h) display plan premiums for VI/non-VI insurers. For each such figure, blue (red) indicates increases (decreases) in the outcome, and the intensity of the color indicates the relative magnitude of the change. This simulation is done assuming a 20% cost efficiency within integrated firms.

Figure C.7: The Role of Price Sensitivity for the Effects of Banning Vertical Integration with 20% Cost Efficiency: Welfare Effects



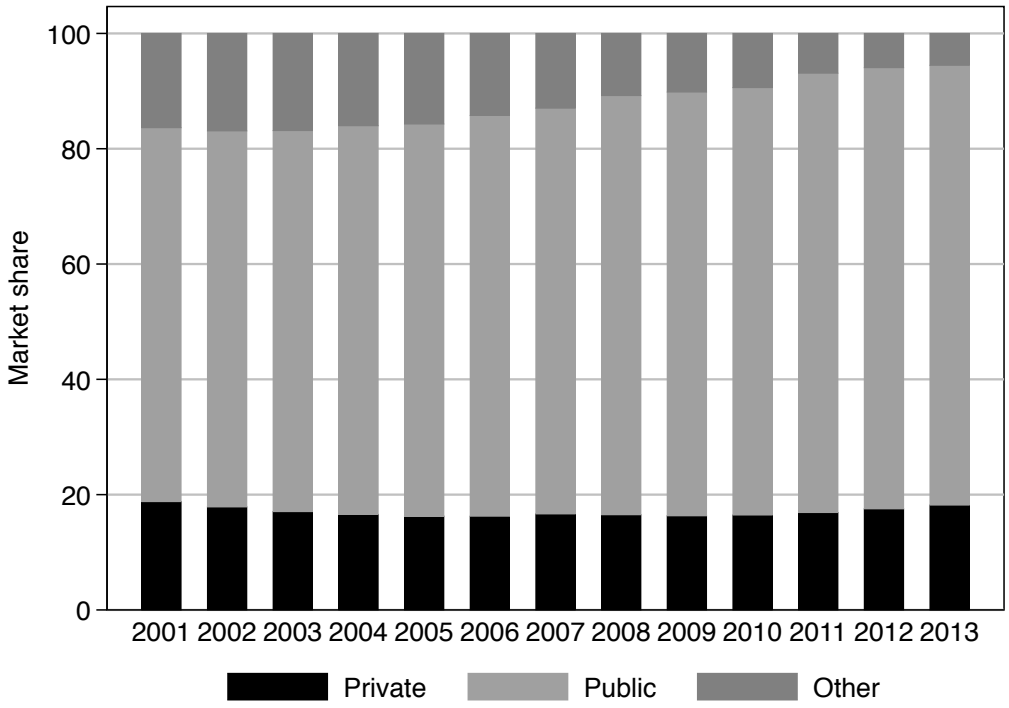
(a) Effects on hospital profits (b) Effects on insurer profits



(c) Effects on consumer surplus (d) Effects on total welfare

Notes: This figure shows the effect of banning vertical integration on a variety of outcomes for a grid of consumer price sensitivity. For each plot, we show results for a 5×5 grid of hospital and plan demand sensitivity defined by $(\tau^M \times \alpha_i^M, \tau^H \times \alpha_f^H)$ for $\tau = \{0.5, 0.75, 1, 1.25, 1.5\}$. Panels (a) through (d) display the effect of banning vertical integration on a variety of equilibrium outcomes, measure as a percentage change relative to the baseline level. The outcomes are hospital and insurer profits, consumer surplus and overall welfare. For each such figure, blue (red) indicates increases (decreases) in the outcome, and the intensity of the color indicates the relative magnitude of the change. This simulation is done assuming a 20% cost efficiency within integrated firms.

Figure C.8: Insurance market shares in across sectors



Notes: This figure displays the evolution of market shares of different types of insurance in Chile.

Table A.1: Determinants of loan performance

	(1)	(2)	(3)	(4)	(5)
	1{Default}				
log(Income)	-0.421***	-0.376***	-0.501***	-0.498***	-0.498***
	(0.004)	(0.005)	(0.005)	(0.005)	(0.005)
Consumer debt to income ratio	0.013***	-0.020***	0.054***	0.054***	0.054***
	(0.004)	(0.006)	(0.006)	(0.006)	(0.006)
Mortgage debt to income ratio	-0.217***	0.136***	0.101***	0.104***	0.104***
	(0.005)	(0.010)	(0.010)	(0.010)	(0.010)
log(Consumer debt)		0.108***	0.111***	0.109***	0.108***
		(0.011)	(0.011)	(0.011)	(0.011)
No consumer debt		1.059***	0.934***	0.747***	0.746***
		(0.029)	(0.029)	(0.031)	(0.031)
No consumer debt ≥90-day default		-0.531***	-0.414***	-0.414***	-0.415***
		(0.017)	(0.017)	(0.017)	(0.017)
No consumer debt ≥90-day default		-0.458***	-0.467***	-0.468***	-0.468***
		(0.008)	(0.008)	(0.008)	(0.008)
Consumer ≥90-day default to debt ratio		0.591***	0.630***	0.618***	0.619***
		(0.035)	(0.036)	(0.036)	(0.036)
Consumer <90-day default to debt ratio		0.425***	0.554***	0.519***	0.518***
		(0.093)	(0.094)	(0.094)	(0.094)
log(Mortgage debt)		-0.746***	-0.794***	-0.821***	-0.822***
		(0.040)	(0.041)	(0.041)	(0.041)
No mortgage debt		-0.663***	-0.838***	-0.911***	-0.912***
		(0.077)	(0.080)	(0.080)	(0.080)
No mortgage debt ≥90-day default		-0.308***	-0.339***	-0.360***	-0.360***
		(0.049)	(0.050)	(0.050)	(0.050)
No mortgage debt <90-day default		-0.626***	-0.620***	-0.625***	-0.625***
		(0.025)	(0.025)	(0.025)	(0.025)
Mortgage ≥90-day default to debt ratio		0.052	0.178	0.195	0.195
		(0.136)	(0.136)	(0.136)	(0.136)
Mortgage <90-day default to debt ratio		-2.557***	-2.063***	-2.116***	-2.117***
		(0.400)	(0.376)	(0.376)	(0.376)
Change in consumer debt		0.226***	0.201***	0.201***	0.201***
		(0.005)	(0.005)	(0.005)	(0.005)
Change in consumer debt ≥90d default		0.010***	0.006*	0.006*	0.006*
		(0.004)	(0.004)	(0.004)	(0.004)
Change in mortgage debt		-0.011***	-0.012***	-0.012***	-0.012***
		(0.003)	(0.003)	(0.003)	(0.003)
Change in mortgage debt ≥90d default		0.042***	0.027***	0.025***	0.025***
		(0.006)	(0.007)	(0.007)	(0.007)
Age			-0.411***	-0.407***	-0.407***
			(0.003)	(0.003)	(0.003)
Female			-0.308***	-0.310***	-0.309***
			(0.007)	(0.007)	(0.007)
Previously related to any bank				-0.283***	-0.283***
				(0.016)	(0.016)
Local unemployment rate					0.015**
					(0.007)
Constant	-1.907***	0.113	0.234***	0.599***	0.603***
	(0.024)	(0.078)	(0.080)	(0.082)	(0.082)
Observations	916,934	916,934	916,436	916,436	916,436
Pseudo R-squared	0.034	0.054	0.079	0.080	0.080
Market FE	Y	Y	Y	Y	Y

