Sick and Locked-In

A Study on Health Status and Consumer Inertia in Health Insurance

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Declaration

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Abstract

Recent evidence documents substantial quality differences across health plans within highly regulated insurance programs, raising the question of what prevents consumers from switching to better options. This thesis provides novel evidence of a key barrier: health risk driving consumer inertia. Using detailed administrative data from Colombia's Régimen Contributivo, a mandated insurance program covering over 20 million enrollees, it examines how health risk affects switching behavior when plans offer identical contracts but provide different effective access to medical care in practice.

The setting is ideal for studying the relationship between health risk and inertia as it features standardized contracts with virtually unrestricted switching yet exhibits substantial quality differences, particularly during the collapse of two large insurers covering 30% of individuals in urban markets.

The study focuses on mean care provision—the amount of medical care plans provide to enrollees with similar needs—and documents substantial heterogeneity across plans in this important dimension of plan quality. Three complementary approaches establish that health risk increases choice persistence. First, a predictive risk score based on diagnostic and demographic data shows higher ex-ante risk correlates with lower switching rates. Second, an event-study design exploiting cancer diagnoses reveals that illness causally reduces switching rates by up to 63%. Third, a structural demand model shows both age and health risk increase switching costs, with the highest-risk individuals exhibiting near-complete inertia.

Unlike standard adverse selection theory, where high-risk individuals seek better coverage, this study uncovers a different phenomenon: high-risk individuals remain in deteriorating plans even when alternatives offer better effective coverage. This creates a death spiral where healthier enrollees exit first, leaving behind a riskier pool that becomes increasingly unprofitable under the program's coarse risk adjustment, highlighting critical flaws in risk-adjustment mechanisms with important implications for managed compe-

tition in health insurance markets.

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Para Anamaría y María Emilia.

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Chapter I

Introduction

1 Motivation, Research Question, and Main Results

Over the past several decades, health systems around the world have shifted toward models in which private insurers or health plans play a central role in managing access to publicly financed medical care. In these regulated markets, insurers are no longer passive payers of claims but active organizers of care delivery, responsible for determining which providers to contract with, what care to authorize, and how to manage utilization. This transformation has made the quality of care contingent not just on the healthcare system as a whole, but on the specific plan to which an individual is enrolled. Recent evidence has now emerged documenting substantial, causal differences in health outcomes and care utilization patterns across health plans that compete within the same regulatory environment—ranging from mortality differences (Abaluck et al., 2021) to variation in medical spending and care utilization (Geruso et al., 2023). That these differences arise despite consumers' ability to choose their plan raises important questions about *who* fails to respond to these quality differences, and *why*.

This dissertation examines a specific barrier to quality-responsive choice: the role of health risk in driving consumer inertia. It asks: When plans differ substantially in quality, does medical care need exacerbate or ameliorate consumer inertia? To answer this question, the study focuses on a key dimension of plan quality: the *effective* access to medical care they provide to their enrollees. Studying inertia, understood as the tendency of indi-

viduals to persist in their choices without evaluating the available alternatives (Spiegler, 2011), is motivated by its pervasiveness in health insurance markets, with prior work showing that it can impose substantial financial losses on individuals (e.g., Handel, 2013; Polyakova, 2016). Its interaction with health status remains understudied but is important, as the value forgone by not switching to a plan that provides more access to care is likely to be commensurate with medical care need. Yet the effect of health risk on inertia is theoretically ambiguous. On the one hand, illness may increase attentiveness, motivating individuals to seek better options. On the other, it may exacerbate the choice frictions behind inertia by raising the cognitive burden of decision-making or increasing the costs of navigating switching processes.

This study exploits the unique features of Colombia's Régimen Contributivo, a large mandated health insurance program covering over 20 million enrollees. Three features make this setting ideal for studying the relationship between health risk and consumer inertia. First, there is compelling evidence of substantial quality differences across plans, particularly during the decline of two large insurers—Cafesalud and Coomeva—that were eventually terminated by the government for poor medical care provision and financial performance. Together, these plans accounted for approximately 30% of enrollment in urban markets at the beginning of 2016, making their decline a significant market event.[1] As Figure I.1 shows, their collapse was marked by declining market shares and sharp increases in patient complaints and switching rates, which suggests a persistent deterioration in quality that consumers could potentially respond to (indeed, many of them did).

Second, the program's regulatory framework creates ideal conditions for studying consumers' responsiveness to plan quality. All plans offer a single standardized contract with identical benefits and financial terms, forcing competition into dimensions that directly affect care access, such as provider networks and utilization management prac-

[1] As explained in Section 3.1, the history of *Cafesalud* is closely linked to that of a third terminated plan, *Saludcoop*, which was closed in November 2015. All enrollees from *Saludcoop* were reassigned to *Cafesalud*, which was later sold and rebranded as *Medimás* in August 2017. For simplicity, some parts of the analysis combine these three plans into one, and we refer to it as *SCM*.

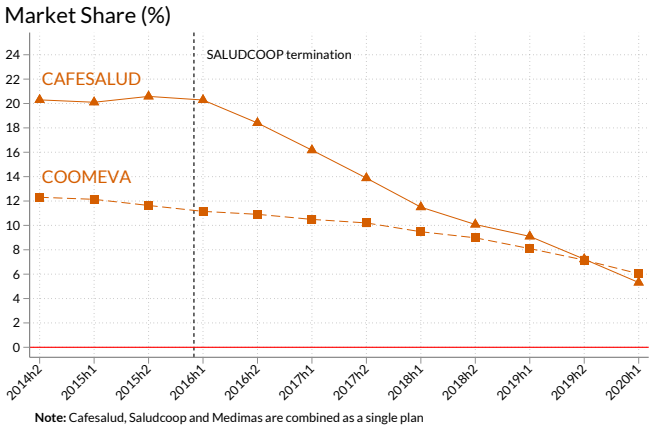
tices. Moreover, switching rules are highly permissive—individuals can change plans monthly after a 12-month enrollment period, without the open enrollment windows or health-based restrictions common in other settings.[2]. Third, the program's large scale provides statistical power to examine relationships involving rare health events like cancer diagnoses. Overall, these features create a setting where inertial consumers could forego substantial value by remaining enrolled in declining plans, and we can study the role health status plays in driving this phenomenon.

The main finding is that health risk increases consumer inertia, creating a "lock-in" effect that makes sicker individuals less likely to switch out of their plans, even when alternative plans would provide them with substantially more care. This dynamic led to a concentration of high-risk enrollees in declining plans, affecting their profitability and potentially contributing to their decline in this period.

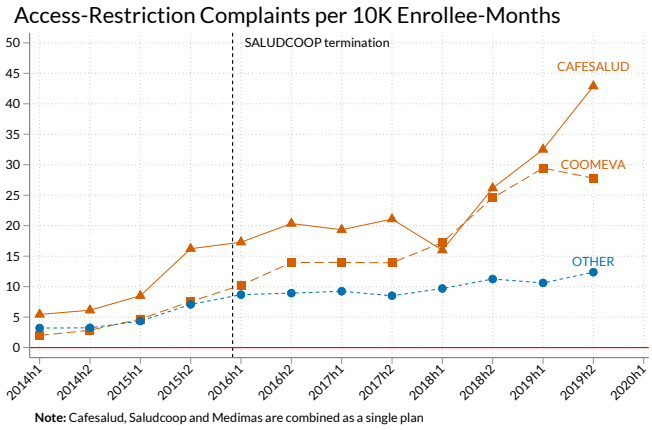
The analysis proceeds in three chapters. Chapter II establishes two key empirical facts through descriptive and causal evidence. First, there are substantial differences in care provision between the collapsing plans and available alternatives, documented through complaint rates and causal estimates of each plan's effect on medical care utilization. These plan effects capture the extent to which enrollees enjoy effective access to medical services—what I term *mean care provision*. To address the econometric challenge that plan choice correlates with unobserved factors that also determine care utilization, I use an instrumental variables strategy exploiting employer-driven steering of new entrants into plans. Second, health risk increases choice persistence substantially. This conclusion is supported through two approaches: a predictive risk score analysis showing that higher-risk individuals within collapsing plans are less likely to switch, and an event-study design showing that cancer diagnoses decrease six-month switching probability by up to 63%. Together, these findings suggest a problematic mismatch: individuals most likely to benefit from switching out of deteriorating plans are also least likely to do so.

Building on this evidence of health-driven choice persistence, Chapter III quantifies

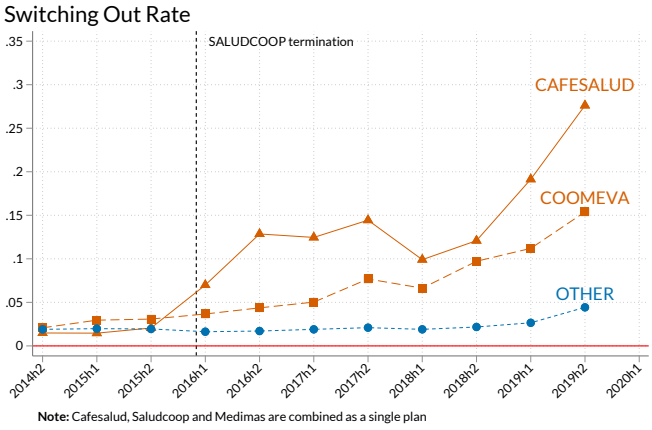
[2]There are some restrictions to switching. For example, patients are not allowed to switch during a hospitalization



(A) Market shares



(B) Average Access-Restriction Complaints



(C) Switching Out Rates

FIGURE I.1: Terminated Plans Collapse

This figure shows relevant time trends for the terminated plans at the half-yearly level. Panel A shows the evolution of market shares in Cafesalud and Coomeva, where enrollment is measured at the end of each half year. For this figure, I combine Saludcoop and Cafesalud into one single plan, as explained in Section 3.2. Panel B shows the time trends for average acess-restriction complaints for Cafesalud, Coomeva, and all other plans (blue line). Panel C shows the time trends for average switching out rates. The sample is restricted to the geographic markets corresponding to the 23 main cities and metropolitan areas. The definition of access-restriction complains can be found in Section 1.1.

how much access to medical care individuals are willing to forgo to remain in their current plan by estimating a discrete choice model of plan demand. In the model, inertia is formalized as a switching cost that individuals trade off against the expected care they would get across different alternatives. The key input to the model is an individual-level prediction of medical care utilization under each plan. These counterfactual predictions are generated by implementing plan-specific machine learning models that are trained on rich demographic and diagnostic data. The results show that both age and risk increase switching costs, and that individuals in the top 5% of the risk distribution exhibit near-complete inertia, requiring implausibly large increases in expected care provision to justify switching. The model identifies switching costs by comparing the choices of new enrollees, who face no inertia, with existing enrollees.

Chapter IV examines how the correlation between risk and choice persistence reshaped plan risk composition during the collapse of Coomeva and Cafesalud, and its consequences for market stability. The analysis explores switchers' contribution to enrollment, their impact on receiving plans' average risk, and their relative profitability. The results reveal a striking pattern: while terminated plans saw their average risk deteriorate as healthier enrollees exited, receiving plans experienced an inflow of switchers who were sicker than their existing populations. This seeming paradox reflects the initially higher risk profile of the terminated plans. Under the program's coarse risk adjustment—which uses only sex, age, and geography—this selective attrition undermined profitability, contributing to the decline of the terminated plans.

Finally, Chapter V concludes by discussing policy implications and directions for future research. To provide context for these contributions, the following sections situate the findings within existing literature and provide additional detail on the institutional setting and data sources used in the analysis.

2 Related Literature and Contributions

This dissertation makes three main contributions to the literature on consumer choice in health insurance markets. First, it documents and quantifies substantial heterogeneity in mean care provision across health plans, and studies how choices respond to differences in this important dimension of plan quality. Second, it establishes health status as a driver of consumer inertia, providing both correlational and causal evidence of this relationship. Third, it explores the implications of health-driven inertia for plan quality and market stability in a program covering over 20 million enrollees.

These contributions situate this thesis within a large body of work that studies the role of consumer choice in the functioning of market-based social insurance programs. In the case of health insurance, the policy rationale for managed competition, as articulated by Enthoven (1978a,b, 1988), holds that consumer choice among competing providers can improve efficiency and foster innovation through market discipline. However, extensive empirical work documents pervasive choice frictions in healthcare markets that challenge this vision. For example, Chandra et al. (2019) review evidence of inertia, inattention, and misperceptions in both insurance and healthcare decisions, emphasizing that healthcare choices are inherently complex and prone to behavioral biases that can impede market functioning. Hendren et al. (2021) formalize this concern about the design of social insurance programs, showing that the social value of choice depends not only on choice frictions, but also on preference heterogeneity, asymmetric information, and the availability of alternative provision outside the regulated market.

Quality Heterogeneity Across Plans

The first contribution builds on recent work documenting substantial quality differences across insurance and healthcare providers. This literature includes studies documenting causal differences in mortality across plans (Abaluck et al., 2021) and in medical spending (Geruso et al., 2023), as well as ongoing work that quantifies quality differences across in-

surers (Handel et al., 2021), hospitals (Hull, 2018), and physician's provision of maternal care (Posso et al., 2024). This literature, in turn, is adjacent to similar studies that quantify provider quality in education, such as Chetty et al. (2014) and Angrist et al. (2017).

The focus on a non-price dimension is also related to recent empirical studies that examine how insurers compete on dimensions other than premiums in the presence of risk selection. This literature has focused mostly on network design, with contributions by Shepard (2022) and Kreider et al. (2024) in the U.S. and by Serna (2024) in the Colombian context. This empirical literature was preceded by a long theoretical tradition studying competition on contract characteristics in insurance settings with asymmetric information. The seminal paper by Rothschild and Stiglitz (1976) studies a perfectly competitive setting with one-dimensional individual heterogeneity where insurers choose the price and coverage levels of contracts. Azevedo and Gottlieb (2017) extend this framework to multidimensional individual types and abstract endogenous contract characteristics, and Veiga and Weyl (2016) consider a monopolist who chooses the price and endogenous quality of a single product. Although the framework in the latter paper can be also used to study the case of a duopoly, competition on quality with horizontal differentiation has received less attention. One such example is Olivella and Vera-Hernández (2007), who consider duopoly model with two types of consumers and a fixed capitated payment.

Health Status and Consumer Inertia

The second contribution addresses a significant gap in our understanding of choice persistence in health insurance markets. While consumer inertia is pervasive across health insurance settings, with many studies documenting its prevalence and the substantial financial costs it entails (e.g., Handel, 2013; Polyakova, 2016), the role of health status as a driving factor has received limited attention. This gap is particularly important because, intuitively, the value of switching to higher-quality plans should be greatest for individuals with high medical care needs, yet we know little about how health risk affects their responsiveness to quality differences.

This dissertation provides compelling evidence that health risk substantially increases choice persistence. The findings show that even in settings with dramatic quality differences between plans and virtually unrestricted switching opportunities, health risk significantly increases choice persistence for those enrolled in low-performing alternatives. While some studies, such as Duijmelinck and van de Ven (2016), also document a positive correlation between risk, age, and choice persistence, this direct relationship has not been the primary focus of the extensive literature examining consumer inertia and risk selection in health insurance markets.

Methodologically, this work is especially close in spirit to Handel (2013) and Polyakova (2016), who model inertia as a switching cost and empirically quantify it using discrete choice models of plan demand. The approach is particularly similar to Polyakova (2016) in modeling the indirect utility of each plan in product-characteristic space, rather than treating insurance options as financial lotteries under uncertainty as in Handel (2013). The model, implemented in Chapter III, stands in contrast to an adjacent strand of literature that focuses on unpacking the specific mechanisms underlying consumer inertia—such as inattention, hassle costs, and preferences for provider continuity—including work by Ho et al. (2017), Handel and Kolstad (2015), Drake et al. (2022), and Heiss et al. (2021).

Rather than exploring the specific mechanisms behind health-driven inertia, this dissertation focuses on establishing and quantifying the overall relationship. This approach reflects the recognition that health status likely interacts in complex ways with multiple behavioral biases and mechanisms underlying choice persistence, making their identification particularly challenging. The study of choice persistence spans multiple literatures and has received considerable attention, with research identifying various driving factors including learning (Crawford and Shum, 2005), search costs (Diamond, 1971), and default bias (Samuelson and Zeckhauser, 1988). As changes in individual health status may simultaneously involve evolving preferences, attention shocks, increased cognitive or hassle costs, and learning processes, they create a web of interactions that is difficult

to disentangle empirically.[3]

Program Design in the Colombian Health System

This dissertation also contributes to a nascent literature that exploits rich administrative data from the Colombian health system to study different aspects of health insurance program design. Recent studies have examined network design in response to selection incentives (Serna, 2024), price sensitivity in medical care demand (Serna, 2021, 2025), and the impact of cost-sharing on mortality (Buitrago et al., 2023). Importantly, Serna (2025) also studies how health plans restrict access to medical care in the same insurance program. The analysis documents how utilization management practices lead to restrictions in access to medical care, but it does not examine how these restrictions vary across health plans, which is the central focus of this dissertation.

The termination of SaludCoop is a particularly significant market event that is currently the subject of multiple ongoing studies. For example, Buitrago et al. (2024b) examine the health consequences of SaludCoop's termination, documenting a 22% increase in mortality among non-SaludCoop enrollees through strategic reductions in provider network breadth by incumbent insurers. While their analysis focuses on health outcomes and supply-side responses to insurer exit, this dissertation primarily examines the demand-side dynamics of consumer choice and risk selection. Rather than studying how incumbent insurers react to termination, the focus here is on understanding why some consumers failed to switch out of declining plans despite substantial quality differences, and how this health-driven inertia reshaped risk composition across plans.

This thesis is more closely related to Buitrago et al. (2024a), who study optimal reassignment policies following plan terminations in the presence of consumer inertia and insurers' strategic network design responses. They also exploit SaludCoop's termination to examine choice responsiveness to plan quality differences, focusing on provider network

[3]For a comprehensive review of the early theoretical literature on switching costs and competition, see Farrell and Klemperer (2007). Recent methodological advances in the empirical literature include Pakes et al. (2022), who propose novel approaches using moment inequalities to quantify switching costs.

breadth as the key quality dimension rather than mean care provision. Their demand model quantifies switching costs by proposing that consumers respond to the different out-of-pocket costs across plans induced by the differences in price between different networks. They estimate a median switching cost of 1.1 million pesos and, consistent with the findings in this dissertation, show that sicker patients exhibit higher switching costs.

3 Institutional Setting and Data

Colombia's Régimen Contributivo provides an ideal laboratory for studying the relationship between health risk and consumer inertia. As detailed in the introduction, three features make this setting particularly well-suited for the analysis. First, there is compelling evidence of substantial quality differences across plans, especially during the decline and eventual termination of two large insurers—Cafesalud and Coomeva—that together covered approximately 30% of urban enrollment. Second, the program's regulatory framework creates near-ideal conditions for studying consumer responsiveness to plan quality: all plans offer identical standardized contracts, and switching rules are highly permissive. Third, the program's large scale provides the statistical power necessary to examine relationships involving rare health events such as cancer diagnoses.

These features create a setting where inertial consumers could forego substantial value by remaining enrolled in declining plans, allowing us to study how health status influences this phenomenon. This section provides additional detail on the institutional context, the history of the terminated plans, and the administrative data sources used in the analysis.