Notes: All columns display results from logit regressions of individual loan default outcomes on borrower covariates. All covariates are standardized. Credit history variables are computed as average over the year previous to each loan. Standard errors in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table A.2: Effects excluding loans around the $8,000 threshold

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	Maximum interest rate			Average interest rate			log(Applications)			log(Credit volume)		
	All	Low-risk	High-risk	All	Low-risk	High-risk	All	Low-risk	High-risk	All	Low-risk	High-risk
Loans in $0 – $2000	-1.003***	-0.907***	-0.996***	-0.220***	-0.108***	-0.253***	-0.005	0.002	-0.013*	-0.019***	-0.013***	-0.029***
	(0.012)	(0.062)	(0.018)	(0.034)	(0.015)	(0.042)	(0.004)	(0.003)	(0.007)	(0.003)	(0.003)	(0.004)
Loans in $2000 – $8000	-0.838***	-0.662***	-0.831***	-0.075***	-0.032***	-0.120***	-0.001	0.001	-0.007	-0.008**	-0.007**	-0.014***
	(0.020)	(0.028)	(0.027)	(0.024)	(0.011)	(0.028)	(0.004)	(0.003)	(0.006)	(0.003)	(0.003)	(0.003)
Observations	2,304	2,304	2,145	2,304	2,304	2,145	2,304	2,304	2,034	2,304	2,304	2,304
R-squared	0.991	0.979	0.985	0.983	0.986	0.964	0.995	0.990	0.994	0.984	0.984	0.966
	log(Number of loans)			Risk-I	Risk-H	log(Income)	Share of loans under 1Y default			Months under 1Y default		
	All	Low-risk	High-risk	All	All	All	All	Low-risk	High-risk	All	Low-risk	High-risk
Loans in $0 – $2000	-0.021***	-0.012***	-0.032***	-0.082***	-0.034***	0.004***	-0.097***	-0.066***	-0.112***	-0.004***	-0.002***	-0.003***
	(0.003)	(0.003)	(0.003)	(0.005)	(0.011)	(0.001)	(0.018)	(0.007)	(0.036)	(0.001)	(0.000)	(0.001)
Loans in $2000 – $8000	-0.008**	-0.006*	-0.015***	-0.027***	-0.009	0.000	-0.050***	-0.023***	-0.080***	-0.002***	-0.001***	-0.003***
	(0.003)	(0.003)	(0.003)	(0.006)	(0.008)	(0.001)	(0.012)	(0.007)	(0.025)	(0.000)	(0.000)	(0.001)
Observations	2,304	2,304	2,304	2,304	2,304	2,304	2,304	2,304	2,145	2,304	2,304	2,145
R-squared	0.992	0.988	0.987	0.988	0.978	0.989	0.909	0.720	0.696	0.907	0.698	0.700
Product bin FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Product bin-month of year FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Month FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y

Notes: This table display estimated coefficients from equation (1.4) using a sample that excludes loans of size between $6,000 and $10,000. For each outcome, the regression is estimated across borrower risk bins and separately by borrower risk bin. All regressions include risk bin-product bin fixed effects and risk bin-month fixed effects. For default outcomes, the estimating sample is restricted to loans originated before December 2015, so as to allow for a year long period after origination in which the outcomes are measured. Share of loans under default in first year is computed in a 0-100 scale. Predicted risk is computed as described in Section 1.2.2 and measured in a 0-100 scale. Income is measured in thousands of U.S. dollars. All regressions are weighted by the number of loans in the product type bin-risk bin before the policy was implemented. Clustered standard errors at the risk bin-product bin level are displayed in parentheses. *** p<0.01, ** p<0.05, * p<0.1.

Table A.3: Summary statistics for household survey

Variable	N	Mean	SD	p10	p50	p90
A - Experience in credit market						
Debit account	1,003	0.84	0.36	0.00	1.00	1.00
Checking account	1,003	0.89	0.31	0.00	1.00	1.00
Debit card	1,003	0.97	0.17	1.00	1.00	1.00
Credit card	1,003	0.98	0.15	1.00	1.00	1.00
Credit line	1,003	0.88	0.32	0.00	1.00	1.00
Consumer loan	1,003	0.98	0.14	1.00	1.00	1.00
Car loan	1,003	0.35	0.48	0.00	0.00	1.00
Mortgage	1,003	0.50	0.50	0.00	0.00	1.00
College loan	1,003	0.34	3.15	0.00	0.00	1.00
B - Shopping behavior						
Number of considered banks	963	2.99	1.65	1.00	3.00	5.00
Number of applications	963	1.35	0.80	1.00	1.00	2.00
Duration of search period in days	963	15.15	28.65	1.00	7.00	30.00
Offline shopping	963	0.81	0.39	0.00	1.00	1.00
Perceived range of prices (%)	994	25.70	12.64	10.53	24.81	41.18
C - Economic hardships						
Experienced economic hardship	1,003	0.59	0.49	0.00	1.00	1.00
Financed economic hardship with formal credit	1,003	0.55	0.50	0.00	1.00	1.00
Financed economic hardship with savings/assets	1,003	0.27	0.44	0.00	0.00	1.00
Financed economic hardship with other	1,003	0.35	0.48	0.00	0.00	1.00
Stopped paying consumer loan	1,003	0.28	0.45	0.00	0.00	1.00
Stopped paying credit card	1,003	0.43	0.50	0.00	0.00	1.00
Stopped paying utility bills	1,003	0.11	0.31	0.00	0.00	1.00
Stopped paying rent	1,003	0.02	0.14	0.00	0.00	0.00
Stopped paying mortgage	1,003	0.06	0.24	0.00	0.00	0.00
Stopped paying car loan	1,003	0.07	0.25	0.00	0.00	0.00
Stopped paying student loan	1,003	0.06	0.23	0.00	0.00	0.00
Stopped paying health bills	1,003	0.07	0.25	0.00	0.00	0.00
Stopped paying other bills	1,003	0.13	0.34	0.00	0.00	1.00
Cut expenditure on non-durables	1,003	0.87	0.34	0.00	1.00	1.00
Cut expenditure on personal care	1,003	0.37	0.48	0.00	0.00	1.00
Cut expenditure on health	1,003	0.18	0.38	0.00	0.00	1.00
Cut expenditure on education	1,003	0.12	0.32	0.00	0.00	1.00
Cut expenditure on home services	1,003	0.36	0.48	0.00	0.00	1.00
Cut expenditure on transportation	1,003	0.15	0.35	0.00	0.00	1.00
D - Borrower attributes						
Age	1,003	42.18	8.80	32.00	41.00	55.00
Female	1,003	0.39	0.49	0.00	0.00	1.00
Approval probability	981	0.59	0.07	0.49	0.60	0.67
Annual income	981	17,557.88	7,803.02	7,772.14	16,853.41	28,616.13
Financial literacy score (1-3)	1,003	1.75	0.81	1.00	2.00	3.00

Notes: This table displays summary statistics for our household survey.

Table A.4: Cost heterogeneity, market power and interest rate regulation

	(1)	(2)	(3)	(4)	(5)
		Alternative market power environments			
Outcome	Baseline	No bank heterogeneity $\tilde{\tau}_j = E[\tau_j]$	No incumbency advantage $\gamma\tilde{r}_{ij} = E[\gamma r_{ij}]$	Lower variance in cost shocks $\tilde{\sigma}_\omega = \frac{\sigma_\omega}{2}$	All (2), (3), (4)
Apply for loans (p.p)	-4.14	-5.63	-6.93	-6.24	-12.46
Unconstrained|Apply (p.p)	-16.83	-21.45	-32.49	-30.24	-77.74
Constrained|Apply (p.p)	12.89	15.91	23.00	25.61	30.29
Rejected|Apply (p.p)	3.94	5.53	9.48	4.62	47.44
Number of loans (%)	-23.78	-33.50	-45.59	-40.02	-99.65
Monthly payment ($)	4.33	8.31	4.99	-3.65	-95.38
Monthly payment on approved under full policy ($)	-2.42	-3.12	-4.45	-4.65	-10.36
Mark-up (p.p)	1.36	3.67	3.20	-0.57	-0.17
Mark-up on approved under full policy (p.p)	-0.63	-0.83	-1.26	-1.53	-5.40
Default probability (p.p)	-0.06	-0.07	-0.16	-0.29	3.63
Consumer surplus ($)	-81.78	-110.21	-139.13	-114.89	-205.42
Monthly profit ($)	5.16	10.62	7.98	-1.81	-10.22
Monthly profit on approved under full policy ($)	-2.41	-3.08	-4.40	-4.60	-9.91
Average monthly profit ($)	-2.41	-2.92	-3.51	-2.47	-2.48
Average welfare ($)	-84.19	-113.14	-142.64	-117.37	-207.90

Notes: This table displays results for simulated effects of moving from the baseline interest rate regulation in November 2013 to interest rate regulation in November 2015, when the policy change was fully in place. Column (1) displays results using the baseline estimates. Column (2) limits bank heterogeneity by removing fixed differences in cost τ_j, for which we impose the average of our estimates across banks. Column (3) removes banks incumbency advantages, by replacing γr_{ij} by the average of that term. Column (4) limits heterogeneity across banks by reducing the variance of idiosyncratic cost shocks σ_ω by half. Column (5) displays results when these three changes in the cost structure are implemented simultaneously. Note that the average cost in the market remains unchanged across all columns.

Table B.1: Policy roll-out groups, molecules and treatments

Group	Molecule	Treatment
1	Acenocumarol	Anticoagulant
1	Acido Valproico	Anticonvulsant
1	Atazanavir	HIV antiviral
1	Atorvastatina	Statin
2	Cefadroxilo	Antibiotic
2	Ciprofloxacino	Antibiotic
2	Clomifeno	Infertility
2	Clomipramina	Antidepressant
2	Clonazepam	Anxiety
2	Digoxina	Antiarrhythmic
2	Furosemida	Diuretic
2	Glibenclamida	Diabetes Mellitus
2	Isosorbida Dinitrato	Chest pain
2	Lamivudina	HIV antiviral
2	Losartan	High blood pressure
2	Metformina	Diabetes Mellitus
2	Metoclopramida	Gut motility stimulator
2	Metotrexato	Cancer
2	Micofenolato Mofetilo	Immunosuppressive
2	Nevirapina	Antiviral
2	Ritonavir	Antiviral
2	Tacrolimus	Immunosuppressive
2	Tenofovir	Antiviral
2	Verapamilo	High blood pressure
3	Alprazolam	Anxiety
3	Atenolol	High blood pressure
3	Darunavir	HIV antiviral
3	Diazepam	Anxiety
3	Enalapril	High Blood Pressure / Heart Failure
3	Espironolactona	Diuretic
3	Fluoxetina	Antidepressant
3	Hidroclorotiazida	Diuretic
3	Propranolol	High blood pressure
3	Salbutamol	Bronchodilator
3	Tamoxifeno	Cancer
4	Aciclovir	Antiviral
4	Acido Mefenamico	Analgesic / Antiinflamatory
4	Amiodarona	Antiarrhythmic
4	Amoxicilina+Clavulanico	Antibacterial
4	Azitromicina	Antibacterial
4	Cefuroxima	Antibiotic
4	Celecoxib	Analgesic / Antiinflamatory
4	Ciclobenzaprina	Muscle Relaxant
4	Claritromicina	Antibiotic
4	Clorpromazina	Antipsychotic
4	Clozapina	Mental disorders
4	Estradiol	Contraceptive
4	Famotidina	Gastric Ulcer and Reflux
4	Fluconazol	Anfifungal
4	Gemfibrozilo	High cholesterol
4	Lorazepam	Anxiety
4	Metilfenidato	Central nervous system stimulant
4	Metronidazol	Antibiotic / Antiparasitic
4	Midazolam	Sedative
4	Montelukast	Antiallergic / Anti-Asthmatic
4	Nitrofurantoina	Antibiotic
4	Olanzapina	Antipsychotic
4	Ondansetron	Antiemetic
4	Zidovudina+Lamivudina	HIV antiviral
4	Zopiclona	Insomnia
5	Amitriptilina	Nerve pain and antidepressant
5	Cetirizina	Antiallergic
5	Citalopram	Antidepressant
5	Desloratadina	Antiallergic
5	Diltiazem	High blood pressure / Angine
5	Donepecilo	Alzheimer
5	Doxazosina	Benign prostatic hyperplasia / High blood pressure
5	Escitalopram	Antidepressant
5	Fexofenadina	Antiallergic
5	Finasterida	Benign prostatic hyperplasia
5	Loratadina	Antiallergic
5	Mirtazapina	Antidepressant
5	Paroxetina	Antidepressant
5	Rivastigmina	Dementia
5	Sertralina	Antidepressant
5	Sildenafil	Erectile dysfunction
5	Terbinafina	Antifungal
5	Trimebutina	Antispasmodic
5	Valaciclovir	Antiviral
5	Zolpidem	Insomnia
6	Acido Ibandronico	Osteoporosis
6	Betahistina	Vertigo
6	Deflazacort	Corticotherapy
6	Hidroxicloroquina	Antimalarial
6	Levofloxacino	Antibiotic
6	Naratriptan	Migraine
6	Pramipexol	Parkinson's disease
6	Pregabalina	Anticonvulsant / Neuralgia
6	Quetiapina	Mental disorders
6	Telmisartan	High blood pressure
7	Aripiprazol	Antipsychotic
7	Atomoxetina	Antidepressant
7	Carvedilol	High Blood Pressure / Heart Failure
7	Cilostazol	Vasodilator
7	Clopidogrel	Blood thinner
7	Haloperidol	Mental disorders
7	Isotretinoina	Acne
7	Lamotrigina	Anticonvulsant / Mood stabilizer
7	Meloxicam	Analgesic / Antiinflamatory
7	Moxifloxacino	Antibiotic
7	Nebivolol	High blood pressure
7	Olmesartan	High blood pressure
7	Risperidona	Mental disorders
7	Topiramato	Anticonvulsant
7	Valsartan	High blood pressure
8	Alendronato	Osteoporosis
8	Bromazepam	Anxiety
8	Candesartan	Antihypertensive
8	Cinarizina	Antihistamine
8	Flunarizina	Migraine
8	Leflunomida	Arthritis
8	Levetiracetam	Anticonvulsant / Mood stabilizer
8	Levocetirizina	Antiallergic
8	Levonorgestrel	Contraceptive
8	Lovastatina	Statin
8	Medroxiprogesterona	Hormone Imbalance
8	Nifedipino	Antihypertensive
8	Nimodipino	Antihypertensive
8	Nitrendipino	High blood pressure / Angine
8	Sulpirida	Antipsychotic
8	Tibolona	Hormone replacement therapy
9	Acetazolamida	Diuretic
9	Captopril	High Blood Pressure / Heart Failure
9	Colchicina	Antiinfalamtory
9	Griseofulvina	Antifungal
9	Imipramina	Nerve pain and antidepressant
9	Metildopa	High blood pressure
9	Nistatina	Antifungal
9	Tetraciclina	Antibiotic
9	Tinidazol	Anti-parasite and antibiotics
9	Tioridazina	Antipsychotic