3.1 Setting: Colombian Régimen Contributivo (RC)

Colombia operates a dual health insurance system that achieved near-universal coverage by 2014, enrolling over 96% of the population.[4] The system consists of two main pro-

[4]The Colombian health system was created in the 1990s and is called Sistema General de Seguridad Social en Salud (General System of Social Security in Health, or SGSSS for short). For detailed explanation

grams, each covering roughly half the population. The *Régimen Subsidiado* (RS) serves households living in poverty and other vulnerable populations, with free enrollment funded entirely through public resources.[5] The analysis focuses on the *Régimen Contributivo* (RC), which provides mandatory coverage for formal sector employees and the self-employed. The RC is funded through monthly payroll tax contributions paid by workers and their employers, and enrolled approximately 20.5 million people, including both contributors and their dependents, in 2016.

In the RC, competing health plans called *Empresas Promotoras de Salud*, or EPSs, all offer the same standardized contract with identical benefits and financial characteristics.[6] The benefit package is comprehensive, covering 90% of approved drugs and 97% of medical procedures as of 2022 (Melo-Becerra et al., 2023). Individuals choose among plans operating in their geographic market, defined at the *municipio* level.

The regulatory framework eliminates most sources of plan differentiation common in other insurance markets. Enrollees pay no premiums beyond their payroll tax contributions, and while cost-sharing rules vary by enrollee status (contributors versus dependents) and income levels, these rules are uniform across all plans. Plans receive capitated payments from the government based solely on enrollee age, sex, and municipality of residence—a relatively coarse risk adjustment that does not account for health status or medical history. The program also limits vertical integration, restricting health plans to contracting no more than 30% of their costs with their own providers.

With contract terms, pricing, and benefits standardized, health plans must compete on operational dimensions that directly affect care delivery: building provider networks, managing utilization, and coordinating patient access to services. This institutional design forces competition into dimensions directly related to care access, which is precisely the dimension of quality this study examines. Differences in plan performance therefore stem primarily from variations in how effectively plans provide access to medical care to

of the institutional design and its history, see Giuffrida et al. (2009) and OECD (2015).

[5]Eligibility for the RS is determined through a proxy means test survey called SISBEN that assesses socio-economic status. Other vulnerable groups, such as displaced persons, are also eligible.

[6]Throughout, I use the terms *EPS*, *plan*, and *insurer* interchangeably.

their enrollees, rather than from differences in contract design or financial incentives.

Plan Switching Rules

Based on the regulatory framework established by Decreto 2353 de 2015 (Ministerio de Salud y Protección Social, 2015a), plan switching in the Régimen Contributivo operates under a set of rules designed to guarantee consumers' right to choose their health plan. The fundamental requirement is a minimum permanence period of 12 months (360 days) with the current plan before switching is permitted, though several exceptions exist, including family unification, cases where the EPS no longer operates in the enrollee's municipality, or documented instances of deficient service provision. Additional prerequisites include being current on all contributions to the program, and neither the enrollee nor their dependents can be hospitalized during the switching process. Only the contributing member (cotizante) can initiate the process for their nuclear family, and it can be done on any day of the month. The timing of the request determines when the change becomes effective: if submitted within the first five days of the month, the change takes effect the following month; if submitted after the fifth day, it becomes effective two months later. The entire process is free of charge and can be completed either through a physical form at the plan office or, since March 2018, online through a government portal.

3.2 Collapsing Plans: Saludcoop, Cafesalud and Coomeva

This study exploits substantial differences in plan quality by focusing on the contrasting performance of Cafesalud and Coomeva relative to other plans during the period 2016-2019. These quality differences provide an ideal setting to study consumer responsiveness to plan performance—the differences were so stark that both plans were eventually terminated by the government in 2022 due to poor service delivery and financial instability.

The history of Cafesalud is closely linked to a third plan called Saludcoop that was terminated in November 2015. Saludcoop participated in most urban geographic mar-

kets and accounted for around 20% of national enrollment when it was terminated. All of their enrollees were automatically moved to Cafesalud in December 2015 and were required to stay with this plan for at least three months, after which they were free to switch. In August 2017, Cafesalud was sold and re-branded as *Medimás* as part of a reorganization effort. Figure I.2 shows the evolution of enrollment in this plan family during the study period (2016-2019). To simplify the presentation, I combine these three plans (Saludcoop, Cafesalud, and Medimás) into one entity and refer to the group as *SCM*.

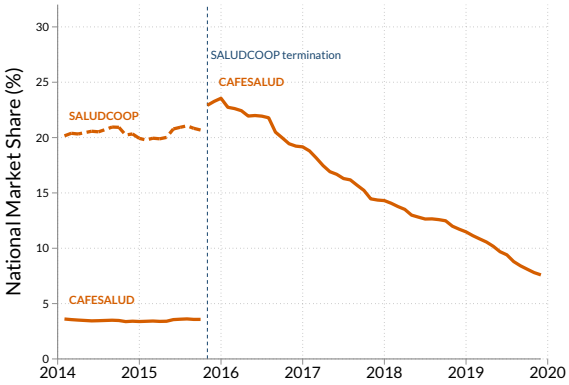


FIGURE I.2: Saludcoop Termination

Note: This figure shows the monthly market shares of Cafesalud and Saludcoop. In December 2015, Saludcoop enrollees were moved to Cafesalud. Enrollment is measured at the monthly level from aggregate data. *Data source:* Cubos Afiliados BDUA.

3.3 Data and Summary Statistics

Using comprehensive administrative data on social security contributions, enrollment, healthcare utilization, and complaints, I construct two main datasets for the analysis. The first is a half-yearly individual panel that includes detailed demographics, health plan choices, mortality, employment, and healthcare measures such as medical care utilization and diagnostic information. The second is a half-yearly plan-market panel of enrollment and quality measures calculated from the universe of complaints filed at *Superintendencia Nacional de Salud* (SNS), the government agency that regulates the healthcare system. As explained below, the analysis focuses on urban markets.

Analysis datasets

Half-Yearly Panel of Individuals—. To construct this dataset, I integrate six years of administrative records from different sources, including social security contributions, enrollment, and claims for the universe of Colombian residents who were enrolled in the RC at least once during 2014 to 2019.[7] For each individual and each year from 2014 to 2019, I observe a snapshot of health plan choice and demographics (including mortality status) at the end of June and December. I complement these demographics with income from formal employment, calculated from the universe of payroll tax contributions for 2016 to 2019. Finally, I use the universe of reported claims in the RC from 2014 to 2019 to construct variables capturing the value of care utilization, as well as events such as the incidence of certain diagnoses and the use of different types of care. These claims data are collected as part of the yearly actuarial calculations of capitated payments conducted by the Colombian Ministry of Health and Social Protection (Ministerio de Salud y Protección Social, 2015b, 2017, 2018a,b, 2019, 2020).

Half-Yearly Panel of Plans-Markets—. Using the universe of complaints filed at the National Superintendency of Health (SNS) and the enrollment data, I construct a half-yearly panel at the geographic market-plan level that includes average enrollment and complaint rates by type. The four types of complaints considered are: (1) any complaints, (2) life-threatening complaints where individuals indicate that their situation creates an immediate risk to the enrollee's life, (3) cancer complaints where the diagnosis reported involves a type of cancer, and (4) access-restriction complaints filed when individuals argue that their health plan is restricting access to medical care.

Limitations

While the administrative data is quite comprehensive, the main limitation is that not all insurers appear in the claims data every year. The reason for this exclusion is that each year the MSPS conducts a data validation process where they cross-check the reported

[7] The original data sources are *BDUA* for enrollment, *PILA* for social security contributions made via payroll tax, *Suficiencia* for claims, and *RIPS* for diagnostic data used in Chapter III.

costs in the claims data against the costs reported to the market regulator (SNS). When inconsistencies between these two sources are substantial, the plan is dropped entirely from the sample used for actuarial calculations of capitated payments, and consequently from the research database.

Overall, insurers that appear in every year of the study period accounted for 60% of enrollment in the RC in 2015, as shown in Table I.2. The analysis focuses primarily on the largest 8 plans by enrollment at the beginning of 2016: Sura, Sanitas, Compensar, Salud Total, Famisanar, Nueva, Coomeva, and Cafesalud. Importantly, we have claims data for 2014-2015 for most plans in this analysis (with the exception of Sanitas), which is particularly valuable because it allows us to compute a measure of individual risk for those years for most enrollees in the sample. This baseline risk measurement is crucial for the analysis in Chapters II and III, as it enables the construction of predictive risk scores and the identification of health-driven choice persistence.

It is important to note that SCM only reported claims in 2014-2015, and Sanitas only for 2016-2019. This temporal variation in data availability is incorporated into the analysis design, with different specifications used depending on the time periods and plans under examination. This limitation also raises the issue of whether there are differences in reporting practices across plans that may be biasing the results. To address this concern, where appropriate, we rely on within-plan comparisons that hold reporting practices constant (for example, in Chapter IV).

A second data limitation is that the claims data only allows us to observe utilization and health measures during enrollment in the RC program. This poses a challenge for the estimation of switching costs in III, as we exploit the decisions of new entrants who transition from the subsidized regime (RS) to the contributory regime (RC) and who, by definition, do not appear in the RC claims data prior to their entry. To address this issue, we leverage diagnostic data from an alternative source called RIPS (*Registro Individual de Prestación de Servicios de Salud*), which includes all insurers across both regimes. While RIPS has important reporting issues and is generally of lower data quality than the Suficiencia claims data, it provides the necessary coverage to construct health histories for

new entrants. This represents the best available solution given the data constraints.

Restriction to Urban Markets and Summary Statistics

This dissertation focuses on urban markets, which I define as those corresponding to the 23 cities and metropolitan areas identified by the national statistical agency—*Departamento Administrativo Nacional de Estadística* (*DANE*). The reason for focusing on these markets is twofold. First, parts of the analysis involve comparing individuals across granular demographic bins that are specific to each municipio. To ensure that each demographic bin has sufficient observations, it is necessary to exclude small municipios. As shown in Table I.1, the median number of enrollees is 763 across all municipios but 139,623 across urban municipios. Second, restricting the geographic scope reduces the dimensionality of estimation routines considerably by limiting the number of demographic bins. To further reduce dimensionality, I combine markets at the metropolitan area or city level, which reduces the number of geographic markets from 44 to 23.

Table I.1 presents additional statistics comparing urban and non-urban markets. Urban markets account for around 75% of national enrollment in the RC, and the median number of plans in operation is higher (6 compared to 4), meaning that individuals tend to have a larger menu of alternatives in urban markets. Moreover, Table I.2 shows descriptive statistics for each plan. Notably, 9 plans accounted for almost 90% of enrollment in the RC in urban markets in 2015, indicating that the market structure of the RC can be characterized as an oligopoly.

The detailed summary statistics by plan for 2015 and 2019 presented in Table A.1 reveal several important patterns within these urban markets. First, there is substantial variation in plan size, with enrollment ranging from around 1.1 million (Compensar) to over 5 million (Cafesalud) in 2015. The terminated plans—Coomeva and Cafesalud—experienced significant enrollment declines over the period, with Coomeva's enrollment falling from 2.78 million to 1.48 million and Cafesalud's from 5.09 million to 1.67 million.

Second, the table reveals notable differences in enrollee demographics across plans. Nueva EPS consistently enrolls an older population, with a mean age of 44 in 2015 compared to 31-36 for most other plans, and has a lower median monthly income of 770 thousand pesos compared to the overall median of 849. This combination of older age and lower income helps explain Nueva's higher baseline risk profile documented in Chapter IV. In contrast, plans like Sanitas and Sura tend to enroll younger, higher-income populations.

Third, the income distribution patterns show considerable heterogeneity across plans, with some plans like Sanitas and Compensar attracting enrollees with higher median incomes, while others serve populations closer to the minimum wage threshold. These income differences raise an important issue: higher-income individuals may purchase private insurance on top of their RC benefits, and utilization under private insurance is not observed in the data. We abstract away from private insurance in the main analysis and, where appropriate, restrict the sample to those close to minimum wage who are unlikely to purchase supplemental private insurance. These demographic differences underscore the importance of controlling for observable characteristics that may drive the demand for medical care when comparing plan performance and analyzing switching behavior, as implemented throughout the empirical analysis.

TABLE I.1: Régimen Contributivo Statistics in 2016

	All municipios	Largest 100 municipios	23 Cities and M.A.
Av. number of enrollees	18,266	183,728	348,934
Median number of enrollees	763	52,747	139,623
Av. number of insurers (sd)	3.9 (1.3)	5.7 (1.5)	6.0 (1.5)
Median number of insurers	4	5	6
Number of municipios	1,121	100	44

Notes: This table shows descriptive market statistics for the RC using enrollment data from January 2016. Insurer participation in a municipio is defined as having a higher than 1% market share in that municipio. The reason for imposing this threshold is that individuals who move across municipios are sometimes allowed to remain enroll with plans that don't operate in their new municipio. In these cases, market shares are positive but very small, even if a plan does not operate in a municipio. *Sources*: Aggregate data on enrollment from Cubos Afiliados SISPRO.

TABLE I.2: Data Availability and Statistics by Insurer

Plan	Market Share in 2015	Market Share in 2019	Municipios in 2015	Municipios in 2019	Claims Data Years
Sura	12.1	19.0	19	21	2014 - 2019
Sanitas	7.5	15.5	36	32	2016 - 2019
Compensar	6.6	8.8	1	3	2014 - 2019
Coomeva	11.4	6.1	41	34	2014 - 2019
Famisanar	7.5	8.6	12	13	2014 - 2019
Salud Total	11.7	15.2	30	31	2014 - 2019
Nueva	11.8	12.9	44	44	2014 - 2019
Cafesalud-Medimas	4.4	5.5	23	39	2014 - 2015
Saludcoop	16.5	-	39	-	2014 - 2015
Other	10.5	8.4	36	41	-

Notes: This table shows descriptive market statistics and data availability for the largest health plans participating in the RC. Plas are ordered by their position in a national ranking of quality published by the Ministry of Health, which is based on survey data collected in 2015. Plan participation in a municipio is defined as having a higher than 1% market share in that municipio. The last column shows the availability of claims data. *Sources*: Aggregate data on enrollment from MSPS: Cubos Afiliados SISPRO.

Chapter II

Reduced-Form Evidence of the "Lock-In" Effect of Risk

This chapter establishes two key empirical facts: that there are substantial differences in care provision across plans, with the terminated plans providing less care, and that health risk increases choice persistence for individuals enrolled in the terminated plans. Together, these findings support the central argument that some individuals become locked into under-performing plans due to their health status.

Section 1 presents descriptive and causal evidence of heterogeneity in care provision across plans. The descriptive evidence leverages the universe of individual complaints filed against health plans at the government agency that regulates the insurance program—*Superintendencia Nacional de Salud* (SNS). The analysis reveals that the terminated plans had systematically higher complaint rates across different types of complaints, including those specifically about health plans restricting access to medical care, and even complaints where claimants report that a patient's life is potentially at risk. The causal evidence of quality disparity involves estimating each plan's causal effect on the medical care utilization of their enrollees. These effects capture the extent to which enrollees enjoy *effective* access to medical care under each plan, which is the key dimension of quality variation given that benefits are standardized across all health plans.

The main econometric challenge in estimating plan effects is that plan choice is likely correlated with unobserved health or preference factors that also determine medical care utilization, such as unobserved health risk or preferences for consuming more medical care. To overcome this challenge, I pursue a strategy similar to Geruso et al. (2023),

who use random assignment into plans in a 2SLS framework to quantify plan effects on medical spending and other utilization measures in Medicaid Managed Care. Instead of random assignment, I exploit new entrants' exposure to employer-driven steering into plans. Since enrollment in the RC must be processed by employers, there is scope for firms to steer their employees into particular plans. The identification strategy relies on the assumption that variation in employer steering—as captured by the share of other employees at the firm enrolled in each plan—is exogenous to the unobserved factors that determine medical care utilization.

The findings show that Coomeva provided significantly less care than other plans. On the extensive margin, the probability of using care within six months after enrollment increases by up to 25 percentage points when comparing the plan with the lowest average complaints to Coomeva. On the intensive margin, this difference amounts to around 3 logarithmic units in medical spending (measured in Colombian pesos). Due to data availability constraints, this causal analysis cannot be conducted for SCM, as these plans do not appear in the claims data during the period where we observe employer identifiers, which are crucial for the identification strategy used in plan effects estimation. To validate these results, I demonstrate that the estimated plan effects align with enrollees' complaints about plans restricting access to medical care.

Section 2 investigates the relationship between health risk and choice persistence using two measures of risk. The first is a *risk score*—a measure of ex-ante health status calculated by estimating a predictive model of medical spending as a function of past diagnoses and observable demographics. Using this measure for a descriptive analysis of switching behavior shows that individuals with higher risk were less likely to switch out of the terminated plans. The six-month probability of switching out is 2.46 percentage points lower for the sickest 5% compared to the healthiest 50%—a substantial difference given that the average six-month switching rate at these plans is 10.4% during this period.

The second measure of risk is the development of one of seven types of cancer. Exploiting the panel structure of the data, I quantify the causal effect of these diagnoses on

switching behavior using a flexible event-study design based on Borusyak et al. (2024). Cancer diagnoses decrease the six-month probability of switching health plans by up to 63%. Exploring heterogeneity by plan, I find that at Coomeva, the decrease in switching rates due to cancer is around 50%, while at other plans the corresponding figure is 85%. This provides evidence that some individuals make choices consistent with the documented quality differences: the decrease in switching probability should be smaller for those enrolled in a "good" plan compared to a "bad" plan, as there is less to gain from switching. However, the fact that the effect remains negative even at the terminated plan shows that, on average, chronic illness leads to increased choice persistence even in low-value options.

1 Evidence of Quality Differences

The first step in the analysis is to provide evidence that there are substantial differences in quality across health plans, where "quality" is broadly understood as the amount of access to medical care a health plan provides to its enrollees. This section documents three key facts: (1) the terminated plans consistently under-perform other plans across quality metrics based on complaints data; (2) while overall market shares are largely unresponsive to these quality differences, switching behavior is responsive; and (3) non-terminated plans causally increase medical care utilization compared to terminated plans.

1.1 Differences in Complaint Rates

I first leverage the universe of complaints that enrollees file against health plans to calculate four measures of average complaints. These complaints are filed directly by individuals at the government agency that regulates the insurance program (SNS). The relative ranking of health plans resulting from each of these measures is consistent, with the terminated plans systematically showing high complaint rates.