Notes: This Table displays the list of molecules included in the sample used for the analysis in the paper, including its group withing the policy roll-out and the treatment of each of them. The 9 policy roll-out groups are the same as in Table 2.1.

Table B.2: Determinants of Assignment of Bioequivalence Decrees to Molecules

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Ordered logit for Policy Assignment Groups						
Branded generic market share	-1.585***	-1.587***	-1.996***	-2.515***	-2.236**	-2.310***	-2.076**
	(0.615)	(0.615)	(0.705)	(0.841)	(0.874)	(0.884)	(0.907)
Unbranded generic market share	-0.844	-0.896	-1.808**	-2.037**	-1.518	-1.509	-1.265
	(0.544)	(0.549)	(0.806)	(0.921)	(0.975)	(0.978)	(1.023)
Δ% Branded generic market share		0.052	0.052	0.065	0.078	-0.451	-0.424
		(0.097)	(0.098)	(0.099)	(0.100)	(0.394)	(0.412)
Δ% Unbranded generic market share		-0.181	-0.322	-0.349	-0.360	-0.332	-0.288
		(0.305)	(0.321)	(0.319)	(0.322)	(0.317)	(0.320)
Any branded generic			0.548	0.304	0.332	0.319	0.406
			(0.553)	(0.723)	(0.728)	(0.728)	(0.735)
Any unbranded generic			0.745	0.662	0.699	0.563	0.706
			(0.534)	(0.637)	(0.640)	(0.648)	(0.667)
log(Number of drugs)				0.382	0.497	0.483	0.461
				(0.369)	(0.385)	(0.384)	(0.393)
log(Number of labs)				0.005	-0.323	-0.301	-0.234
				(0.559)	(0.604)	(0.599)	(0.615)
HHI of drug types				0.925	0.661	0.541	0.753
				(1.034)	(1.046)	(1.062)	(1.085)
log(Market revenue)					0.260	0.237	0.225
					(0.240)	(0.239)	(0.240)
log(Average price)					0.121	0.090	0.073
					(0.081)	(0.083)	(0.085)
Δ% Average price						1.081	1.033
						(0.785)	(0.817)
Share imported							0.743
							(0.783)
Chronic							0.149
							(0.338)
Observations	131	131	131	131	131	131	131
Pseudo R^2	0.0123	0.0135	0.0198	0.0248	0.0303	0.0337	0.0359

Notes: This table displays results from ordered logit models for the policy groups defined in Table 2.1. The analysis is implemented using the cross section of molecules in the sample for 2010, before the first decree for the first group. Percentage changes in variable measure the change between 2011 and 2010 relative to the baseline level of the variable in 2010. A caveat with this definition is that the first decrees occur in 2011. However, lack of data for 2009 limit the extent to which we can compute growth rates for these variables using data from before the first policy events. Positive coefficients indicate that molecules with a higher value in that variable had a higher likelihood of being assigned to an earlier policy group. Standard errors in parentheses. *p<0.10, **p<0.05, ***p<0.01.

Table B.3: Heterogeneity in Hazard Model for Bioequivalence and Exit

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	Panel A: Bioequivalence approval hazard					*Panel B: Exit hazard*				
After first deadline=1	2.38***	2.77***	1.69**	0.21	0.94	0.40	0.66	-0.05	0.19	-0.18
	(0.47)	(0.57)	(0.85)	(1.27)	(1.28)	(0.38)	(0.45)	(0.36)	(0.46)	(0.52)
× Above median price, 2010		-0.87*			-1.03**		-0.41			0.07
		(0.45)			(0.44)		(0.39)			(0.43)
× log(Sales, 2010)			0.12		0.00			0.20**		0.11
			(0.12)		(0.11)			(0.09)		(0.12)
× log(Revenue, 2010)				0.15*	0.13				0.04	0.04
				(0.09)	(0.08)				(0.03)	(0.05)
Above median price, 2010		0.46			0.67*		0.34			-0.17
		(0.43)			(0.41)		(0.39)			(0.43)
log(Sales, 2010)			0.08		0.14			-0.42***		-0.29**
			(0.11)		(0.09)			(0.09)		(0.12)
log(Revenue, 2010)				-0.01	-0.05				-0.14***	-0.08*
				(0.07)	(0.06)				(0.03)	(0.04)
Reference						-1.20***	-1.20***	-1.18***	-1.03***	-1.10***
						(0.34)	(0.34)	(0.32)	(0.32)	(0.31)
Imported	0.38**	0.37**	0.39**	0.37**	0.38**	0.60***	0.61***	0.54***	0.56***	0.55***
	(0.17)	(0.17)	(0.16)	(0.17)	(0.16)	(0.19)	(0.19)	(0.16)	(0.17)	(0.16)
log(Market revenue)	0.67***	0.67***	0.49***	0.63***	0.53***	-0.32***	-0.32***	-0.07	-0.22**	-0.07
	(0.15)	(0.15)	(0.17)	(0.15)	(0.16)	(0.10)	(0.10)	(0.11)	(0.10)	(0.10)
log(Number of branded)	-0.01	-0.00	0.05	0.01	0.05	0.01	0.01	-0.12	-0.03	-0.11
	(0.27)	(0.27)	(0.22)	(0.26)	(0.22)	(0.15)	(0.15)	(0.14)	(0.14)	(0.14)
log(Number of unbranded)	-0.29	-0.26	-0.37**	-0.28	-0.32*	0.11	0.11	0.09	0.08	0.09
	(0.18)	(0.18)	(0.18)	(0.18)	(0.18)	(0.17)	(0.17)	(0.14)	(0.15)	(0.14)
Time FE	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Observations	51,114	51,114	51,114	51,114	51,114	79,657	79,657	79,657	79,657	79,657
ln L	-1,209	-1,203	-1,181	-1,191	-1,173	-891	-890	-853	-864	-848

Notes: This table displays results from hazard models in equation (2.1) for bioequivalence approval and market exit. Results in this table highlight heterogeneity in the relationship between quality regulation and drug bioequivalence approval or exit along baseline drug characteristics. Estimation is implemented through maximum likelihood. All specifications include time fixed effects. Standard errors in parentheses clustered at molecule level. *p<0.10, **p<0.05, ***p<0.01.

Table B.4: Effects of Quality Regulation on Market Structure: Number of Drugs

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Dep. var.: $\sinh^{-1}$(Number of Drugs)							
	All	Innovator	Branded generics			Unbranded generics		
			All	BE	Non-BE	All	BE	Non-BE
Panel A: Average effects								
Share of market under regulation	-0.32***	-0.12	-0.35***	0.74***	-0.49***	-0.36***	0.79***	-0.49***
	(0.09)	(0.09)	(0.06)	(0.20)	(0.08)	(0.09)	(0.14)	(0.10)
R^2	0.95	0.94	0.96	0.71	0.95	0.92	0.64	0.92
Panel B: Heterogeneity by baseline market size								
Share of market under regulation × Low revenue	-0.48***	-0.27***	-0.49***	0.26	-0.55***	-0.52***	0.42***	-0.51***
	(0.10)	(0.10)	(0.09)	(0.21)	(0.11)	(0.11)	(0.15)	(0.12)
Share of market under regulation × High revenue	-0.18**	-0.00	-0.23***	1.15***	-0.44***	-0.22**	1.10***	-0.47***
	(0.09)	(0.09)	(0.08)	(0.23)	(0.10)	(0.11)	(0.18)	(0.11)
R^2	0.95	0.95	0.96	0.73	0.95	0.92	0.66	0.92
Pre-regulation average	31.25	3.43	17.36	0.10	17.26	10.45	0.01	10.45
Observations	12,576	12,576	12,576	12,576	12,576	12,576	12,576	12,576
Market FE	Y	Y	Y	Y	Y	Y	Y	Y
Month FE	Y	Y	Y	Y	Y	Y	Y	Y

Notes: Each column in this Table is a regression of the inverse hyperbolic sine of number of drugs in a segment on the policy roll-out variable constructed using the first decree deadline. Panel B provides results by baseline revenue. Markets are classified as having a low or high revenue according to the average level of the variable in 2010 relative to the median across markets in that year. Clustered standard errors in parentheses. ***p<0.01, **p<0.05, *p<0.1.

Table B.5: Effects of Quality Regulation on Market Structure: Number of Laboratories

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Dep. var.: $\sinh^{-1}$(Number of Laboratories)							
	All	Innovator	Branded generics			Unbranded generics		
			All	BE	Non-BE	All	BE	Non-BE
Panel A: Average effects								
Share of market under regulation	-0.18***	-0.01	-0.17***	0.67***	-0.23***	-0.23***	0.71***	-0.31***
	(0.05)	(0.02)	(0.04)	(0.16)	(0.04)	(0.08)	(0.12)	(0.08)
R^2	0.93	0.96	0.96	0.71	0.96	0.92	0.65	0.91
Panel B: Heterogeneity by baseline market size								
Share of market under regulation × Low revenue	-0.30***	-0.07*	-0.29***	0.28	-0.30***	-0.37***	0.41***	-0.32***
	(0.06)	(0.04)	(0.06)	(0.17)	(0.06)	(0.09)	(0.14)	(0.10)
Share of market under regulation × High revenue	-0.08	0.04**	-0.07	1.00***	-0.16***	-0.12	0.97***	-0.30***
	(0.05)	(0.02)	(0.05)	(0.19)	(0.04)	(0.09)	(0.15)	(0.09)
R^2	0.93	0.96	0.96	0.73	0.96	0.92	0.67	0.91
Pre-regulation average	24.97	3.37	17.31	0.10	17.21	4.29	0.01	4.29
Observations	12,576	12,576	12,576	12,576	12,576	12,576	12,576	12,576
Market FE	Y	Y	Y	Y	Y	Y	Y	Y
Month FE	Y	Y	Y	Y	Y	Y	Y	Y

Notes: Each column in this Table is a regression of the inverse hyperbolic sine of number of laboratories in a segment on the policy roll-out variable constructed using the first decree deadline. Panels B provides results by baseline revenue. Markets are classified as having a low or high revenue according to the average level of the variable in 2010 relative to the median across markets in that year. Clustered standard errors in parentheses. ***p<0.01, **p<0.05, *p<0.1.