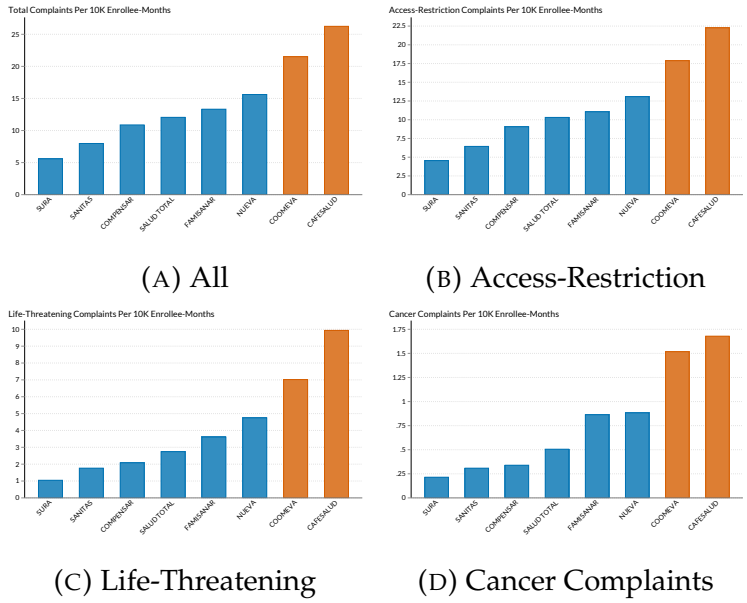
When individuals file complaints against health plans, the regulator captures detailed information about the complaint type, whether it involves a life-threatening issue, and any health conditions related to it. Based on this information, I construct four measures of average complaints for each plan at both the national and geographic-market levels:

1. **All:** Total number of complaints of any type.
2. **Access-Restriction:** Complaints filed when individuals argue that their health plan is restricting access to medical care.
3. **Life-Threatening:** Complaints where individuals indicate that the situation creates an immediate risk to the enrollee's life.
4. **Cancer:** Complaints where the diagnosis reported by individuals involves a type of cancer.

Figure II.1 shows the aggregate results for all urban markets across 2016-2019. The two terminated plans, highlighted in red, have the highest complaint rates across all four dimensions. The relative ranking of plans according to these quality measures remains stable over time, as shown in Figure B.1 in the appendix. These patterns also hold at the market-plan level: as explained below and shown in Figure II.2, the terminated plans consistently have higher complaint rates than their respective geographic market averages across most markets.

1.2 Correlation Between Choices and Quality

To understand which groups of consumers may be more responsive to the quality differences documented above, I explore the correlation between enrollment and complaints within each geographic market. For each plan-market combination j, I analyze how its quality (normalized relative to the average in its geographic market) correlates with the choices of two groups: switchers and all enrollees in the market. The analysis of switching out rates itself constitutes another measure of quality, as consumers vote with their



(A) All (B) Access-Restriction

(C) Life-Threatening (D) Cancer Complaints

FIGURE II.1: Average Complaints by Type and Insurer

Note: This figure plots the four measures of average complaints discussed in the main text for each plan for the period 2016-2019: Panel A for all complaints, Panel B for access-restriction complaints, Panel C for life threatening complaints, Panel D for cancer complaints. The numerator for each measure is the total number of complaints in 2016-2019, the denominator is the total number of enrollee-months in the same period. Health plans are ordered according to the results of Panel A. *Sources:* Complaints data from SNS and enrollment data from Cubo Afliados SISPRO.

feet by leaving plans that provide poor service. To account for geographic differences in baseline quality levels and the number of participating insurers, I normalize both enrollment and quality measures by regressing each on market fixed effects and computing standardized residuals. Specifically, I estimate linear regressions of the form:

$$y_j = \alpha_{m(j)} + \varepsilon_j \tag{II.1}$$

where j is an index for plan-markets, $m(j)$ is j's market, y_j is an outcome to be residualized, $\alpha_{m(j)}$ is a market fixed effect, and ε_j is a mean-zero error term. The standardized residuals capture the relative quality or enrollment of each plan within each demographic market and can be written as $\tilde{\varepsilon}_j \equiv \frac{\hat{\varepsilon}_j}{s_j} = \frac{y_j - \hat{\alpha}_{m(j)}}{s_j}$, where s_j is the standard error of the residual $\hat{\varepsilon}_j \equiv y_j - \hat{\alpha}_{m(j)}$.

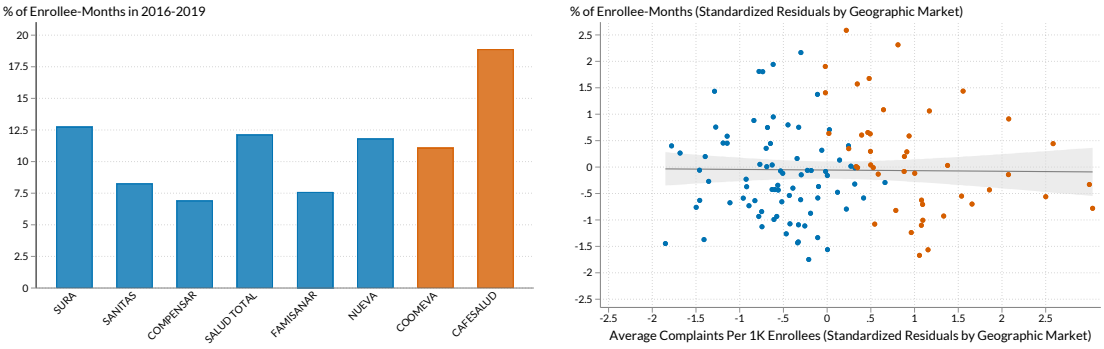
I consider three different measures of enrollment, all aggregated across 2016-2019. First, total enrollment, measured by the proportion of enrollee-months corresponding

to each plan. Second, the probability of switching out to a different plan, measured by the ratio of "switchers out" to enrollment at the beginning of 2016. Third, market shares among switchers, measured by the percentage of switchers each plan attracts. I compute the standardized residuals for each of these enrollment measures and for the four complaint measures described above.

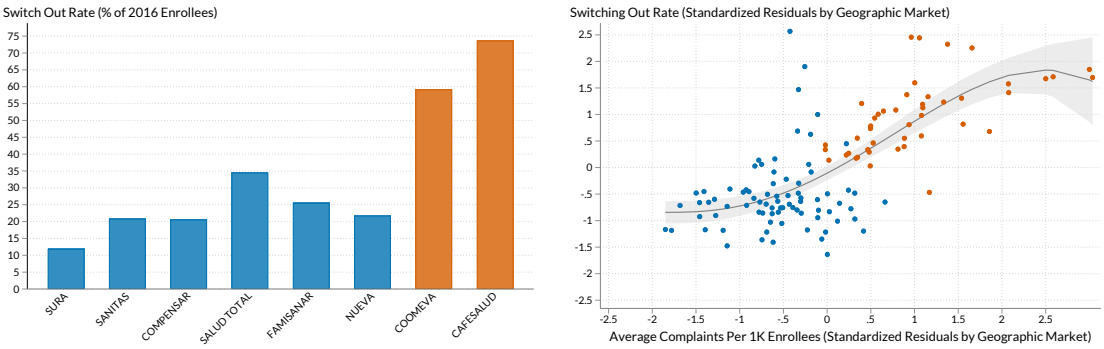
The results from this exercise are shown in Figure II.2. Each plot in the right column shows a scatter plot at the plan-market level, along with fitted predictive models to capture the relationship between variables. For total enrollment I use a linear fit, while for the other two measures I use polynomial regression to capture the nonlinearity evident in the scatter plots. Several conclusions emerge from this analysis. First, the terminated plans have high complaint rates relative to other plans consistently across markets, not only in the aggregate. This is evident in the right column of Figure II.2, where terminated plans (shown in red) always lie to the right of zero, indicating that their complaint rates exceed their respective market averages. Second, switching behavior is responsive to within-market quality differences, but total market shares are not. This pattern is also shown in the right column of Figure II.2, where the relationship between choices and average complaints is clearly positive for switching out rates (Panel B) and clearly negative for market shares among switchers in (Panel C), but flat for total market shares (Panel A). At the national level, the results are particularly striking: despite having switching out rates 2-3 times higher than other plans, the terminated plans still managed to attract large market shares over time.

1.3 Plan Effects on Medical Care Utilization

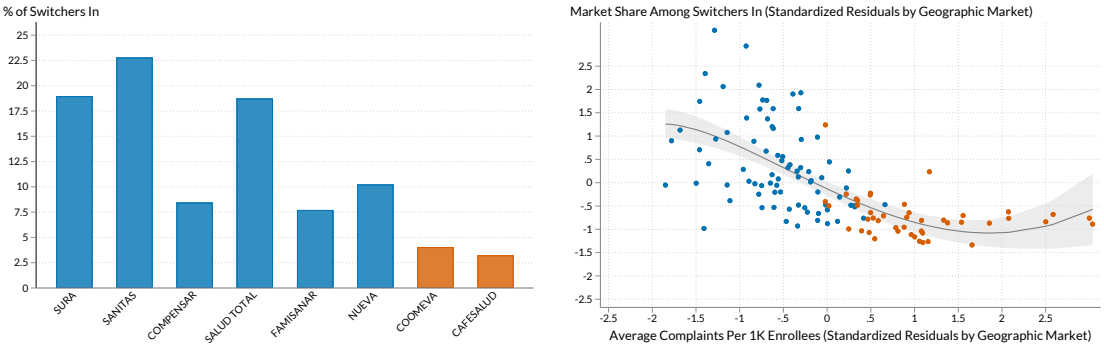
Section 1.1 showed that there are striking differences in complaint rates, but do these measures actually capture differences in effective access to medical care? In this section, I estimate the causal effect that health plans have on medical care utilization, showing that non-terminated plans causally increase medical care utilization by a substantial amount. Given that all plans offer the same contract with standard benefits and prices, I argue



(A) Total Enrollment



(B) Switching Out Rate



(C) Market Shares Among Switchers In

FIGURE II.2: Correlation Between Choices and Quality

Note: This figure shows the three measures of enrollment discussed in the main text for the period 2016-2019. Terminated plans are colored in red and non-terminated plans in blue. The left column shows results at the plan level, where the aggregation is done across all urban markets, and with plans ordered by increasing order of average complaints (same order as in Figure II.1). The right column shows results at the plan-market level. Specifically, it shows the standardized residuals from equation II.1. Each row corresponds to a different measure of enrollment. The first row is the total percentage of enrollee-months in 2016-2019. The second row is the total number of switchers in 2016-2019 as a percentage of enrollment at the beginning of 2016. The third row is the percentage of switchers that each plan attracted in 2016-2019. Each of the plots in the right column includes the result of a predictive model for the scatter, along with 95% confidence intervals. For the first row, I use a linear fit and for the other two a polynomial regression. The data used for this figure corresponds to a 10% random sample of Colombian residents.

that this constitutes evidence that non-terminated plans provide better access to medical care.

The main empirical challenge is that the choice of health plan may depend on unobserved factors—such as underlying health status or preferences for care—that also determine medical care utilization. For example, sicker individuals might be more attentive to the quality of different health plans and therefore more likely to enroll with better-performing plans. If this were the case, higher utilization at non-terminated plans would not result from their providing better access to medical care, but rather reflect that their enrollee pool is less healthy and demands more care. To overcome this challenge, I focus on a sample of employees who transition into the insurance program upon becoming employed, and exploit their exposure to employer-driven steering into plans as a source of variation in plan choice that is exogenous to these unobserved factors. The econometric framework and estimation closely follow the approach in Geruso et al. (2023), where the authors exploit random assignment of Medicaid beneficiaries into plans. Due to imperfect compliance, they use assignment indicators as instruments for actual enrollment.

This analysis excludes SCM due to data limitations. The identification strategy requires observing employer identifiers, which are only available from 2016 onwards. However, SCM do not appear in the claims data during this period, making it impossible to estimate plan effects for these terminated plans using the instrumental variables approach.

Econometric Framework and Identification Strategy

The estimation target is the causal effect of enrollment in a health plan $j \in \{0, 1, 2, ..., 6\}$ on individual outcomes that measure medical care utilization, where $j = 0$ is a reference plan. Following Finkelstein et al. (2016) and Geruso et al. (2023), I use a linear model where a measure of medical care utilization, denoted by Y, is determined by a plan component γ_j, a vector of observable covariates $\mathbf{X}$, and a mean-zero error term ε. The main estimating equations take the following form:

$$Y_i = \alpha + \boldsymbol{\nu}\mathbf{X}_i + \sum_{j=1}^{6} \gamma_j D_{j,i} + \varepsilon_i \tag{II.2}$$

In this equation, each observation corresponds to an enrollee (indexed by i) for a fixed time period. I include the following variables in the vector of observed covariates $\mathbf{X}$: metropolitan area fixed effects, monthly income quintiles, full interactions between age deciles and sex. I also include the following firm characteristics: number of employees, and average monthly income. The $D_{j,i}$ terms are indicators for enrollment in plan j, where the omitted plan $j = 0$ is Nueva.

The main econometric challenge is the endogeneity of the plan-enrollment indicators $D_{j,i}$. To overcome this challenge, I use an 2SLS IV strategy, focusing on individuals that enter the insurance program due to starting formal employment and instrumenting each plan indicator with the share of other employees at the firm who choose that plan. The rationale for using these instruments is that enrollment in the RC is administratively processed by employers as part of paying social security contributions, which are based on their employees' salary. This opens the door for employers to influence plan choice. For example, it could be the case that different employers have different default options, or that they nudge their employees into specific plans. The main idea is that variation in plan choice coming from differences in employer-driven steering into plans is exogenous to the unobserved individual characteristics that may influence health care utilization.

The 2SLS estimation approach is standard. The first stage consists in estimating $J - 1$ regressions of the following form (we use Nueva as a reference plan):

$$D_{k,i} = \alpha_k + \boldsymbol{\delta_k}\mathbf{X}_i + \sum_{j=1}^{6} \lambda_{k,j} z_{j,i} + \epsilon_{k,i} \tag{II.3}$$

In this equation, I use the same exogenous covariates as in equation II.2 and $z_{j,i}$ is the share of other employees at i's firm that choose insurer j. For the second stage, the single estimating equation is

$$Y_i = \alpha + \boldsymbol{\nu}\mathbf{X}_i + \sum_{j=1}^{6} \gamma_j \widehat{D}_{j,i} + \varepsilon_i \tag{II.4}$$

where the $\widehat{D}_{j,i}$ terms are the predicted enrollment indicators from the first stage.

The identification assumptions required are that the instruments are relevant and exogenous. The instruments are relevant if employer steering into plans, as captured by the market share at the employer, are indeed predictive of actual enrollment. This assumption is empirically verifiable and, as shown in the first-stage results, is strongly supported by the data.

The instruments are exogenous if they are independent of any unobserved factors in ε that determine medical care utilization Y in equation II.4. This assumption is more challenging to verify directly and requires careful consideration of potential threats to validity. Several factors could potentially violate the exogeneity assumption. First, unobserved firm characteristics that simultaneously affect plan choice and employee health outcomes could bias the results. For example, firms with different workplace safety cultures might both steer employees toward better plans and be more permissive of employees receiving care during work hours. Second, geographic proximity to provider networks represents a particularly important threat to exogeneity. Firms located near the provider networks of certain plans may be more likely to steer their employees toward those plans for convenience reasons. Simultaneously, employees at these firms would have easier access to care, potentially leading to higher utilization that reflects geographic accessibility rather than plan-driven access to medical care. This could generate a spurious positive correlation between employer steering and utilization outcomes.

Unfortunately, the data available does not locate individuals or firms at a level more granular than the municipio, as geographic identifiers such as zip codes are not used in the administrative records. This data limitation prevents us from directly controlling for proximity to provider networks, which represents the most significant potential threat to the exogeneity assumption. Ideally, we would control for the distance between each firm's location and the provider networks of different plans. The inability to address this

concern directly means that the estimated plan effects should be interpreted with caution, particularly if there is systematic geographic clustering of firms and provider networks within municipios.

Sample and Measures of Utilization

The main outcomes of interest Y_i are $\log(1 + \text{Medical Spending}_i)$ and an indicator for positive medical spending $1[\text{Medical Spending}_i > 0]$, where medical spending is calculated as the total cost reported by plans in the claims data for each individual. These outcomes capture both the extensive margin (whether an individual uses any medical care) and the intensive margin (the amount of care consumed) of healthcare utilization. The analysis focuses on the first semester of 2016 to ensure a stable comparison period. This timing is crucial because the collapse of the terminated plans accelerated after this semester, potentially inducing systematic changes in care provision. By focusing on the first half of 2016, we capture plan effects during a period when quality differences were present but before the most dramatic disruptions occurred, providing a cleaner measure of the underlying differences in care provision across plans. The resulting sample has 234,969 individuals.

Results and Discussion

First stage results. Panel Figure II.3 shows that the instruments have a strong influence on plan choice. This figure shows the first stage coefficients $\widehat{\lambda}_{k,k}$ for $k \in \{1, 2, ..., 6\}$, with 95% confidence intervals. The interpretation of these coefficients is that a 1 pp increase in the market share of plan j at a new entrant's firm increases the probability of enrollment in that plan by $\frac{1}{100}\hat{\lambda}_j$. As it can be seen in the figure, most of the first-stage coefficients are very close to one and statistically significant. This means that the probability of a new employee enrolling with a particular plan increases at a rate close to one with the probability that other employees at the firm choose that same plan.

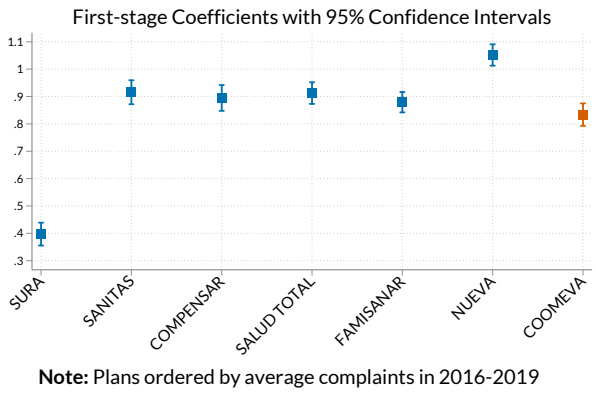


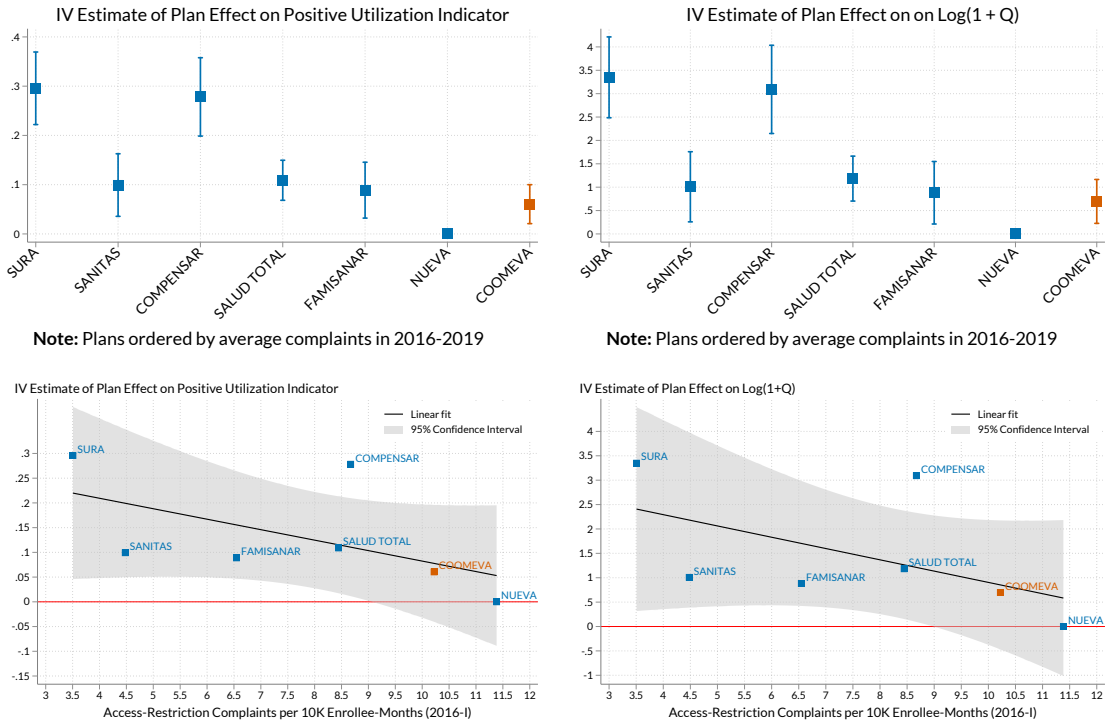
FIGURE II.3: First stage coefficient

Notes: This figure plots, for each plan, the estimated coefficient $\hat{\lambda}_{k,k}$ that corresponds to the first-stage equation for $D_{k,i}$. The point estimate is plot as a square with 95% confidence intervals in brackets. Coomeva is shown in red and the rest of the plans are shown in blue.