Table B.6: Effects of Quality Regulation on Market Structure: Number of Drugs per Laboratory

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Dep. var.: log(1 + Number of drugs per laboratory)							
	All	Innovator	Branded generic			Unbranded generic		
			All	BE	Non-BE	All	BE	Non-BE
Panel A: Average effects								
Share of market under regulation	-0.10	-0.10	-0.13***	0.24***	-0.18***	-0.14**	0.37***	-0.17***
	(0.06)	(0.07)	(0.04)	(0.09)	(0.05)	(0.06)	(0.08)	(0.06)
R^2	0.93	0.94	0.91	0.67	0.90	0.80	0.60	0.78
Panel B: Heterogeneity by baseline market size								
Share of market under regulation × Low revenue	-0.12*	-0.20**	-0.15**	0.09	-0.18***	-0.16**	0.26***	-0.18**
	(0.06)	(0.08)	(0.06)	(0.09)	(0.06)	(0.07)	(0.08)	(0.07)
Share of market under regulation × High revenue	-0.08	-0.01	-0.11**	0.35***	-0.18***	-0.12*	0.47***	-0.17***
	(0.07)	(0.07)	(0.05)	(0.10)	(0.05)	(0.06)	(0.09)	(0.06)
R^2	0.93	0.94	0.91	0.68	0.90	0.80	0.61	0.78
Pre-regulation average	2.79	3.14	2.11	0.10	2.10	1.70	0.00	1.70
Observations	12,576	12,576	12,576	12,576	12,576	12,576	12,576	12,576
Market FE	Y	Y	Y	Y	Y	Y	Y	Y
Month FE	Y	Y	Y	Y	Y	Y	Y	Y

Notes: Each column in this table is a regression of the log number of drugs per laboratory in a segment on the policy roll-out variable constructed using the first decree deadline. Panels B provides results by baseline revenue. Markets are classified as having a low or high revenue according to the average level of the variable in 2010 relative to the median across markets in that year. Clustered standard errors in parentheses. ***p<0.01, **p<0.05, *p<0.1.

Table B.7: Summary Statistics from Consumer Survey Data

Variable	N	Mean	SD	p10	p50	p90
Perceived quality of innovator drug (1-7)	361	6.32	1.01	5.00	7.00	7.00
Perceived quality of bioequivalent branded drug (1-7)	378	5.69	1.31	4.00	6.00	7.00
Perceived quality of bioequivalent unbranded drug (1-7)	386	5.63	1.28	4.00	6.00	7.00
Perceived quality of non-bioequivalent unbranded drug (1-7)	381	4.68	1.65	3.00	5.00	7.00
Perceived price of bioequivalent branded drug (CLP 1,000s)	398	25.37	14.13	6.00	25.00	45.00
Perceived price of bioequivalent unbranded drug (CLP 1,000s)	401	15.69	10.98	3.00	15.00	30.00
Perceived price of non-bioequivalent unbranded drug (CLP 1,000s)	399	12.60	9.97	2.00	10.00	25.00
Recognizes bioequivalent drug label	401	0.84	0.37	0.00	1.00	1.00
Understanding about bioequivalence (1-5)	401	2.91	1.47	1.00	3.00	5.00
=1 if physicians specify brand in prescriptions	299	0.65	0.48	0.00	1.00	1.00
=1 if always purchases physician recommendation	310	0.15	0.36	0.00	0.00	1.00
=1 if sometimes deviate from physician recommendation	310	0.52	0.50	0.00	1.00	1.00
=1 if always chooses cheapest available drug	310	0.34	0.47	0.00	0.00	1.00
Purchases innovator drugs	338	0.41	0.49	0.00	0.00	1.00
Purchases bioequivalent branded drugs	338	0.20	0.40	0.00	0.00	1.00
Purchases bioequivalent unbranded drugs	338	0.28	0.45	0.00	0.00	1.00
Purchases non-bioequivalent unbranded drugs	338	0.11	0.31	0.00	0.00	1.00
Chronic illness by household member	401	0.58	0.49	0.00	1.00	1.00
Atorvastatin consumption by household member	401	0.34	0.48	0.00	0.00	1.00

Notes: This table displays summary statistics from our consumer survey. The total number of surveys is $N = 401$. Whenever the number of observations is smaller, is due to the consumer not answering the question, except for the case of questions regarding physicians' prescription behavior, which were added to the survey with a lag and are therefore not available for a around a fourth of the sample.

Table C.1: Reduced Form Estimates of Vertical Integration on Payments, Alternative Specification

	(1)	(2)	(3)	(4)	(5)
	Panel A - OLS estimates on Total bill				
Vertically integrated	-0.313***	-0.035	-0.002	-0.073***	-0.080***
	(0.107)	(0.028)	(0.022)	(0.020)	(0.019)
Observations	545,718	545,718	545,718	545,718	545,716
R-squared	0.026	0.120	0.398	0.402	0.419
	Panel B - OLS estimates on Patient copayment				
Vertically integrated	-0.368***	-0.100***	-0.091**	-0.214***	-0.217***
	(0.098)	(0.034)	(0.035)	(0.031)	(0.030)
Observations	545,718	545,718	545,718	545,718	545,716
R-squared	0.041	0.177	0.302	0.418	0.431
	Panel C - OLS estimates on Insurer coverage				
Vertically integrated	-0.160*	0.021	0.051***	0.051**	0.045*
	(0.082)	(0.024)	(0.016)	(0.023)	(0.023)
Observations	545,718	545,718	545,718	545,718	545,716
R-squared	0.008	0.057	0.311	0.362	0.375
Hospital FEs	N	Y	Y	Y	Y
Diagnosis FEs	N	N	Y	Y	Y
Diagnosis public prices	N	N	Y	Y	Y
Insurer controls	N	N	N	Y	Y
Patient controls	N	N	N	N	Y

Notes: This table shows results from estimating equation (3.1) using the inverse hyperbolic sine transformation ($\log(y + \sqrt{y^2 + 1})$) of total bill (Panel A), patient copayment (Panel B), and insurer coverage (Panel C) as dependent variables. Each column includes a different set of control variables. Diagnosis fixed effects are based on ICD10 chapters, and diagnosis public system prices are the prices of the same admissions in public hospitals. Insurer controls include insurer fixed effect, plan premium, coinsurance rate for inpatient and outpatient admissions, and dummies for whether the plan has a coverage cap and a preferential hospital. Patient controls include gender, age, income, number of dependents, an indicator for independent worker and fixed effects by county of residence. The sample considers the admissions in the 12 main private hospitals. Standard errors in parentheses are clustered by insurer-hospital combination. P-values notation: *** p<0.01, ** p<0.05, * p<0.1.

Table C.2: Price prediction error distribution

	(1)	(2)	(3)	(4)
Statistic	First stage	Second stage		
Min	-13.856	-14.374	-14.373	-14.540
Median	0.037	0.093	0.082	0.070
Mean	0.000	0.000	-0.000	0.000
Max	6.784	6.057	6.346	6.529
MSE	0.872	1.215	1.131	1.098
Diagnosis FE	-	N	Y	N
Diagnosis-age-gender FE	-	N	N	Y

Notes: This table displays the distribution of the in-sample prediction error of our negotiated price estimation routine. Column (1) shows the prediction of our method using public system prices, which is used to estimate negotiated prices. Column (2) is the our final estimate, which recovers the resource intensity weights. Columns (3) and (4) show results using fixed effects for diagnosis and for diagnosis-age-gender interactions, respectively. The error is defined as observed minus predicted.

Table C.3: Diagnosis cost intensity weights by demographic group, in logs

	Panel A: Females				Panel B: Males			
	0-25	26-45	46-60	60+	0-25	26-45	46-60	60+
Diagnosis								
I - Infections and parasites	0.467	0.466	0.480	0.472	0.466	0.508	0.498	0.505
	[0.45, 0.48]	[0.44, 0.49]	[0.45, 0.51]	[0.44, 0.51]	[0.45, 0.48]	[0.49, 0.53]	[0.47, 0.53]	[0.47, 0.54]
II - Neoplasms	0.597	0.599	0.605	0.550	0.548	0.562	0.552	0.553
	[0.59, 0.61]	[0.59, 0.60]	[0.60, 0.61]	[0.55, 0.55]	[0.54, 0.55]	[0.56, 0.57]	[0.55, 0.56]	[0.55, 0.56]
III - Blood diseases	0.373	0.374	0.371	0.403	0.478	0.392	0.459	0.580
	[0.33, 0.42]	[0.31, 0.45]	[0.32, 0.43]	[0.32, 0.50]	[0.40, 0.57]	[0.30, 0.52]	[0.31, 0.68]	[0.45, 0.74]
IV - Endocrine	0.662	0.813	0.786	0.692	0.563	0.670	0.648	0.549
	[0.64, 0.69]	[0.80, 0.83]	[0.77, 0.81]	[0.66, 0.73]	[0.54, 0.58]	[0.66, 0.68]	[0.63, 0.67]	[0.52, 0.58]
VI - Nervous system	0.516	0.535	0.599	0.572	0.543	0.512	0.519	0.531
	[0.50, 0.53]	[0.52, 0.55]	[0.58, 0.61]	[0.55, 0.59]	[0.53, 0.56]	[0.50, 0.52]	[0.51, 0.53]	[0.51, 0.55]
VII - Ocular diseases	0.818	0.844	0.889	0.935	0.858	0.851	0.887	0.932
	[0.79, 0.84]	[0.83, 0.86]	[0.87, 0.91]	[0.92, 0.95]	[0.83, 0.88]	[0.84, 0.86]	[0.87, 0.91]	[0.91, 0.95]
VIII - Ear diseases	0.783	0.692	0.687	0.609	0.794	0.778	0.814	0.607
	[0.71, 0.86]	[0.61, 0.79]	[0.61, 0.78]	[0.51, 0.73]	[0.74, 0.86]	[0.68, 0.90]	[0.71, 0.94]	[0.49, 0.76]
IX - Circulatory	0.573	0.709	0.726	0.690	0.619	0.683	0.682	0.709
	[0.55, 0.59]	[0.70, 0.72]	[0.71, 0.74]	[0.68, 0.70]	[0.60, 0.64]	[0.67, 0.69]	[0.67, 0.69]	[0.70, 0.72]
X - Respiratory	0.606	0.655	0.595	0.520	0.599	0.714	0.649	0.534
	[0.60, 0.61]	[0.64, 0.66]	[0.58, 0.61]	[0.51, 0.53]	[0.59, 0.60]	[0.70, 0.72]	[0.63, 0.66]	[0.52, 0.55]
XI - Digestive	0.629	0.647	0.643	0.622	0.663	0.660	0.652	0.639
	[0.62, 0.64]	[0.64, 0.65]	[0.64, 0.65]	[0.61, 0.63]	[0.66, 0.67]	[0.66, 0.67]	[0.65, 0.66]	[0.63, 0.65]
XII - Skin diseases	0.595	0.575	0.569	0.523	0.580	0.598	0.531	0.537
	[0.56, 0.63]	[0.55, 0.61]	[0.53, 0.61]	[0.48, 0.56]	[0.55, 0.61]	[0.57, 0.63]	[0.50, 0.57]	[0.49, 0.58]
XIII - Musculoskeletal	0.764	0.679	0.751	0.778	0.782	0.768	0.768	0.823
	[0.75, 0.78]	[0.67, 0.69]	[0.74, 0.76]	[0.77, 0.79]	[0.77, 0.80]	[0.76, 0.78]	[0.76, 0.78]	[0.81, 0.84]
XIV - Genitourinary	0.609	0.701	0.749	0.698	0.693	0.765	0.778	0.817
	[0.60, 0.62]	[0.69, 0.71]	[0.74, 0.76]	[0.68, 0.71]	[0.69, 0.70]	[0.76, 0.77]	[0.77, 0.79]	[0.80, 0.83]
XV - Pregnancy	0.587	0.778	0.608	0.532	0.420	0.576	0.544	0.410
	[0.58, 0.59]	[0.77, 0.78]	[0.59, 0.63]	[0.50, 0.56]	[0.41, 0.43]	[0.57, 0.58]	[0.52, 0.57]	[0.38, 0.45]
XVI - Perninatal	0.404	0.293	0.304	0.154	0.407	0.376	0.393	0.330
	[0.39, 0.41]	[0.26, 0.33]	[0.19, 0.48]	[0.05, 0.51]	[0.40, 0.42]	[0.23, 0.61]	[0.26, 0.59]	[0.23, 0.47]
XVII - Congenital malformation	0.860	0.799	0.839	0.775	0.918	0.772	0.689	0.743
	[0.82, 0.90]	[0.75, 0.85]	[0.77, 0.92]	[0.68, 0.88]	[0.89, 0.95]	[0.72, 0.83]	[0.63, 0.76]	[0.65, 0.85]

Notes: This table displays diagnosis cost intensity weights by gender and age group. These cost weights are used for constructing hospital prices. Number in braces correspond to 90% confidence intervals, estimate via 100 bootstrap draws.