Main results. Figure II.4 shows the IV estimates for both outcomes in the first row, and the correlation between the estimated plan effects and the average of access-restrictions complaints. The results show substantial variation in plan effects. Taken at face value, they imply that enrolling in the highest-ranked plan (Sura) increases the probability of using medical care by 30 pp relative to the reference plan, and that it increases medical care utilization by 3.5 log points. These magnitudes are very large and require further analysis and validations. As a first step to validate these results, the second row of Figure II.4 plots the correlation of the plan effects with the average access-restriction complaints at each insurer during the first semester of 2016, which is the period where we measure utilization for this exercise. The negative correlation suggests that access-restriction complaints indeed capture underlying differences in how much medical care health plans provide to their enrollees.

2 Evidence of Health Risk Driving Choice Persistence

The substantial differences in quality across plans documented in the previous section raise an important question: why do people remain enrolled in plans that provide subpar access to medical care? This is particularly puzzling in this setting, given that all health plans offer the same standard contract, and individuals are allowed to switch in any



(A) Positive Utilization Indicator (B) Log (1 + Medical Spending)

FIGURE II.4: Plan Effects IV Estimates and Correlation with Complaints

This figure plots the IV estimates of plan effects, that is, of the coefficients γ_j from the second stage (Equation II.4). The dependent variable for the right column is a positive utilization indicator, and for the right column $\log(1 + \text{Medical Spending})$. All variables are measured for the first semester of 2016. The top row shows the plan effect estimates for each plan, where plans are ordered by average complaints in 2016-2019. The sample consists of individuals who were part of the RS in December 2015 and who transitioned into the RC due to starting employment in January 2016.

given month.[1] In this section, I show evidence that health status is an important driver of choice persistence: sicker individuals are more likely to persist in their choices. The evidence focuses on two measures of health status: a measure of ex-ante health risk, and cancer diagnoses.

2.1 Positive Correlation Between Ex-Ante Risk and Choice Persistence

To investigate the relationship between health status and choice persistence, I test whether sicker individuals, as measured by their ex-ante health risk, were less likely to switch out of the terminated plans. The measure of ex-ante risk is computed by predicting mean

[1] Recall that, as explain in Section 3, benefits and prices are fixed across health plans and are independent of health status. Moreover, plans are not allowed to screen or deny enrollment.

medical expenditures in 2015 as a function of diagnoses and detailed demographics in 2014. This prediction yields a "risk score" that captures ex-ante health risk at the start of 2015. The analysis consists of two parts. First, I show using OLS regressions that individuals in the highest quantiles of the risk score distribution have lower switching rates. Second, I implement a survival analysis showing that, among individuals who were originally enrolled at Saludcoop and then automatically moved to Cafesalud, sicker individuals persisted with their assigned plan substantially longer than healthier individuals.

Risk score calculation

For measuring ex-ante health status, it is important to take into account that naive measures, such as comparing medical care utilization in past periods, can capture both individual health and plan characteristics (e.g., the extent to which plans provide different levels of care for the same baseline health event). For this reason, I construct a risk score based on a predictive model of medical spending in 2015 as a function of diagnoses and demographics in 2014. Crucially, I estimate the parameters of this model using data from a single plan (Nueva EPS), and then construct predictions using these parameters. For enrollees at other plans, the risk score is an out-of-sample prediction. The interpretation of this measure of *ex-ante* risk is that it measures the expected medical spending of an individual *under a fixed reference plan*.[2]

The baseline model is an exponential mean model for medical spending:

$$E[Q_i \mid X_i] = \exp(\boldsymbol{\beta}\mathbf{X_i}) \tag{II.5}$$

where i indexes individuals, $\mathbf{X_i}$ is a vector of demographics and past diagnoses data observed in year 2014, Q_i is medical spending in 2015. The main sample restriction is that I only include individuals who were continuously enrolled in Nueva across both years. The predictive model is then estimated using a random sample comprising 90% of these

[2]This approach is similar to how risk adjustment works in settings like Medicare in the US, where the Centers for Medicare and Medicaid Services (CMS) uses data from a single program (Medicare FFS) to estimate the risk model that determines risk adjustment.

individuals, with the remaining 10% reserved as a validation (or hold-out) sample.

Results. The model demonstrates strong predictive accuracy across the ex-ante risk distribution. As shown in Table II.1, the ratio of average predicted medical spending to average realized spending in 2015 is close to one across all deciles of the risk score. These results are based solely on the hold-out sample, reducing potential concerns about overfitting.

TABLE II.1: Risk Model Results

Group	Number of obs.	Mean of Q_{2015}	Mean of $\hat{Q}_{2015}$	Predictive ratio
All	132,531	1,050	1,061	1.01
Deciles of $\hat{Q}_{2015}$				
1	13,254	295	281	.9542
2	13,254	352	424	1.206
3	13,252	405	530	1.307
4	13,253	519	614	1.183
5	13,253	605	700	1.156
6	13,253	775	792	1.021
7	13,254	913	923	1.011
8	13,252	1,211	1,128	.9315
9	13,253	1,634	1,500	.9181
10	13,253	3,794	3,716	.9795

Notes: This tables shows the fit of the risk score model using the observations from the 10% testing sample.

Ex-Ante Risk and Switching Out Rates

This subsection examines whether individuals with higher predicted health risk were less likely to switch out of the terminated plans. By leveraging the computed risk score, I explore switching patterns across risk quantiles, focusing on how these patterns differ between terminated and non-terminated plans. The analysis consists in estimating linear models of the following form using OLS:

$$\begin{aligned}\text{Switch Out}_{it} = &\sum_{r \in R} \beta_r \times 1[\text{Risk bin}_{it} = r] + \sum_{r \in R} \alpha_r \times 1[\text{Risk bin}_{it} = r] \times \text{Terminated}_{it} \\ &+ \phi_{D(i,t)} + \boldsymbol{\omega} \mathbf{X_{it}} + \varepsilon_{it}\end{aligned} \tag{II.6}$$

In this equation, i is an index for individuals and t for time in half-years; Switch Out$_{it}$ is an indicator for being enrolled with a different health plan in $t+1$; Risk bin$_{it}$ is an indicator corresponding to one of four risk score bins: $< p50$, $p50 - p75$, $p75 - p95$, $> 95p$; Terminated$_{it}$ is an indicator for enrollee i being enrolled with one of the terminated plans at time t, $D(i,t)$ is i's plan at time t, and $\phi_{D(i,t)}$ is a plan fixed effect. The vector $\mathbf{X_{it}}$ of observables demographics includes age fixed effects, sex, and municipio fixed effects.

The results of this exercise are shown in Figure II.5. I estimate separate regressions for 2015 and all periods after that. For the earlier period, which occurs before the rapid unravelling of Cafesalud, there is a clear but small risk gradient in the probability of switching out at the non-terminated plans. The mean switching out rate for individuals at the non-terminated plans is 1.27%, and the probability of switching out decreases for each risk bin. The probability of switching out for the sickest 5% is .35 pp lower than for the healthiest 50%. At the terminated plans, the mean switching out rate for the earlier period is larger at 2.15 %. However, the difference in the probability of switching out for the sickest 5% is not statistically significant at the 95% confidence level.

The results for the later period are highly suggestive that risk increases choice persistence. Recall that the later period is characterized by the widening of quality differences between the terminated and non-terminated plans (Figure I.1). At the non-terminated plans, the mean switching out rate remains low, with no clear risk gradient. At the terminated plans, however, the mean switching out rate spikes from 2.15% to 10.4%. Moreover, there differences in switching rates are substantially lower for riskier enrollees. For example, the switching out rate is 2.46 pp lower for the sickest 5% of individuals compared to the healthiest 50%. This is a substantial difference, given that the six-months average switching out rate at these plans is 10.4% during this same period. These results suggest that healthier individuals were more likely to respond to the deteriorating quality of the terminated plans, while sicker individuals exhibit higher choice persistence.

Survival Analysis of Saludcoop Enrollees Moved to Cafesalud

We can also study the relationship between choice persistence and ex-ante health status by focusing on the group of enrollees that were moved from Saludcoop to Cafesalud. As explained in Section 3.2, Saludcoop was terminated in November 2015 and all of their enrollees were moved to Cafesalud. Saludcoop enrollees had to stay for three months in Cafesalud, but were allowed to switch at any point after that. We can study how choice persistence relates to health status by implementing a survival analysis exercise where the failure event is defined as switching out of Cafesalud.

The results show that ex-ante risk is positively correlated with choice persistence. Figure II.6 below plots the Kaplan-Meier survival estimates by groups that are defined according to the value of utilization in 2014. We can see that for those in the highest 5% of this measure of ex-ante risk the survival curve is always above the other two. The probability of staying in Cafesalud four years after the termination of Saludcoop is 11 p.p. higher for the sickest 5% compared to the healthiest 75% (Panel A).

2.2 Causal Effect of Chronic Illness on Switching Behavior

The descriptive evidence in section 2.1 shows that risk is correlated with choice persistence. Moreover, the results in Section 1.1 showed that choices do not seem to respond to plan quality overall, but switchers' choices do. Overall, these results are suggestive of consumer inertia, where a large group of people may inattentively continue enrolling with their default option over time, but a smaller group of individuals become active and rationally decide to move away from low-quality plans into high quality plans. To investigate whether illness has a causal effect on choice persistence, I focus on the onset of chronic illness, as captured by the occurrence of cancer. Using a flexible event-study design, I find that cancer increases choice persistence substantially at the non-terminated plans, effectively locking consumers in these low-value alternatives. The event-study design is based on the econometric framework proposed by Borusyak et al. (2024) and exploits the panel-nature of the data.

Treatment Definition

The analysis focuses on seven common types of cancer: breast, prostate, cervix, lung, colon, stomach, and leukemia. Using data on the procedures and medications typically used as treatments for each of these conditions, *treated individuals* are defined as those who appear in the data with a cancer diagnosis and who received at least 3 treatment instances corresponding to that diagnosis. The *treatment date* is defined as the first half-year with a treatment instance. To ensure the capture of new cancer cases rather than continuations of pre-existing cases, individuals with treatment dates before 2016 are dropped. Treatment instances are used rather than diagnoses because reporting is not as reliable for diagnoses, which could lead to a high number of false positives. Additional details on the activities used to identify cancer diagnoses are provided in Appendix C. The main results are based on a set of 28,027 treated individuals. As a control group, we take a 5% random sample of individuals who are not treated, which amounts to 310,646.[3]

TABLE II.2: Summary Statistics for Event-Study Design

Statistic	Control	Treated
Number of individuals	322,813	29,576
Mean Age in 2016	40.0	62.9
Median Age in 2016	39	64
Switchers in 2014-2015 (%)	2.4	1.1
Switchers in 2016-2019 (%)	14.9	6.6
Mortality in 2016-2019 (%)	1.6	18.1
Medical Spending per Year in 2014-2015 (COP)	608	1,552
Medical Spending per Year in 2016-2019 (COP)	945	9,642

Notes: This table shows summary statistics for the treated and control groups. The treated group corresponds to those individuals who get a cancer diagnosis in, or after, 2016. Medical spending is measured in thousands of Colombian Pesos (current).

[3]The panel drops any observations where the individual is no longer enrolled in the insurance program. This means that the switching out indicator is missing when individual leaves program in the next half-year. The switching out indicator captures switching to another insurer while remaining enrolled in the program, not exiting the program.

Econometric Framework

Let E_i be the treatment date, where $E_i = \infty$ denotes the case where individual i never gets cancer in the panel. We are interested in the causal effect of cancer, which we denote by a treatment indicator W_{it}, on a "switching out" indicator defined as

$$\text{Switch Out}_{it} \equiv 1[D(i, t+1) \neq D(i, t)]$$

where i index individuals, t half-years and $D(i, t)$ is the plan individual i chooses at time t. Treated observations are those where $W_{it} \equiv 1[t \geq E_i]$ equals one. For each treated observation, the causal effect of cancer on switching plans is denoted by $\tau_{it} = \mathbb{E}[\text{Switch Out}_{it} - \text{Switch Out}_{it}(0)]$, where $\text{Switch Out}_{it}(0)$ is the potential switching indicator of individual i at time t if it is never treated.

In the first instance, the main estimation targets are averages of these causal effects at different time horizons since treatment started. They can be written as

$$\tau_h \equiv \frac{1}{N_h} \sum_{it \in \Omega_{1,h}} \tau_{it} \tag{II.7}$$

where $\Omega_{1,h}$ is the set of treated observations that correspond to h time periods after the treatment date and N_h is the size of that group. Formally, $\Omega_{1,h} \equiv \{it : t - E_i = h\}$ and $N_h \equiv |\Omega_{1,h}|$. I also estimate averages of the treatment effects for one of the terminated plans (Coomeva) vs for non-terminated plans. These estimation targets can be written as

$$\tau_h^g \equiv \frac{1}{N_h^g} \sum_{it \in \Omega_{1,h}^g} \tau_{it} \tag{II.8}$$

where $\Omega_{1,h}$ is the set of treated observations that correspond to h time periods after the treatment date and where i is enrolled in a plan belonging to group g, which can be either "terminated group" for Coomeva or "non-terminated group", for Sura, Famisanar, Compensar, Salud Total, or Nueva. N_h^g is the number of observations that belong to group

g at time horizon h. Formally, $\Omega_{1,h}^{g} \equiv \{it : t - E_i = h \text{ and } G_{i,t} = g\}$, and $N_h^g \equiv |\Omega_{1,h}^{g}|$.

The main two assumptions we need for identification are a generalized parallel-trends assumption and a limited anticipation assumption. Each of these is listed below.

Assumption 1. (Model of Potential Outcomes)

$$\mathbb{E}[\text{Switch Out}_{it}(0)] = \alpha_{a(i,t),m(i,t),s(i),D(i,t),t} \tag{II.9}$$

where, for individual i at half-year t, $a(i,t)$ is an age bin, $m(i,t)$ a metropolitan area, $s(i)$ is sex, and $D(i,t)$ is plan chosen.

Assumption 2. (No Anticipation)

$$\text{Switch Out}_{it} = \text{Switch Out}_{it}(0) \ \textit{for all} \ \ it \ \ \textit{such that} \ \ t < E_i \tag{II.10}$$

Assumption 1 says that the expected potential outcome is equal to a fixed effect that is specific to a granular demographic bin given by the interaction of age, metropolitan area of residence, sex, and current plan. Assumption 2 rules out that being treated in the future has a causal effect on current outcomes.

I follow the recommended practice in Borusyak et al. (2024) and separate the estimation of treatment effects from testing for the validity of Assumptions 1 and 2. The tests consists in assessing the statistical significance of the pre-trend coeffcients γ_k for $k = -8, -7, ..., -1$ obtained from running an OLS regression of the following linear model:

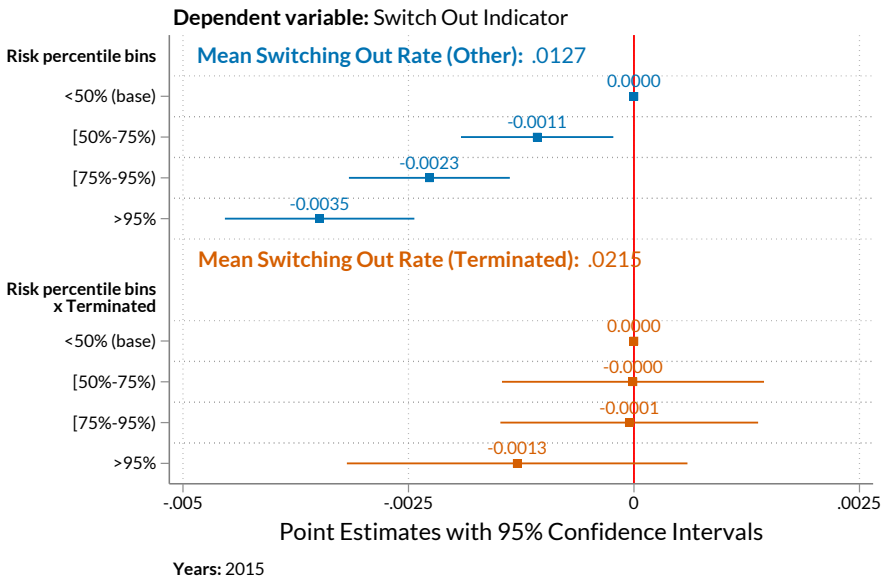
$$\text{Switch Out}_{it} = \alpha_{a(i,t),m(i,t),s(i),D(i,t),t} + \sum_{k=-8}^{-1} \gamma_k \mathbb{1}[t - E_i = k] + \eta_{it} \tag{II.11}$$

Under the null hypothesis that all the coefficients γ_k are equal to zero, Assumptions 1 and 2 imply equation II.11.

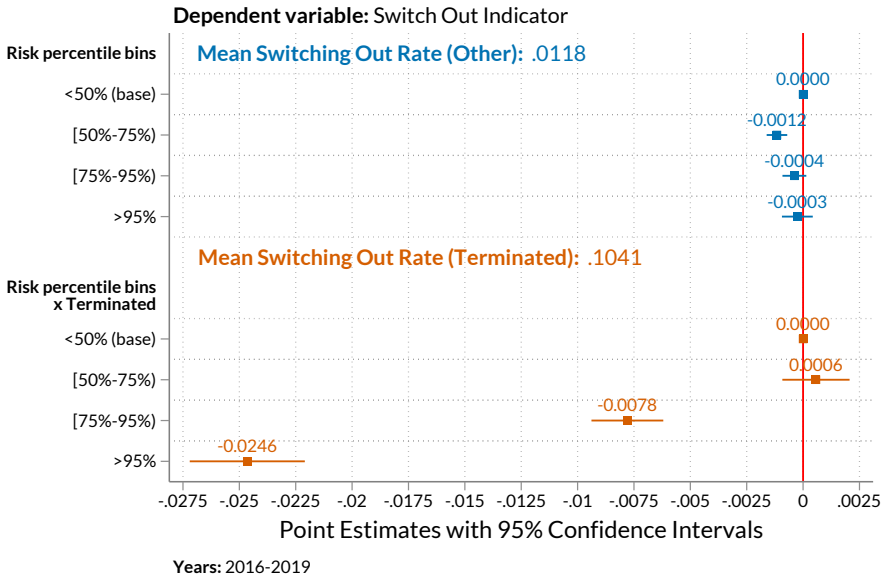
Results

Panel A of Figure II.7 shows the results of the pre-trend test and the estimates of the average treatment effects τ_h (average treatment effects across all plans). Panel B plots the same for the estimates of τ_h^g, which are average treatment effects specific to Coomeva vs the other plans. The pre-trend coefficients are very close to zero until the year before starting cancer treatment. The fact that the coefficients for $k = 1, 2$ are statistically different from zero at the 5% level is evidence of small but negative anticipatory effects. The magnitudes of these deviations from zero are very small relative to the big drop in switching out rates that happens upon treatment. As a matter of fact, the fact that the pre-trend coefficients are not positive is evidence that individuals are not strategically switching plans before starting cancer treatment. This is an important result, given that the RC does not have open enrollment periods and therefore is susceptible to this behavior, which could create a severe adverse selection problem. This type of *ex-post* adverse selection has been studied by Cabral (2017) in the context of dental insurance, finding that a large group of individuals strategically delay dental treatments, which creates an adverse selection incentive that can explain the unravelling of dental insurance markets.

The average treatment effects show a steep decline in the probability of switching out to a different plan. For non-terminated plans, the effect is persistent and amount to around 60%-90% of counterfactual switching out rates. For Coomeva, the effect is persistent for 1.5 years and then becomes statistically indistinguishable from zero at the 95% level. During the first three half-years relative to the start of cancer, the decrease in the probability of switching out of Coomeva amounts to between 30% and 50% of counterfactual switching out rates.



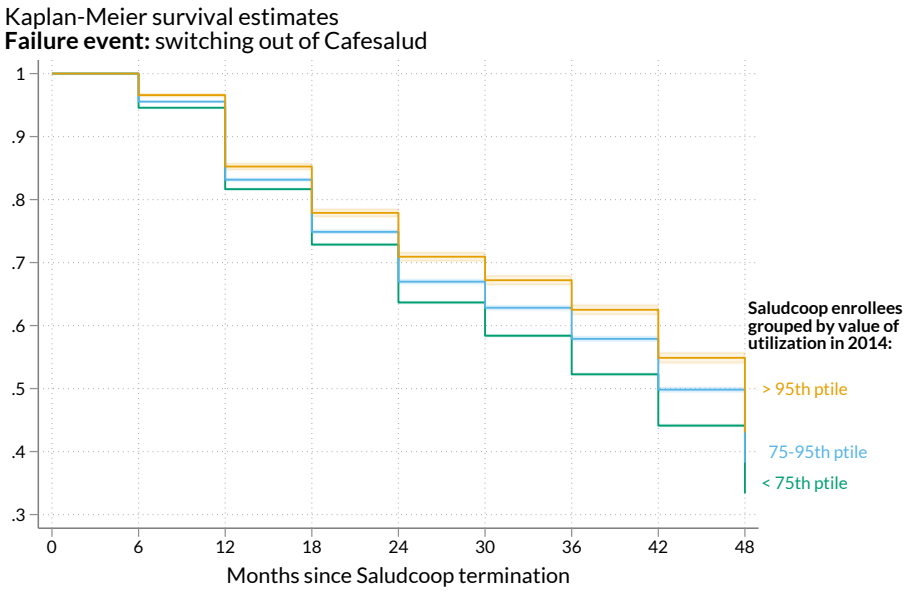
(A) 2015



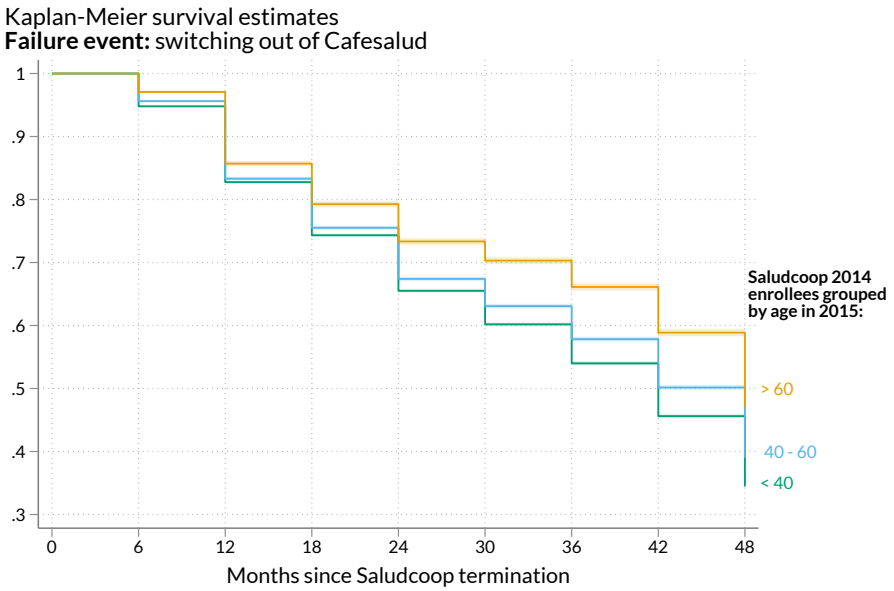
(B) 2016-2019

FIGURE II.5: Correlation Between Health Risk and Choice Persistence

This figure plots the estimated coefficients β_r and α_r from equation II.6, along with 95% confidence intervals. Each observation is an enrollee-half-year. Standard errors are clustered by individual. For each time period, the sample is restricted to individuals in the RC who are enrolled with one of the insurers in Urban sample. I use a 5% random sample of the universe of residents for these results.



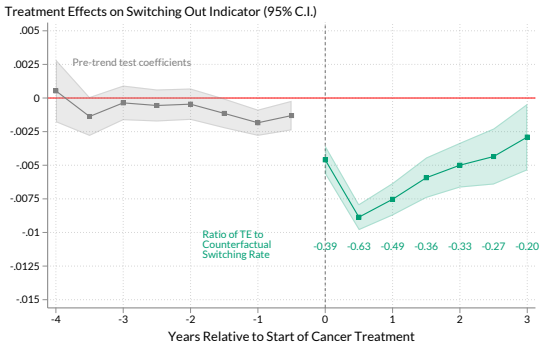
(A) Heterogeneity by Value of Utilization in 2014



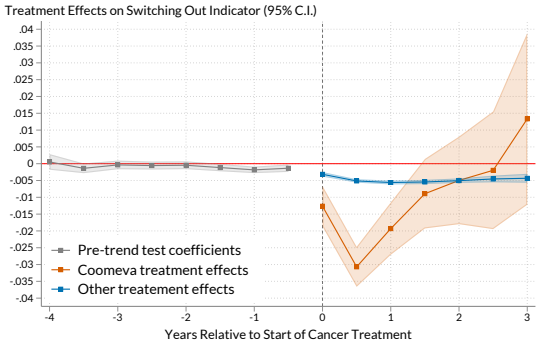
(B) Heterogeneity by Age

FIGURE II.6: Continued Enrollment in Cafesalud By Risk and Age

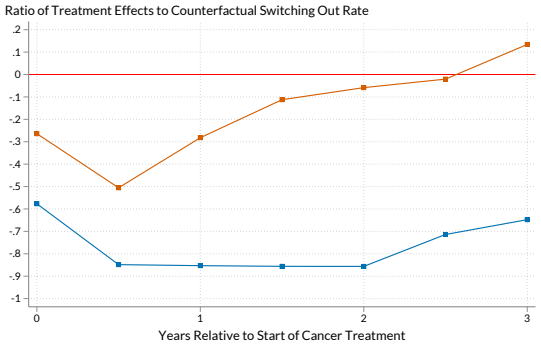
This figure shows Kaplan-Meier survival estimates for the group of enrollees that were enrolled with Saludcoop in June 2014 and who were enrolled in Cafesalud in December 2015. The failure event is defined as enrolling with a plan other than Cafesalud. The survival function is estimated by groups according to quantiles of the value of services used in 2014. The shaded areas around each darker line denote 95% confidence intervals (standard errors are calculated using the Greenwood formula).



(A) Average Treatment Effects (All Plans)



(B) Average Treatment Effects by Plan



(C) Ratio of τ_h^g to $\widehat{\text{Switch Out}}_h(0)$

FIGURE II.7: Event-Study Results for Cancer

Notes: This figure plots the main event-study results. Panels A and B show, in grey, the estimated coefficients of the pre-trend test corresponding to equation II.11. Panel A shows the estimated average treatment effects τ_h for each time horizon h after the start of cancer treatment, where the average is taken over all treated observations in each time horizon. Panel B shows the estimated average treatment effects τ_h^g for each time horizon h after the start of cancer treatment, where the average is taken separately for Coomeva (colored in red) and for all other plans in the sample (colored in blue). The shaded areas show 95% confidence intervals based on standard errors that are clustered at the individual level. Panel C shows the ratio between the estimated average treatment effects τ_h^g and the average counterfactual switching rate for each group at time horizon h. The counterfactual is constructed following the imputation approach described in the main text.

Chapter III

Quantifying Inertia Using Plan Demand Estimation Approach

Chapter II documented that health risk—both ex-ante and ex-post—is strongly correlated with choice persistence, and provided evidence of substantial differences in care provision across health plans, measured through average complaints and causal effects on medical care utilization. These findings are consistent with health risk driving consumer inertia. However, they do not, by themselves, rule out an alternative explanation: that there are other plan characteristics that rationalize individuals' choices to remain in their current plan, even when that plan performs poorly on care provision.

The objective of this chapter is to quantify the magnitude of consumer inertia by asking: how much access to medical care are individuals giving up by not switching to better plans, after accounting for other important plan characteristics? The central econometric challenge is to separately identify inertia from persistent unobserved preference heterogeneity, a well-known identification problem in the literature on state-dependence and switching costs (Heckman, 1978; Farrell and Klemperer, 2007; Handel, 2013; Polyakova, 2016; Ho et al., 2017) To address this, we exploit the choices of new enrollees—individuals who, by construction, face no switching frictions—and allow for flexible, plan-specific unobserved preferences in the model that depend on rich individual characteristics.

Our approach is to estimate a discrete choice model of health plan demand, in which consumers choose between plans that differ in the mean quantity of medical care they provide to individuals of their health type. This plan characteristic is not directly observed and must be estimated. To do so, I develop and estimate a model of medical

care provision using a machine learning algorithm trained on realized utilization data. This allows me to construct each plan's provision of mean medical care conditional on enrollee characteristics, which is the key plan characteristic in the choice model.

This approach is similar to that in Handel (2013). In that paper, the key input for the demand model is the distribution of out-of-pocket expenses a family faces under each of the alternative plans, which differ only by the financial protection they offer. The author exploits a policy change at the firm in which all employees were required to make an active plan choice in one period, which then became the default option in later periods. This change enables the clean identification of inertia separately from unobserved preference heterogeneity: preference heterogeneity is identified using choices in the active decision period, while inertia is identified based on plan switching behavior over time, as the predicted value of plans evolves due to price adjustments and changes in health status (which alter the value of the financial lotteries entailed by each plan).

The main methodological distinction relative to this dissertation is that Handel (2013) models plan choice within a standard framework of decision-making under uncertainty, where insurance options are treated as financial lotteries. In contrast, the approach here is to model the indirect utility of each plan in product-characteristic space, which is more similar to Polyakova (2016).[1] That paper also models the indirect utility of each plan and allows for rich observed and unobserved preference heterogeneity. While not a main focus of the analysis, the results in both papers find that some proxies for high-risk increase switching costs, consistent with the main results in this dissertation.

1 Model of Plan Choice

The choice of health plan is made by each individual i in each year t, conditional on the quantity of medical care they expect to receive at t from each plan $j \in \mathcal{J}_t$, where $\mathcal{J}_t = 1, 2, ..., J_t$ denotes the menu of available alternatives. We refer to these ex-ante expectations as *mean care provision* and denote them by $m_{ijt} \equiv \widehat{m}_{jt}(\mathbf{X}_{it})$, where $\mathbf{X}_{it}$ is a vector

[1] This is what Einav et al. (2010) call a contract valuation approach.

of individual observed characteristics. The interpretation of $\widehat{m}_{jt}(\mathbf{X}_{it})$ is that it captures the expected medical care that plan j would provide to an individual as a plan-time specific function of health type $\mathbf{X}_{it}$, which is a vector of observable characteristics. We assume that individuals can perfectly observe m_{ijt}. The empirical model used to obtain $\widehat{m}_{jt}(\mathbf{X}_{it})$ is presented in the next section.

Individuals choose the plan that maximizes their utility, which is given by

$$U_{ijt} = \alpha_{it} \log m_{ijt} + \eta_{it} \cdot \mathbb{1}[\text{Default}_{it} = j] + \xi_{ijt} + \varepsilon_{ijt} \tag{III.1}$$

$$\alpha_{it} = \alpha_0 + \pi^{\alpha} \mathbf{D}_{it}^{\alpha} \tag{III.2}$$

$$\xi_{ijt} = \gamma_{jt} + \pi_j^{\xi} \mathbf{D}_{it}^{\xi} + \nu_{ij} \tag{III.3}$$

$$\eta_{it} = \eta_0 + \pi^{\eta} \mathbf{D}_{it}^{\eta} \tag{III.4}$$

$$\varepsilon_{ijt} \sim \text{Type I EV} \tag{III.5}$$

$$\nu_{ij} \sim \mathcal{N}(0, \sigma_j) \tag{III.6}$$

Here, α_{it} and η_{it} capture heterogeneous preferences for mean care provision and for remaining with the default option, respectively. This way of modeling inertia interprets η_{it} as a utility premium for staying with the current plan—or, equivalently, as a switching cost that must be overcome for a consumer to change plans. The vectors $\mathbf{D}_{it}^{\alpha}$ and $\mathbf{D}_{it}^{\eta}$ contain the observable individual characteristics that these preferences depend on, with associated coefficients π^{α} and π^{η}. The term ξ_{ijt} captures preferences for all other (possibly unobserved) plan attributes, including provider networks, customer service, or convenience.[2] These are flexibly modeled using plan-by-time fixed effects γ_{jt} and interactions between plan indicators and demographics $\mathbf{D}_{it}^{\xi}$. Finally, ν_{ij} denotes a random, time-invariant preference shock for plan j, capturing unobserved individual-level tastes (e.g., proximity to providers), which we assume are normally distributed with standard deviation σ_j. [3]

[2]There is a large literature that includes network value as a key plan charcteristic. For example, Ho (2009b), Ho (2009a), and Ho and Lee (2019).

[3]To avoid underidentification, some of these components are excluded in estimation. Plan Nueva serves as the reference category.

We model utility as a function of the logarithm of expected medical care provision, $\log m_{ijt}$, rather than the level of m_{ijt} directly. This functional form captures diminishing marginal utility of medical care: as the provision of care increases, each additional unit provides less incremental utility. Using this transformation is also useful because it accounts for the large differences in baseline medical care need across individuals. For example, if α is constant, assuming that utility is linear in m_{ijt} would unrealistically imply that one additional unit of care has the same utility impact for a healthy and a high-risk individual. In contrast, the log specification allows α to reflect marginal utility in proportional terms, regardless of their baseline level of expected utilization.

In the baseline specification, we impose the following restrictions:

$$
\begin{aligned}
\alpha_{it} &= \alpha_{r(i,t)} \\
\xi_{ijt} &= \gamma_{jt} + \pi_j^{\xi} \mathbf{D}_{it} \\
\eta_{it} &= \eta_0 + \pi^{\eta} \mathbf{D}_{it} \\
\varepsilon_{ijt} &\overset{\text{i.i.d.}}{\sim} \text{Type I EV} \\
\mathbf{D}_{it} &: \text{indicators for risk-score bins, age bins, income bins, sex}
\end{aligned}
$$

The demographic variables included in the baseline specification allow preferences to vary by levels of ex-ante risk, age, income and sex. The term $r(i,t)$ denotes the ex-ante risk bin that i is in at time t. Following the approach in Chapter II, we measure individual risk with the prediction of mean care provision under a fixed reference plan (Nueva). The age and risk bins split individuals using the following percentile ranges: $< p20$, $p21 - p40$, $p41 - p60$, $p61 - p80$, $p81 - p90$, $p91 - p95$, $\geq p96$. The income bins correspond to three categories: low, medium, high.

Identification

The main econometric challenge in quantifying inertia lies in separating it from persistent unobserved preference heterogeneity. Our identification strategy exploits two features of

the institutional setting and data structure.

First, the central plan characteristic in our model—mean care provision—varies both across plans and across individuals. While we do not explicitly include other important plan features, such as provider networks or customer service quality, we flexibly control for them using plan-by-time intercepts and interactions between plan fixed effects and individual characteristics. These terms also absorb time-varying plan quality.

Second, the panel structure of the data, combined with the presence of new enrollees each year, allows us to identify inertia by leveraging within-individual variation in plan choices over time. Specifically, we account for time-invariant unobserved preferences for specific plans via the individual-by-plan random effects ν_{ij}, which capture persistent tastes–such as geographic proximity to a plan's provider network—that may drive inertia-like behavior in the absence of true switching frictions.

Our empirical approach follows closely the identification strategies used in prior work on inertia in health insurance markets (Handel, 2013; Polyakova, 2016; Ho et al., 2017). These studies estimate static discrete choice models using panel data and rely on periods of "active" choice to separately identify switching costs. Importantly, we assume a static model of choice: individuals are forward-looking only to the extent that expected care in the current period reflects their anticipated health needs. They do not account for the fact that their current choice becomes the default option in future periods, nor do they anticipate changes in their health status beyond the care they expect to use in the present. This simplifying assumption is common in the literature and can be interpreted as reflecting myopia.

A key limitation of our approach stems from the assumption that the idiosyncratic utility shocks ε_{ijt} are independently and identically distributed across individuals and alternatives. This assumption implies that unobserved determinants of plan utility are uncorrelated with the key regressor in the model—mean care provision m_{ijt}. In other words, we assume that, conditional on observables and random preference shocks, there are no unmeasured individual-level factors that simultaneously affect expected care uti-

lization and preferences over plans. This assumption is unlikely to hold exactly in our setting. While our measure of mean care provision flexibly captures plan-specific expected utilization based on a rich set of observables, it remains an imperfect proxy for health status and medical need. In particular, we reduce the multidimensional complexity of health to a single summary measure of risk, and then discretize that measure into bins for estimation. This effectively restricts unobserved health heterogeneity to operate only through mean care provision and its interaction with risk type. As a result, any residual variation in health that affects both expected utilization and utility from plan characteristics—such as preferences for specific providers or treatment styles stemming from particular diagnoses—will be absorbed into the logit error term, thereby violating the independence assumption.

This limitation could potentially bias the estimate of α_{it}, the marginal utility of care provision. While we attempt to mitigate this concern through the plan intercepts and their interactions, a fully satisfactory solution would require more granular health characteristics or an instrumental variables strategy, both of which are beyond the scope of the present analysis.

Another concern is that individuals who enter the contributory regime for the first time may be systematically different from the average enrollee. In our sample, new enrollees were previously enrolled in the subsidized regime (RS), a program that targets the poorest segments of the population. Eligibility for the subsidized regime is determined through a proxy means test that incorporates variables such as income, housing conditions, and access to public services. As a result, new entrants are likely to differ substantially from existing RC enrollees along a number of socioeconomic dimensions. These differences may affect both their preferences over plans and their underlying demand for care, raising concerns about the validity of estimates that rely heavily on their choices for identification. For this reason, we estimate the baseline model only including individuals who transitioned from the RS to the RC in the period 2015-2018. This restriction also leaves out SCM enrollees, as these plans didn't report claims after 2015.