Table C.4: Diagnosis probabilities by demographic group

	Age group													
Diagnosis	0-2	3-5	6-10	11-15	16-20	21-25	26-30	31-35	36-40	41-45	46-50	51-55	56-60	61+
Panel A: Females														
I - Infections and parasites	0.009	0.004	0.002	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.002
II - Neoplasms	0.004	0.005	0.004	0.004	0.005	0.004	0.006	0.009	0.014	0.021	0.029	0.034	0.041	0.058
III - Blood diseases	0.000	0.001	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.001
IV - Endocrine	0.002	0.000	0.000	0.001	0.002	0.002	0.003	0.004	0.005	0.004	0.004	0.004	0.004	0.003
VI - Nervous system	0.003	0.001	0.001	0.001	0.002	0.001	0.002	0.002	0.003	0.003	0.004	0.005	0.006	0.006
VII - Ocular diseases	0.001	0.001	0.001	0.001	0.002	0.005	0.008	0.009	0.007	0.005	0.007	0.010	0.014	0.025
VIII - Ear diseases	0.001	0.001	0.001	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.001	0.001
IX - Circulatory	0.002	0.001	0.000	0.000	0.001	0.001	0.002	0.003	0.003	0.004	0.005	0.007	0.009	0.019
X - Respiratory	0.037	0.032	0.013	0.005	0.007	0.004	0.004	0.004	0.003	0.003	0.003	0.003	0.004	0.010
XI - Digestive	0.012	0.007	0.006	0.008	0.010	0.008	0.010	0.012	0.012	0.011	0.012	0.015	0.018	0.023
XII - Skin diseases	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.002
XIII - Musculoskeletal	0.001	0.001	0.001	0.003	0.004	0.003	0.003	0.005	0.006	0.008	0.010	0.014	0.018	0.022
XIV - Genitourinary	0.006	0.003	0.002	0.001	0.004	0.004	0.005	0.009	0.011	0.012	0.011	0.010	0.010	0.014
XV - Pregnancy	0.030	0.000	0.000	0.000	0.009	0.018	0.044	0.078	0.056	0.016	0.002	0.001	0.001	0.001
XVI - Perinatal	0.052	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
XVII - Congenital malformation	0.006	0.002	0.001	0.001	0.001	0.001	0.000	0.001	0.001	0.001	0.000	0.001	0.001	0.001
Panel B: Males														
I - Infections and parasites	0.010	0.005	0.002	0.001	0.001	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.005
II - Neoplasms	0.004	0.006	0.005	0.004	0.006	0.007	0.008	0.011	0.013	0.018	0.026	0.037	0.064	0.134
III - Blood diseases	0.001	0.001	0.001	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.001	0.001	0.001	0.002
IV - Endocrine	0.003	0.000	0.000	0.000	0.001	0.001	0.002	0.003	0.004	0.005	0.005	0.005	0.005	0.006
VI - Nervous system	0.003	0.002	0.002	0.001	0.002	0.002	0.003	0.006	0.008	0.009	0.012	0.014	0.014	0.015
VII - Ocular diseases	0.001	0.001	0.001	0.001	0.002	0.009	0.016	0.019	0.016	0.013	0.014	0.016	0.018	0.037
VIII - Ear diseases	0.001	0.002	0.001	0.000	0.000	0.000	0.000	0.000	0.001	0.001	0.001	0.001	0.001	0.001
IX - Circulatory	0.002	0.001	0.001	0.001	0.002	0.003	0.003	0.005	0.006	0.010	0.015	0.022	0.029	0.060
X - Respiratory	0.046	0.039	0.016	0.005	0.007	0.007	0.007	0.008	0.008	0.007	0.007	0.008	0.009	0.022
XI - Digestive	0.016	0.009	0.008	0.009	0.010	0.012	0.014	0.018	0.022	0.025	0.030	0.035	0.040	0.059
XII - Skin diseases	0.001	0.001	0.001	0.001	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.002	0.004
XIII - Musculoskeletal	0.001	0.002	0.001	0.002	0.006	0.008	0.010	0.014	0.018	0.021	0.024	0.026	0.027	0.029
XIV - Genitourinary	0.011	0.020	0.011	0.005	0.006	0.006	0.008	0.009	0.012	0.015	0.017	0.018	0.024	0.043
XV - Pregnancy	0.033	0.000	0.000	0.000	0.000	0.000	0.000	0.001	0.001	0.000	0.000	0.000	0.001	0.001
XVI - Perninatal	0.072	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
XVII - Congenital malformation	0.009	0.004	0.002	0.002	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.002

Notes: This table displays diagnosis probabilities by gender and age group. These probabilities are used for calculating the expected utility from health care services from insurance plans.

Table C.5: Financial Data on Aggregated Revenues and Costs for Hospitals and Insurers (2013-2016)

	(1)	(2)	(3)	(4)
	Revenues (1)	Direct Costs (2)	Profits (1)-(2)	Mark-up Rate ((1)-(2))/(1)
Panel A - Hospitals				
h_2	249	187	61	25%
h_3	310	252	58	19%
h_4	744	593	151	20%
h_5	726	507	220	30%
h_6	1,106	875	232	21%
h_7	1,113	819	294	26%
h_8	236	183	53	23%
h_{11}	316	240	76	24%
Holding 1	2,174	1,652	522	24%
Holding 2	795	622	173	22%
Total Hospital Industry	4,802	3,656	1,146	24%
	Revenues (1)	Direct Costs (2)	Profits (1)-(2)	Mark-up Rate ((1)-(2))/(1)
Panel B - Insurers				
m_1	2,531	2,218	313	12%
m_2	2,427	2,124	303	12%
m_3	2,161	1,865	296	14%
m_4	2,765	2,412	353	13%
m_5	871	770	101	12%
m_6	774	665	109	14%
Holding 1	3,305	2,883	422	13%
Holding 2	2,161	1,865	296	14%
Total Insurance Industry	11,528	10,054	1,474	13%

Notes: Panel A is based on the Annual Financial Report of each hospital for years 2013-2016. Public financial data are unavailable for the following unintegrated hospitals: h_1, h_9, h_{10}, and h_{12}. Holding 1 includes h_7, h_4 and h_{11} (data on h_{11} unavailable for 2013). Holding 2 includes h_2, h_3, and h_8. Panel B is based on the Annual Financial Report of each insurer for years 2013-2016. Data on m_5 unavailable for 2015-2016. Amounts expressed in millions of dollars, using the exchange rate of Dec 31, 2014. Direct costs do not consider investments or financial costs.

Table C.6: First Stage of GMM Instruments as Predictor of Negotiated Prices

	(1)	(2)	(3)	(4)	(5)	(6)
	$\hat{\beta}$	S.E.	z	$P > \|z\|$	[0.025	0.975]
WTP hospital	-0.0596	0.051	-1.164	0.244	-0.160	0.041
WTP system	0.0631	0.052	1.207	0.227	-0.039	0.166
WTP rivals	-0.2873	0.058	-4.922	0.000	-0.402	-0.173
WTP system rivals	0.3046	0.060	5.110	0.000	0.188	0.421
Observations	288					
R-squared	0.854					
F-statistic	437.0					

Notes: This table shows the first stage estimates of regressing negotiated prices on our instruments. The four instruments are willingness to pay metrics built using public system prices.