2 Model of Care Provision

In this section, we present the model of m_{ijt} and the chosen estimation approach. The main idea is to model medical care using a Tweedie distribution and use a machine learning algorithm called Extreme Gradient Boosting (XGBoost) to predict mean medical expenditures under each plan as a function of rich individual observables that past health status and other determinants of the demand for medical care, including information from past diagnoses, age, sex, and past enrollment patterns. We start by discussing the main challenges that arise when estimating medical care utilization and how the proposed approach addresses them. We then present the model in detail, and describe how it is trained.

Challenges and solutions

There are a number of challenges in modeling medical care utilization:

1. **How to measure it from the claims data?** The cost reported for each activity in the claims data is a mix of the medical care expenditures paid by insurers for that activity, as well as imputations done by the MSPS for activities whose cost is originally reported as zero due to the activity being performed as part of a capitated contract or episode-based (bundled) payment. The reported insurer costs then, can be highly influenced by features of plan-provider agreements that do not necessarily capture how much care is being provided to an individual.[4] For this reason, we calculate the value of medical care utilization by inputting a standard price for each activity, using prescription codes (ATC) and procedure codes (CUPS). For the baseline model, we use the average price for each code across the full claims data for each year as the standard price.

2. **How to model the variation in medical care utilization across individuals?** As

[4] For example, two patients with the same medical care need could have the same procedure at the same provider on the same day and yet the reported cost can differ depending on their plan and the agreements they have with the provider. However, our measure of medical care should be the same for both.

shown in Figure III.1, the distribution of medical care utilization is highly right-skewed, with a small fraction of individuals accounting for a large proportion of total care. Individuals in the top 1% of medical care use account for 30.4% of total care, with the figure going up to 40% for the top 5%. Meanwhile, the bottom 50% only account for 3.6% of total care. It is also characterized by a large number of zeros, as shown in Panel (A) of Figure III.1. These features pose challenges for approaches like linear regression. As we explain in more detail below, the Tweedie distribution is well-suited for right-skewed, heavy-tailed, zero-inflated data, once some of its parameters are constrained.

3. **How to model interactions between the drivers of medical care utilization?** Medical care utilization is likely driven by complex interactions between demographics, past health status, preferences, and plan-specific factors. It is difficult to capture this complexity using a parametric approach to estimation, as it becomes computationally intensive to search for the most predictive interactions. Using XGBoost effectively addresses this challenge by using gradient boosting and decision trees to automatically capture complex, non-linear interactions and perform feature selection without requiring manual specification.

Model

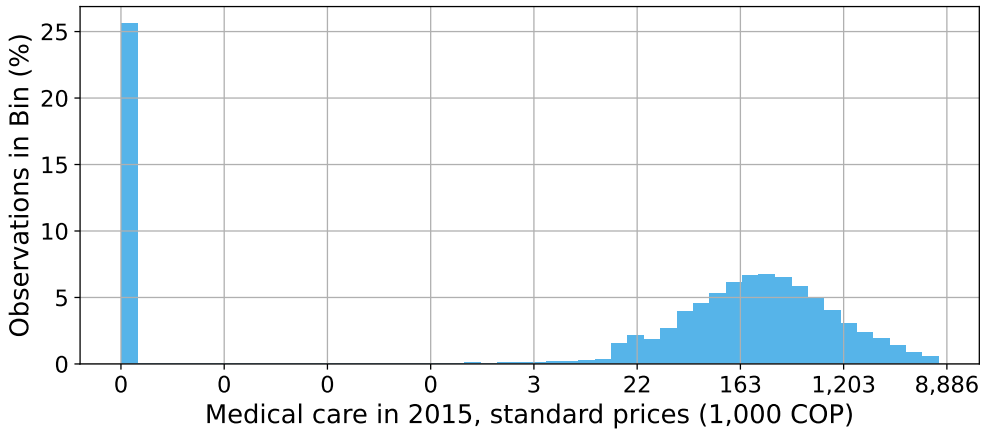
Let y_{ijt} denote the medical care utilization of individual i if they choose plan j at time t, and assume that it follows a Tweedie distribution:

$$y_{ijt} \sim \text{Tweedie}(m_{ijt}, \phi_{jt}, \rho_{jt}) \tag{III.7}$$

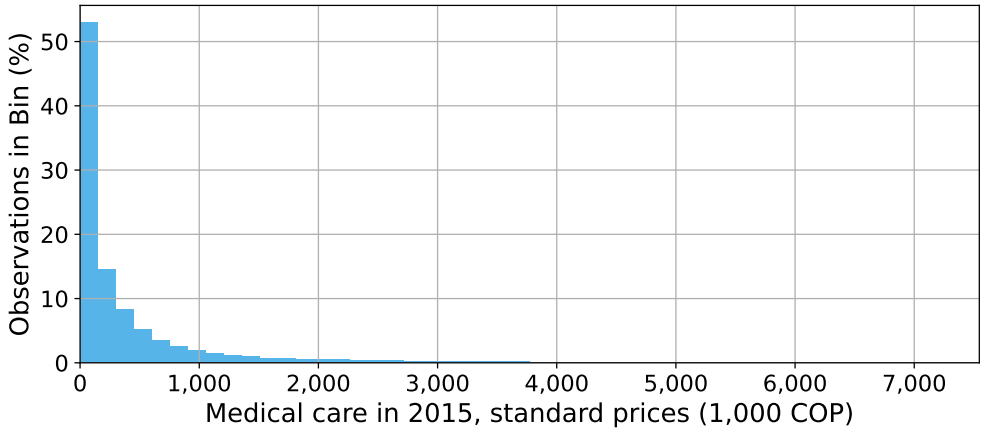
$$m_{ijt} \equiv E[y_{ijt}] = \exp\left(f_{jt}(\mathbf{X}_{it})\right) \tag{III.8}$$

$$\rho_{jt} \in (1, 2) \tag{III.9}$$

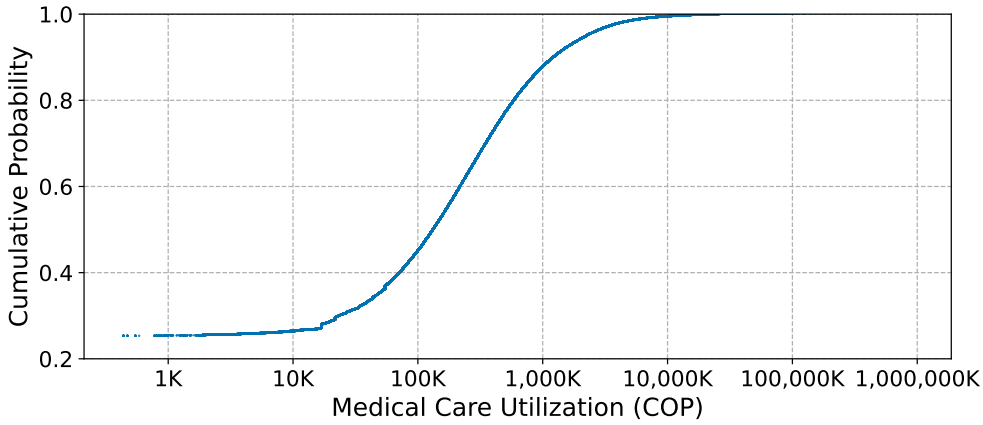
$$\phi_{jt} > 0 \tag{III.10}$$



(A) Logarithmic scale



(B) Natural scale



(C) Empirical CDF

FIGURE III.1: Distribution of Medical Care Utilization

Note: This figure shows the distribution of medical care utilization in 2015 calculated at standard prices, as described in the main text. The sample used corresponds to individuals in the holdout sample for model training. Panel (B) is restricted to those individuals below the 99 percentile. Number of individuals: 516,361.

Here, m_{ijt} is the mean of medical care expenditures, $\mathbf{X}_{it}$ is a vector of observed individual characteristics, $f_{jt}(\cdot)$ is a function linking these characteristics to the log of expected medical care utilizaiton. The parameters ρ_{jt} and ϕ_{jt} denote the Tweedie power and dispersion parameters, respectively. The restriction $\rho_{jt} \in (1, 2)$ implies that the distribution belongs to the Compound Poisson-Gamma family, a widely used specification in actuarial science to model the total value of insurance claims (Jørgensen, 1997).

The Tweedie distribution is particularly well suited to modeling medical spending data, as it naturally accommodates both a point mass at zero (for individuals with no utilization in a given period) and a positively skewed continuous distribution (for those who do use care). This eliminates the need to separately model the extensive and intensive margins of healthcare use. Moreover, the Tweedie has two key advantages. First, its variance takes the form $\text{Var}(y_{ijt}) = \phi_{jt} \cdot m_{ijt}^{\rho_{jt}}$, allowing for a flexible and transparent relationship between the mean and variance of spending. This is appropriate for healthcare applications, where higher expected costs are typically associated with greater variation in those costs too. Moreover, sums of independent Tweedie-distributed random variables with the same power parameter also follow a Tweedie distribution, making the model particularly convenient for aggregating predictions across individuals to the plan level.

The vector of covariates $\mathbf{X}_{it}$ includes a rich set of variables designed to capture observable determinants of the *demand* for medical care, drawing from both current and lagged individual characteristics. We include basic demographics such as sex, age, and municipality of residence, along with detailed enrollment information. The enrollment information allows us to control for factors that determine the demand for care, including length of enrollment spells, income (which determines cost-sharing and copayments, but also influences care demand directly), and the cost-sharing and copayment schedules enrollees face (which depend on enrollee type). Importantly, we do not include any variables that may capture plan characteristics. Instead, we estimate the model separately for each plan, allowing supply-side heterogeneity to be fully absorbed by plan-specific models. This strategy isolates individual-level predictors of demand while flexibly ac-

counting for differences in how plans translate enrollee characteristics into medical care.

Current enrollment variables include enrollee type (contributor, dependent, or additional beneficiary), enrollment status (active vs. suspended), total days enrolled during the year (exposure), and—for dependents—the number of days the main contributor was enrolled. The observables also include income bin indicators (low, medium, high), along with the contributor's income bin for dependent enrollees. In addition, we incorporate one-period lags of all enrollment variables, which help approximate enrollment status at the beginning of the yea, which is an important adjustment given that we observe enrollment only at half-yearly intervals.

To capture health status, we include a broad set of lagged indicators for diagnostic groups that capture chronic or other persistent conditions. These indicators are included separately for one-year and two-year lags, allowing the model to account for both recent and persistent health conditions. We intentionally exclude measures of intensity—such as the number of diagnoses or prescriptions—since these may be influenced by the supply of care, rather than underlying health needs. The goal is to isolate variation in the demand for medical care, conditional on observable risk, without conflating it with plan generosity or access. The full list of diagnoses groups can be found in the appendix.

The diagnostic data comes from the RIPS dataset, rather than from the Suficiencia claims data used to measure utilization and estimate plan effects. While RIPS is generally of lower data quality than Suficiencia—due to more frequent coding errors and missing values—it has substantially better coverage across plans and regimes. In particular, it includes health histories for individuals who were previously in the subsidized regime and later joined the contributory regime, a group that is critical for identifying switching costs. Because these individuals have no default plan assignment in the contributory regime, their choices are unconstrained by prior enrollment and provide the necessary variation for identifying switching costs.

Identification

The key identifying assumption of the medical care provision model is that the covariates in $\mathbf{X}_{it}$ are exogenous measures of underlying health status, reflecting true medical need rather than plan-driven variation in care delivery. In other words, we assume that—conditional on $\mathbf{X}_{it}$—differences in observed utilization reflect individual risk, not differences in medical care provision across plans.

There are three main reasons why this assumption may fail. First, it could be violated if lower-provision plans systematically under-diagnose their enrollees. In such cases, indicators for past diagnoses may understate true health needs, leading the model to underestimate the expected utilization of individuals coming from more restrictive plans. Second, the model may omit relevant demand-side factors that are correlated with both utilization and plan choice. For example, the baseline model does not include variables capturing distance to providers. If plans' provider networks vary significantly in geographic location, and if individuals have strong preferences for proximity, then our model may overestimate the expected care an individual would receive from a plan whose network is farther away than that of their current plan. Third, the model assumes away transition frictions, such as administrative barriers, referral delays, or informational frictions faced by switchers when navigating a potentially new provider network. These frictions could limit access to care even when individuals switch into plans with higher predicted provision.

Training

The key object of interest in the model is the function $f_{jt}(\mathbf{X}_{it})$, which maps observed individual characteristics to the log of expected medical care utilization if they choose plan j at time t. Because this function is likely to be complex and highly nonlinear—involving intricate interactions between the myriad determinants of the demand for medical care—we approximate it using a machine learning algorithm. Specifically, we use Extreme Gradient Boosting (XGBoost), a decision tree-based ensemble method that is well suited to

high-dimensional data and flexible function approximation (Chen and Guestrin, 2016).

With the restriction that $\rho_{jt} \in (1,2)$, the XGBoost algorithm minimizes a Tweedie-specific loss function derived from the Tweedie compound Poisson-Gamma model.[5] The outcome variable y_{it} is medical care utilization in year t, calculated at standard prices according to the following formula:

$$y_{it} = \sum_{a \in \mathcal{A}_{it}} \bar{P}_{c(a)} \tag{III.11}$$

Here, $\mathcal{A}_{it}$ denotes the set of activities i had in year t and $\bar{P}_{\text{code}(a)}$ is the average value of activities with code $c(a)$ across all plans in year t, where activities are identified by either their prescription code (ATC) or procedure code (CUPS).

As mentioned before, we train the models separately for each health plan in each year, using a framework called Optuna to fine-tune the hyper-parameters of the model, including the parameter ρ_{jt}. For each plan j in year t, we follow each of the steps below:

1. Restrict the sample to individuals enrolled with plan j in year t, where enrollment variables are measured at the end of June.

2. Randomly split the data into an 85% training sample and a 15% testing sample, which is reserved to evaluate out-of-sample model performance after hyperparameter tuning.

3. Perform hyperparameter optimization using Optuna on the training sample. Each Optuna trial executes the following steps:

   (a) Randomly split the training sample again into a 90% sub-training sample and a 10% validation sample

   (b) Train an XGBoost model on the sub-training set using the Tweedie loss function and the current set of candidate hyperparameters.

   (c) Predict outcomes on the 10% validation set.

[5] Further details on the Tweedie distribution can be found in Jørgensen (1997), and on the mechanics of computing its density in Dunn and Smyth (2005).

(d) Compute the mean Tweedie deviance (Optuna minimizes searches the hyper-parameters that minimize this metric)

4. Train the model using the optimal hyper-parameters.

5. The trained model is applied to obtain $\widehat{m}_{jt}(\mathbf{X}_{it})$ for all enrolled individuals at time t, not only the ones enrolled in j. The predicted value for individual i is denoted by $\widehat{m}_{jt}(\mathbf{X}_{it})$.

We use Optuna to tune the following hyperparameters for each plan-specific model:

TABLE III.1: XGBoost Hyperparameters for Tweedie Regression

Hyperparameter	**Description**	**Tuning Range**
`learning_rate`	Step size shrinkage to prevent overfitting	$(0.001, 0.5)$
`max_depth`	Maximum depth of trees (higher = more complex)	$(3, 20)$
`subsample`	Fraction of data used in each boosting round	$(0.2, 1.0)$
`colsample_bytree`	Fraction of features used per tree	$(0.2, 1.0)$
`lambda`	L2 regularization (Ridge)	$(10^{-5}, 10)$
`alpha`	L1 regularization (Lasso)	$(10^{-5}, 10)$
`gamma`	Minimum loss reduction for tree split	$(0, 10)$
`min_child_weight`	Minimum sum of instance weights for leaf nodes	$(0.1, 10)$
`max_delta_step`	Maximum step size for weight update	$(0, 10)$
`tweedie_variance_power`	Controls variance function in Tweedie loss	$(1.45, 1.95)$

3 Results

This section presents the main empirical results. We begin by evaluating the performance of the medical care provision model and then turn to the results of the health plan demand model.

3.1 Results - Medical Care Provision

Model evaluation

We use the 15% testing sample to assess the performance of the model. The model prediction for individual i is given by

TABLE III.2: Predictive Ratios by Decile (2015)

Prediction decile	Predicted Utilization in COP	Actual Utilization in COP	Predictive Ratio
1	100,797	113,699	0.887
2	179,593	190,375	0.943
3	228,170	243,030	0.939
4	280,807	295,857	0.949
5	345,867	363,900	0.950
6	435,501	474,790	0.917
7	557,822	574,508	0.971
8	726,891	770,576	0.943
9	1,070,325	1,138,655	0.940
10	2,308,124	2,529,757	0.912
All	623,392	669,517	0.931

Note: This table shows the predictive ratios of the baseline model by percentile groups, where the percentiles are calculated for the value of the predicitons. The outcome variable is medical care utilization in 2015 calculated at standard prices in current COP, as explained in the main text. The sample used corresponds to the main validation sample described in the text. Number of individuals in the validation sample: 538,932.

$$\widehat{y}_i \equiv \sum_j \widehat{m}_j(\theta_i) \times 1[D_{ij} = 1] \tag{III.12}$$

be the model's prediction for individual i, where D_{ij} is an indicator function for individual i enrolling with plan j in 2015. To assess the performance of the model, a useful statistic is the predictive ratio R, which is defined as the ratio between average model predictions and average realized medical care utilization:

$$R = \frac{\frac{1}{N}\sum_i \widehat{y}_i}{\frac{1}{N}\sum_i y_i} \tag{III.13}$$

Tables III.2, III.3, III.4 show the predictive ratios for each year in the sample, calculated for each decile of the distribution of $\widehat{y}_i$. These ratios suggest a robust model fit that reasonably captures both common and extreme cost levels. However, the model exhibits a consistent tendency to under-predict the outcome variable overall, signaling an area for potential improvement. This under-prediction is also reflected in the smaller predictive ratios observed in the first and tenth deciles.

TABLE III.3: Predictive Ratios by Decile (2016)

Prediction decile	Predicted Utilization in COP	Actual Utilization in COP	Predictive Ratio
1	139,390	159,293	0.875
2	226,911	211,198	1.074
3	284,354	290,257	0.980
4	338,733	338,198	1.002
5	402,143	413,249	0.973
6	509,727	520,035	0.980
7	653,613	664,086	0.984
8	819,950	869,222	0.943
9	1,185,626	1,291,183	0.918
10	2,493,717	2,808,249	0.888
All	705,416	756,497	0.932

Note: This table shows the predictive ratios of the baseline model by percentile groups, where the percentiles are calculated for the value of the predicitons. The outcome variable is medical care utilization in 2016 calculated at standard prices in current COP, as explained in the main text. The sample used corresponds to the main validation sample described in the text. Number of individuals in the validation sample: 568,880.

TABLE III.4: Predictive Ratios by Decile (2017)

Prediction decile	Predicted Utilization in COP	Actual Utilization in COP	Predictive Ratio
1	131,905	152,129	0.867
2	214,952	215,754	0.996
3	272,192	289,228	0.941
4	328,799	339,884	0.967
5	397,310	440,116	0.903
6	495,586	545,344	0.909
7	615,079	639,031	0.963
8	803,765	871,851	0.922
9	1,190,589	1,293,094	0.921
10	2,649,369	3,002,274	0.882
All	709,956	778,872	0.912

Note: This table shows the predictive ratios of the baseline model by percentile groups, where the percentiles are calculated for the value of the predicitons. The outcome variable is medical care utilization in 2017 calculated at standard prices in current COP, as explained in the main text. The sample used corresponds to the main validation sample described in the text. Number of individuals in the validation sample: 594,427.

3.2 Results - Demand Estimation

Table III.5 presents the parameter estimates from the baseline demand model, which we estimate excluding the random coefficients. The coefficients capture individual preferences for mean care provision and for remaining with the default option, and how these preferences vary with observable characteristics such as risk, age, income, and gender. We begin by discussing the estimated parameters and the patterns of heterogeneity they reveal. We then use these estimates to quantify the trade-off between mean care provision and plan persistence—that is, to measure the magnitude of switching costs implied by the model.

Preferences for mean care provision are allowed to vary across risk bins. The estimated coefficients exhibit a clear downward gradient: individuals with lower ex-ante risk are significantly more responsive to differences in expected care provision. For example, those in the lowest risk bin—i.e., the healthiest 20%—have an α of 1.97, whereas the individuals in the highest risk bin—the riskiest 5%— have an estimated α of just 0.30.

Turning to the inertia parameters, the estimate for the baseline coefficient η_0 is precisely estimated at 5.77, suggesting that the value of choosing the default is high compared to care provision. The model allows this inertia to vary by observable characteristics. The coefficient for the high income bin is -0.94, implying an inertia coefficient of 4.83 for this group. Medium-income individuals also exhibit a lower inertia coefficient, with an estimated inertia parameter of 5.54, although this difference is smaller in magnitude and not very precise. In contrast, inertia is higher among women. The estimated coefficient on the female indicator is 0.40, implying that, conditional on observables, women face higher switching costs than men.

The third column of Table III.5 reports how the inertia coefficient varies across ex-ante risk levels. The estimates suggest that the probability of choosing the default decreases with risk, except for the very highest risk group. For example, individuals in the 21st–40th percentile of the risk distribution have a coefficient of -0.47, indicating a lower utility from remaining with the default compared to healthiest 20%. This pattern contin-

ues through the 41st–60th (-0.95), 61st–80th (-1.39), and 81st–90th percentile bins (-1.68), reaching a minimum at the 91st–95th percentile (-1.92). These estimates imply that individuals with higher predicted health expenditures would be less likely to choose the default. This decreasing trend reverses in the highest risk bin. For individuals above the 95th percentile of risk, the coefficient is -0.99, suggesting a higher relative likelihood of remaining in the default plan compared to individuals in the adjacent (91st–95th) risk group. While still less likely to stay with the default than the healthiest group, these individuals exhibit less active choice behavior than their slightly less risky counterparts. One plausible explanation is that individuals with extremely high health needs may face constraints related to provider continuity or administrative burdens that limit their ability or willingness to switch.

The final column of Table III.5 reports how the utility associated with remaining in the default plan varies across age groups. The omitted category corresponds to the youngest individuals, those below the 20th percentile of the age distribution. The estimated coefficients show a steep increase in the utility associated with remaining in the default plan for the oldest groups. Compared to the youngest 20%, those in the 81st–90th percentile of the age distribution are significantly more likely to remain with the default plan, with a coefficient of 0.42. This difference grows larger for the 91st–95th percentile group, whose estimated difference is 0.82, and reaches 1.34 among the oldest 5% of individuals in the sample. These results suggest that the oldest are markedly less likely to switch plans relative to younger enrollees, even after controlling for income, risk, and gender. While the coefficients for younger and middle-aged individuals are small and imprecise, the estimates for the oldest age groups are larger and statistically meaningful. The pattern is consistent with age-related differences in cognitive frictions, administrative burden, or preferences for continuity in care. These findings highlight age as an important factor behind choice persistence.

To interpret the estimated utility parameters in terms of economic behavior, we now turn to the implied switching costs faced by individuals across the population. We focus on the statistic $\frac{\eta_{it}}{\alpha_{it}}$, which captures the log increase in mean care provision that an

individual would require to be indifferent between remaining in their default plan and switching to an alternative that offers the same non-care characteristics.[6]. This measure provides a tractable and interpretable summary of inertia: higher values indicate that individuals require larger improvements in expected medical care to justify switching, and therefore face higher effective switching costs.

We examine this measure across combinations of age and risk bins. These two dimensions are strongly correlated in the data but may influence plan choice through distinct channels. While health risk proxies expected utilization, age may capture non-health-related switching frictions such as cognitive costs or higher preferences for continuity of providers. By jointly stratifying the population along both dimensions, we can explore how switching costs vary by age holding risk constant, and vice versa.

Table III.6 reports the average value of the statistic $\frac{\eta_{it}}{\alpha_{it}}$ across combinations of age and risk bins. In general, the estimated switching costs are very large across the board. The values of $\frac{\eta_{it}}{\alpha_{it}}$ frequently exceed 4 or 5, implying that individuals require multi-fold increases in expected care provision to justify switching away from their default plan.

Two main patterns emerge from the table. First, holding risk fixed, switching costs tend to increase with age. For example, among individuals in the 41st–60th risk bin, the switching cost rises from 4.06 for those in p41-p60 to 5.63 for those aged $\geq$ p96. This gradient suggests that age captures non-health-related barriers to active choice, and is consistent with past work that documents the choice frictions faced by the elderly (e.g., Abaluck and Gruber, 2011, 2016). Second, holding age fixed, the relationship between risk and switching costs is non-monotonic. The healthiest individuals (¡p20) consistently exhibit the lowest switching costs, typically around 3, suggesting they are more responsive to differences in mean care. In contrast, switching costs are higher and relatively flat across the middle of the risk distribution, and then spike dramatically in the riskiest 5%. For example, among the oldest 5% of individuals, switching costs rise from 3.56 in the lowest risk group to 21.15 in the highest. This steep rise at the top suggests that the

[6]Note that the medical care m^* that would make a consumer indifferent between switching and staying with a plan that offers them m units of mean care, holding all other plan characteristics constant, is implicitly defined by $\alpha_{it} \log m^* - \alpha_{it} \log m - \eta_{it} = 0$, so that $\frac{\eta_{it}}{\alpha_{it}} = \log m^* - \log m$

sickest individuals face near-complete inertia, requiring implausibly large increases in expected care to be induced to switch.

These findings highlight a key insight: inertia is not only widespread but most acute among those who have the most to gain from better plan access. While healthier individuals exhibit greater responsiveness, the oldest and sickest enrollees—despite facing the highest potential costs from poor plan quality—are effectively locked into their defaults.

TABLE III.5: Demand Estimation Results

Care Provision		**Inertia**		**Inertia: Risk**		**Inertia: Age**	
Parameter	Coefficient (SE)	Parameter	Coefficient (SE)	Parameter	Coefficient (SE)	Parameter	Coefficient (SE)
$\alpha_{\text{Risk bin: <p20}}$	1.9663	η_0	5.7673	$\pi^{\eta}_{\text{Risk bin: <p20}}$	—	$\pi^{\eta}_{\text{Age bin: <p20}}$	—
	(0.0937)		(0.3004)				
$\alpha_{\text{Risk bin: p21-p40}}$	0.8793	$\pi^{\eta}_{\text{Income bin: medium}}$	-0.2294	$\pi^{\eta}_{\text{Risk bin: p21-p40}}$	-0.6631	$\pi^{\eta}_{\text{Age bin: p21-p-40}}$	-0.1740
	(0.0310)		(0.1461)		(0.3047)		(0.2048)
$\alpha_{\text{Risk bin: p41-p60}}$	1.1221	$\pi^{\eta}_{\text{Income bin: high}}$	-0.9363	$\pi^{\eta}_{\text{Risk bin: p41-p60}}$	-0.9250	$\pi^{\eta}_{\text{Age bin: p41-p60}}$	-0.0914
	(0.0336)		(0.3523)		(0.2985)		(0.2252)
$\alpha_{\text{Risk bin: p61-p80}}$	1.2299	$\pi^{\eta}_{\text{Female}}$	0.3976	$\pi^{\eta}_{\text{Risk bin: p61-p80}}$	-1.3568	$\pi^{\eta}_{\text{Age bin: p61-p80}}$	0.1458
	(0.0378)		(0.1397)		(0.3084)		(0.2295)
$\alpha_{\text{Risk bin: p81-p90}}$	1.1034			$\pi^{\eta}_{\text{Risk bin: p81-p90}}$	-1.6392	$\pi^{\eta}_{\text{Age bin: p81-p90}}$	0.4174
	(0.0475)				(0.3334)		(0.2632)
$\alpha_{\text{Risk bin: p91-p95}}$	0.9769			$\pi^{\eta}_{\text{Risk bin: p91-p95}}$	-1.9222	$\pi^{\eta}_{\text{Age bin: p91-p95}}$	0.8246
	(0.0659)				(0.3923)		(0.3704)
$\alpha_{\text{Risk bin: } \geq \text{p96}}$	0.2952			$\pi^{\eta}_{\text{Risk bin: } \geq \text{p96}}$	-0.9927	$\pi^{\eta}_{\text{Age bin: } \geq \text{p96}}$	1.3375
	(0.0458)				(0.4476)		(0.4366)

Notes: This table reports estimates from the baseline demand model, obtained via standard maximum likelihood estimation. Standard errors (reported in parentheses) are clustered at the individual level. The estimation sample includes the years 2015 to 2017 and is restricted to individuals who live in Bogota and transitioned from the Régimen Subsidiado (RS) to the Régimen Contributivo (RC) exactly once during that period.

TABLE III.6: Estimated Switching Costs By Risk and Age

	Age bin						
Risk bin	<p20	p21-p40	p41-p60	p61-p80	p81-p90	p91-p95	≥ p96
<p20	3.04	2.90	2.90	3.03	3.17	3.35	3.56
p21-p-40	5.89	5.52	5.51	5.76	6.39	6.79	7.37
p41-p-60	4.48	4.13	4.06	4.32	4.61	5.03	5.63
p61-p80	3.80	3.58	3.48	3.67	3.99	4.43	4.77
p81-p90	3.91	3.78	3.82	4.08	4.28	4.50	4.99
p91-p95	4.21	3.95	4.06	4.32	4.63	4.80	5.38
≥ p96	16.70	16.21	16.40	16.86	18.31	19.69	21.15

Notes: This table reports the average $\frac{\eta_{it}}{\alpha_{it}}$ within groups that are defined by the combination of risk and age bins. The sample used corresponds to the 5% random sample used for the estimation, and we restrict to $t = 2015$. The statistic for each individual is calculated using the parameters of the baseline demand model.

Chapter IV

Implications for Quality Provision

Building on the evidence from Chapters II and III that health risk—both ex-ante and ex-post—is strongly correlated with choice persistence, and that switching costs are highest for the sickest 5%, this section examines how increased switching from Cafesalud and Coomeva after 2016 reshaped the risk composition of health plans, and the consequences of this shift. The analysis focuses on three dimensions of this reallocation: the contribution of switchers to total enrollment, their impact on the receiving plans' average risk, and their profitability. The findings reveal a tension: while the terminated plans experienced a deterioration in average risk as healthier enrollees exited, the plans that received these switchers saw an inflow of individuals who were sicker than their existing populations. This tension is resolved by observing that the terminated plans had a riskier pool of enrolees from the outset.

The chapter is structured as follows. Section 1 shows that switchers constituted a substantial portion of total enrollment in the plans that received them, with considerable heterogeneity across plans in the scale of these inflows. The second section analyzes the relative profitability of these switchers by comparing their medical care utilization to that of enrollees who belong to the same plan (after the switch) and capitation bin, revealing that switchers imposed persistently higher costs. The third section documents the increased in average risk in the terminated plans and the plans that received them by focusing on the subset of individuals who were already enrolled in 2015. These findings underscore how the correlation between risk and choice persistence, combined with

coarse risk adjustment, may have been a driving factor of the financial instability of the terminated plans.

1 Switchers' Contribution to Plan Enrollment

This subsection documents how the sharp increase in switching rates after 2016 translated into heterogeneous changes in enrollment across health plans. As discussed in Chapter II, switching rates rose markedly during the 2016–2019 period, especially relative to the low levels observed in the preceding two years (Figure I.1). During this period, approximately 20.8 % of individuals in the RC switched plans at least once, and an additional 1.5 % switched more than once.[1] These elevated switching rates generated substantial enrollment inflows for most plans, though the magnitude of these inflows varied considerably.

Figure IV.1 shows the contribution of switchers to total enrollment in the RC for the subset of plans that reported claims data during 2015–2019. Panel A plots the share of individuals who switched into a reporting plan in each half-year, disaggregated by the insurance program from which they originated. The share of "switchers in" from the RC begins to rise sharply in 2016h2, and continues increasing through 2019. In the second half of 2019, over 7% of current enrollment in these plans consisted of individuals who had switched in during that six-month period alone. Panel B displays the cumulative share of enrollment accounted for by individuals who had ever switched in since 2014. By the end of the sample period, more than 22% of enrollees in the reporting plans had switched in from another RC plan.

Although the focus of this chapter is on switching within the RC, the figure also shows that there was a steady, albeit smaller, flow of individuals into the RC from the subsidized regime (RS). These RS-to-RC switches accounted for between 1.5% and 2.5% of enrollment in each half-year over most of the period. Their contribution to cumulative

[1] Enrollment is observed semiannually. As a result, switches occurring between snapshots may be missed if individuals are temporarily absent from the enrollment records.

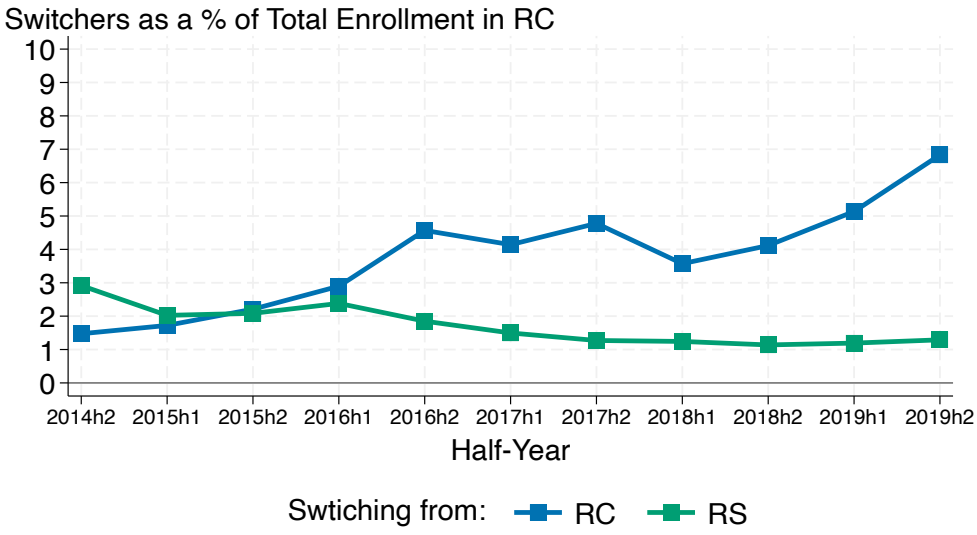
enrollment was nontrivial—reaching about 7.5% by the end of 2019—but remained far smaller than that of within-RC switches. These flows reflect transitions into the contributory system (e.g., due to formal employment).

To further understand the sources of these enrollment inflows, Figure IV.2 disaggregates the RC switchers in Figure IV.1 by plan of origin, grouping them into SaludCoop/Cafesalud/Me (SCM), Coomeva, and all other plans. Importantly, this classification includes all origin plans in the RC, not just those in the claims-reporting sample. Panel A shows the share of current enrollment composed of individuals who switched in from each group in a given half-year. The sharpest spike corresponds to 2016h2, when switchers from SCM accounted for almost 3% of enrollment in reporting plans during that semester alone. This surge followed the end of the 3-month period in which reassigned enrollees were not allowed to switch out of Cafesalud. While switching from Coomeva also rose steadily over time, its magnitude remained lower and its increase is more gradual. Notably, the contribution to enrollment of switchers from other plans is also substantial, higher than 1% in every semester.[2] Panel B, which aggregates all switchers since 2014, shows that by the end of 2019 nearly 10% of current enrollees in reporting plans had originated from SCM, compared to just above 4% from Coomeva and approximately 9.5% from all other plans combined.

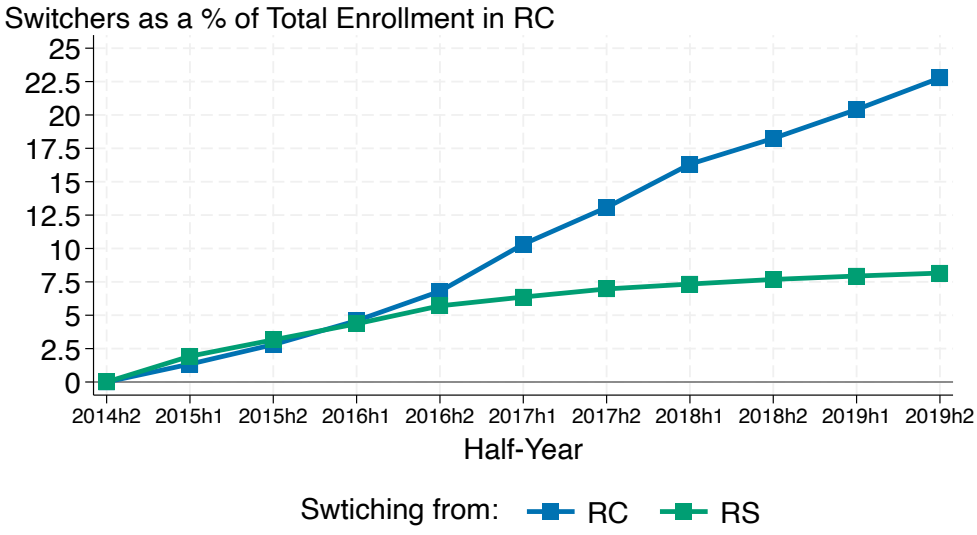
Together, Figures IV.1 and IV.2 confirm that switching became a dominant force in reshaping the enrollment of the reporting plans. Moreover, they underscore the disproportionate role played by terminated plans—particularly SCM—in driving this reallocation.

To understand whether some plans were more exposed than others to these enrollment flows, Figures IV.2 and IV.4 disaggregate the trends in Figure IV.1 by receiving plan. This plan-level perspective is crucial for identifying which insurers bore the brunt of the reallocation and for, subsequently, interpreting the consequences for plan risk composition. As before, switchers are distinguished by whether they came from another RC plan or from the RS.

[2]The sharp increase in 2019H2 is due to the termination of a plan called Cruz Blanca that operated mainly in Bogota.



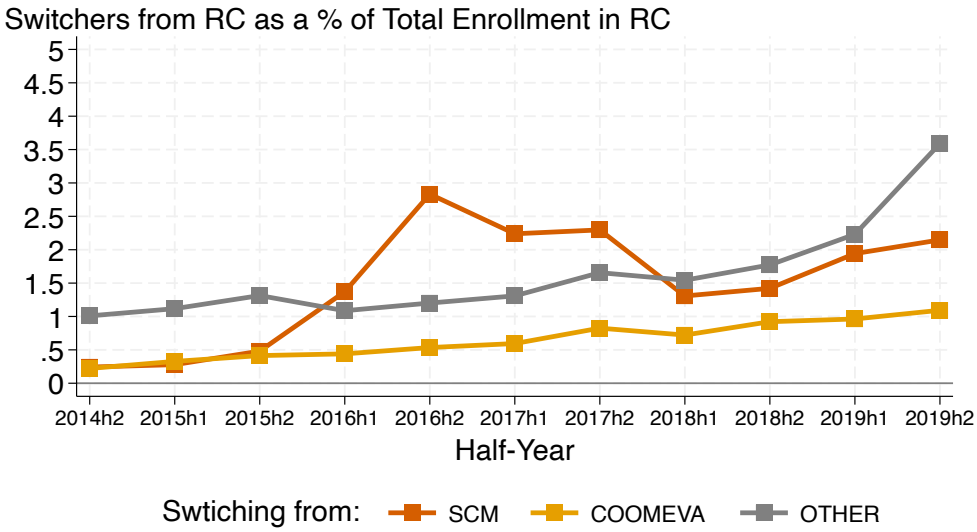
(A) Current Switchers



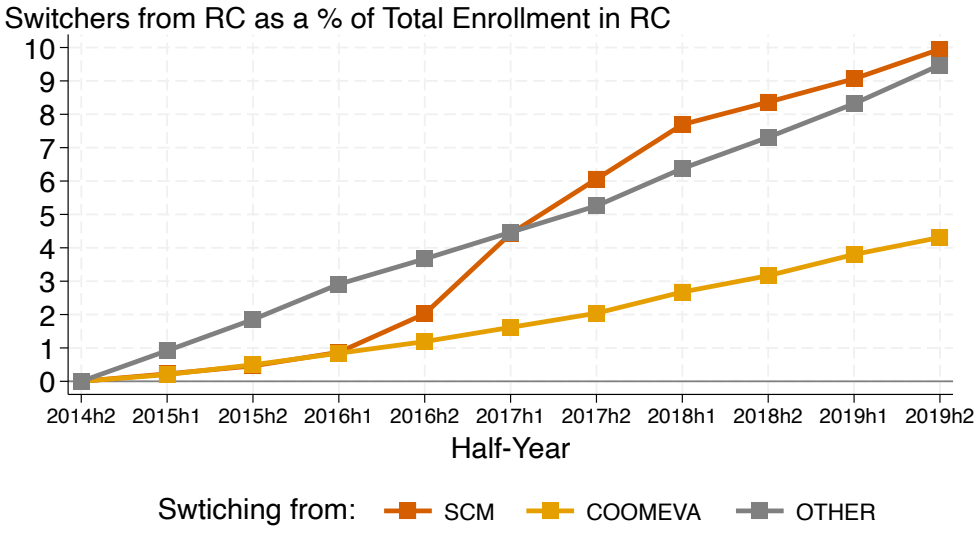
(B) All Switchers in 2014-2019

FIGURE IV.1: Importance of Switchers to RC Enrollment (Grouped by Program of Origin)

Note: This figure plots the contribution of switchers in to total enrollment at the plans in the main sample for the period 2014-2019. Switchers are grouped by the insurance program they are switching from (RS or TC). Panel A show the total numbers of "Switchers In" in each half year as a percentage of total enrollment in the plans in that half-year. Panel B shows the cumulative number of "Switchers In" since 2014 as a percentage of current enrollment in each half year. Switches from Saludcoop to Cafesalud are not counted.



(A) Current Switchers



(B) All Switchers in 2014-2019

FIGURE IV.2: Importance of Switchers to RC Enrollment (Grouped by Plan of Origin)

Note: This figure plots the contribution of "Switchers In" to total enrollment for all plans in the main sample for the period 2014-2019. Switchers are grouped by the insurance program they are switching from (Saludcoop-Medimas-Cafesalud, Coomeva, or Other). Panel A shows the total number of "Switchers In" in each half year as a percentage of total enrollment in the plans in that half-year. Panel B shows the cumulative number of "Switchers In" since 2014 as a percentage of current enrollment in each half year. Switching from Saludcoop to Cafesalud is not counted a a switch.

The variation across plans is stark. For Salud Total, Sanitas, Compensar, Sura, and Famisanar, switchers from the RC represent a growing share of current enrollment between 2016 and 2019, peaking above 10% in some semesters. In contrast, Coomeva and SOS absorbed fewer switchers over time, and in some cases even saw their share of incoming RC switchers decline. Nueva stands out with a delayed but sharp increase at the end of the period. Across all plans, RS switchers contributed a much smaller and more stable share of enrollment, generally remaining below 3% per semester.

These differences compound over time. By the end of 2019, RC switchers made up around 30% of enrollment or higher at Sura and Sanitas, and of around 15% or higher at Famisanar, Salud Total and Compensar. In contrast, cumulative shares remained below 15% at Nueva,, Coomeva and SOS. These patterns are likely a result of factors that drive individual choice—plan reputation, quality, distance to providers—but also factors like geographic presence and administrative reassignments in certain regions for other plans (e.g., Cruz Blanca).[3] The fact that a small subset of plans absorbed a disproportionate share of switchers is particularly relevant in light of the findings that follow.

To explore the role of origin plans, Figures IV.5 and IV.6 break down switchers at the plan level by origin—SCM, Coomeva, or other. These figures serve as the plan-level counterpart to Figure IV.2 and help identify which collapsed plans drove the inflows to each destination.

The largest spikes in enrollment at around 2016h2–2017h1 were driven by enrollees exiting SCM at all plans. For example, in Sanitas, more than 6% of current enrollment in 2016h2 came from SCM alone. Flows from Coomeva were more heterogeneous across plans: Salud Total, Sanitas, and Sura saw large increases in their contribution to enrollment, but this is not the case for the rest of plans.

These differences in origin composition also accumulated over time. By the end of 2019, over 16% of enrollment at Sanitas and around 10% at Salud Total, Sura, and Compensar came from former SCM enrollees. The figure for Coomeva is smaller but still

[3]For example, Coomeva ceased operating in some non-urban municipalities. It is possible that these reassignments affected enrollment flows in urban municipalities due to imperfect recording of municipality of residence.

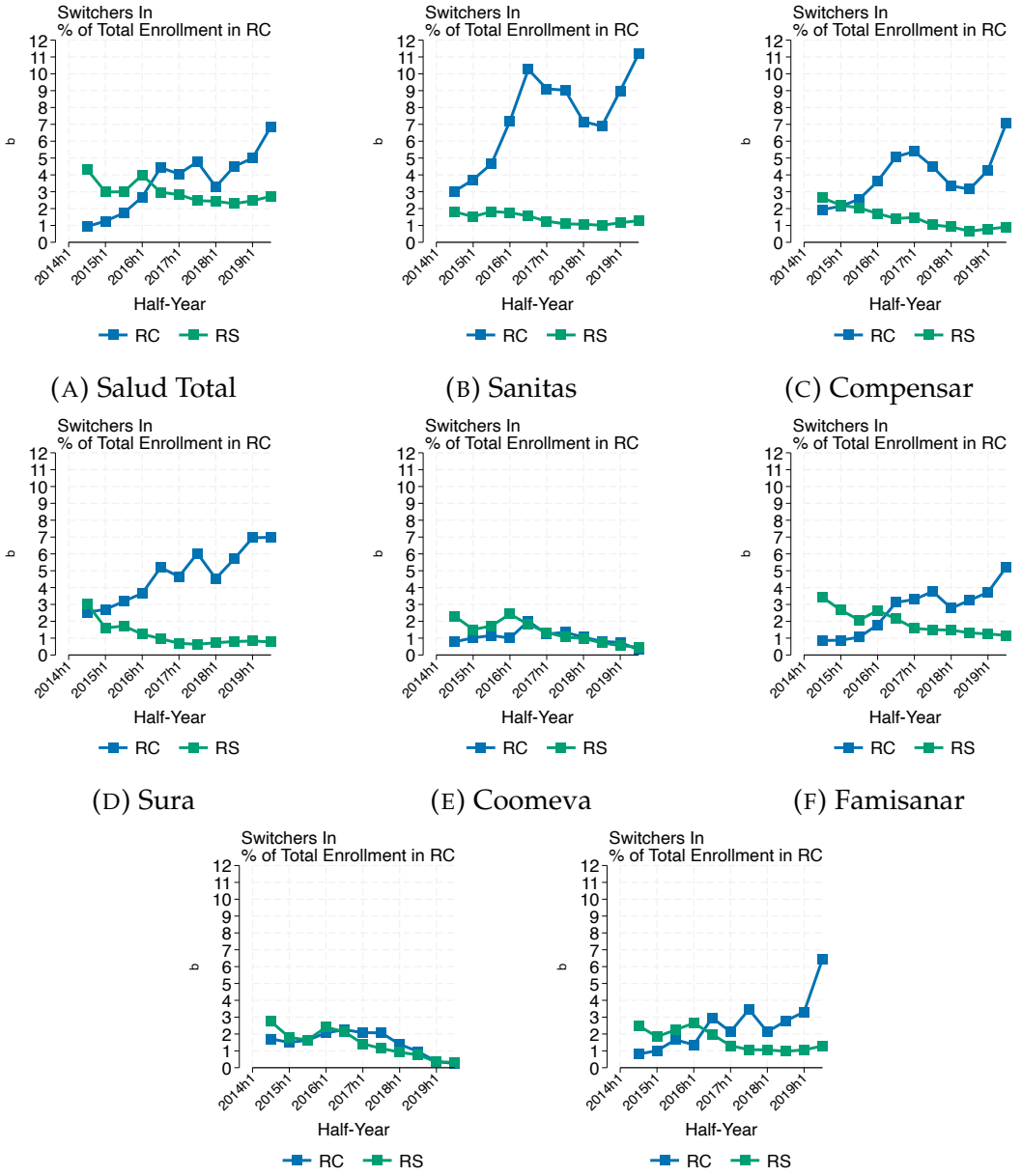
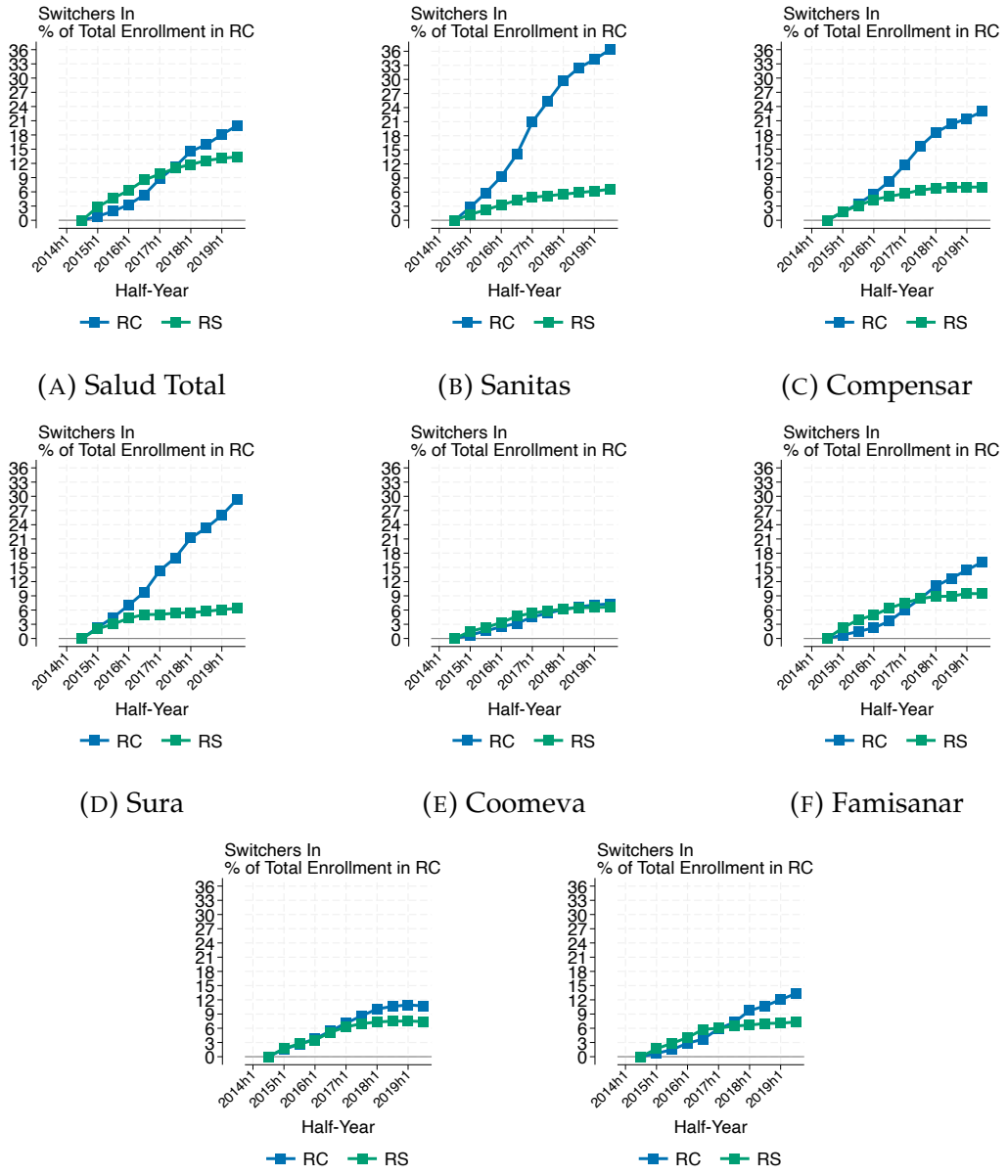


FIGURE IV.3: Importance of Switchers to Each Plan's Enrollment (By Program)

Note: This figure plots the contribution of Switchers In to the enrollment of each plan in the main sample for the period 2014-2019. Switchers are grouped by the program they are switching from (RS or RC). Each plot shows the number of "Switchers In" for each half year as a percentage of plan enrollment in that half-year.



(A) Salud Total (B) Sanitas (C) Compensar

(D) Sura (E) Coomeva (F) Famisanar

(G) SOS (H) Nueva

FIGURE IV.4: Cumulative Importance of Switchers to Each Plan's Enrollment (By Program)

Note: This figure plots the contribution of Switchers In to the enrollment of each plan in the main sample for the period 2014-2019. Switchers are grouped by the program they are switching from (RS or RC). Each plot shows the cumulative number of "Switchers In" since 2014 as a percentage of plan enrollment in each half-year.

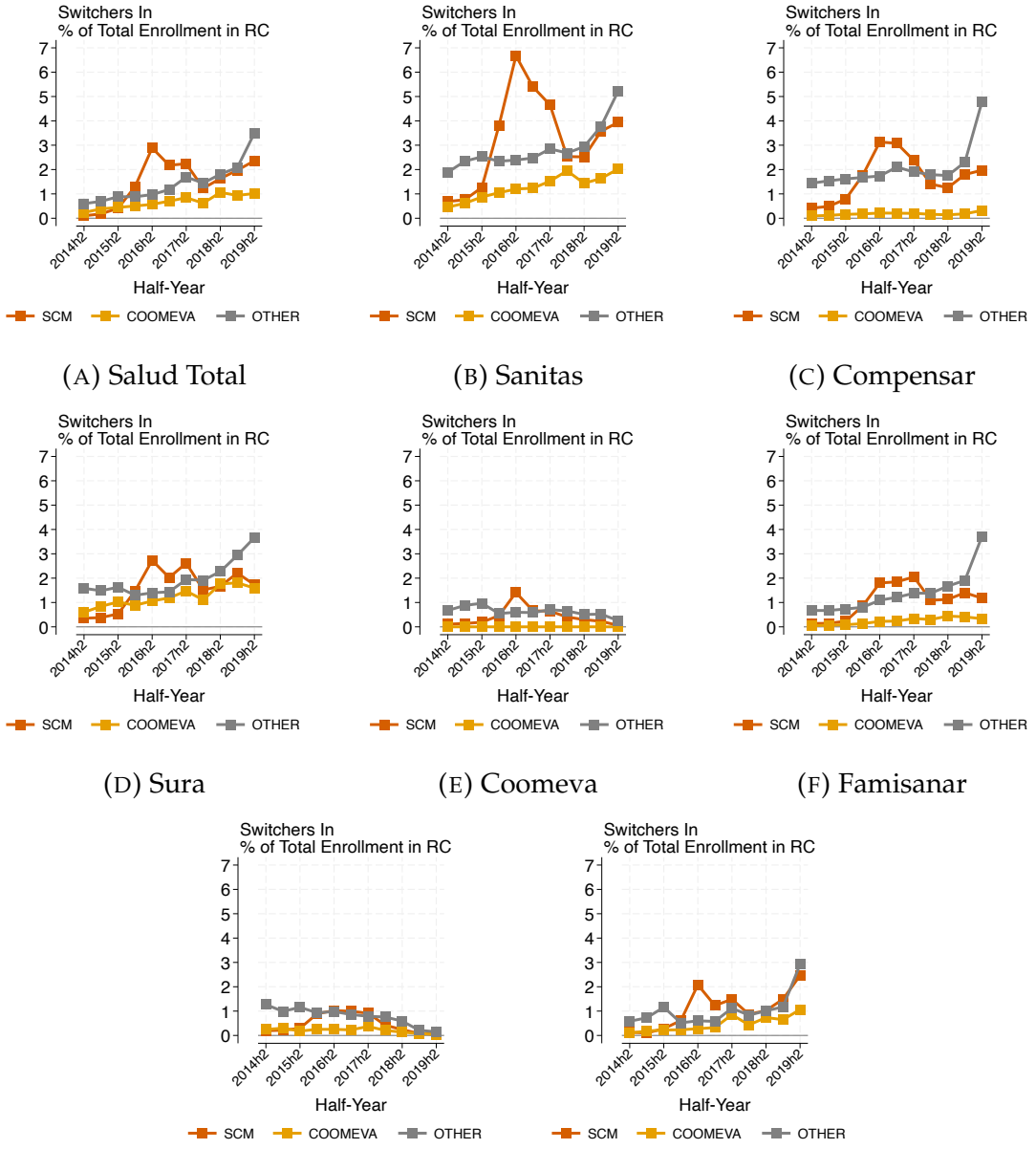
substantial for these plans with the exception of Compensar, which likely reflects the fact that Coomeva had low participation in Bogota, one of the main geographic markets for Compensar. This asymmetric exposure to terminated plans—particularly the outsized role of SCM—helps explain some of the risk composition shifts explored in the next sections.

The substantial and heterogeneous contribution of switchers to plans' total enrollment documented in this section is a key empirical fact for the remainder of the analysis. In the next sections, we show that these flows implied significant changes in the utilization patterns and risk composition of plans, with important consequences for financial sustainability under coarse risk adjustment.

2 Excess Utilization Among Switchers

Having established that switchers accounted for a substantial share of enrollment across health plans, this section explores how costly these individuals were to insure. We compare the realized medical care utilization of switchers to that of other enrollees who belong to the same capitation payment bin. Since capitation payments are a coarse function of demographics, individuals within a bin generate the same revenue for the receiving plan, regardless of their underlying health needs. The results show that switchers consistently utilized more care than incumbents in the same revenue category. This difference persists over time and remains sizable even 3.5 years after the switch, underscoring the financial strain imposed by switchers in under imperfect risk adjustment.

To assess the relative profitability of switchers, we estimate two sets of linear models using OLS. Each model is designed to test whether switchers use more medical care than individuals already enrolled in the plans they switch to, conditional on belonging to the same capitation bin. We focus on comparisons within capitation bins because plan revenues are determined by these categories; holding capitation bin fixed ensures that differences in utilization translate directly into differences in profitability:



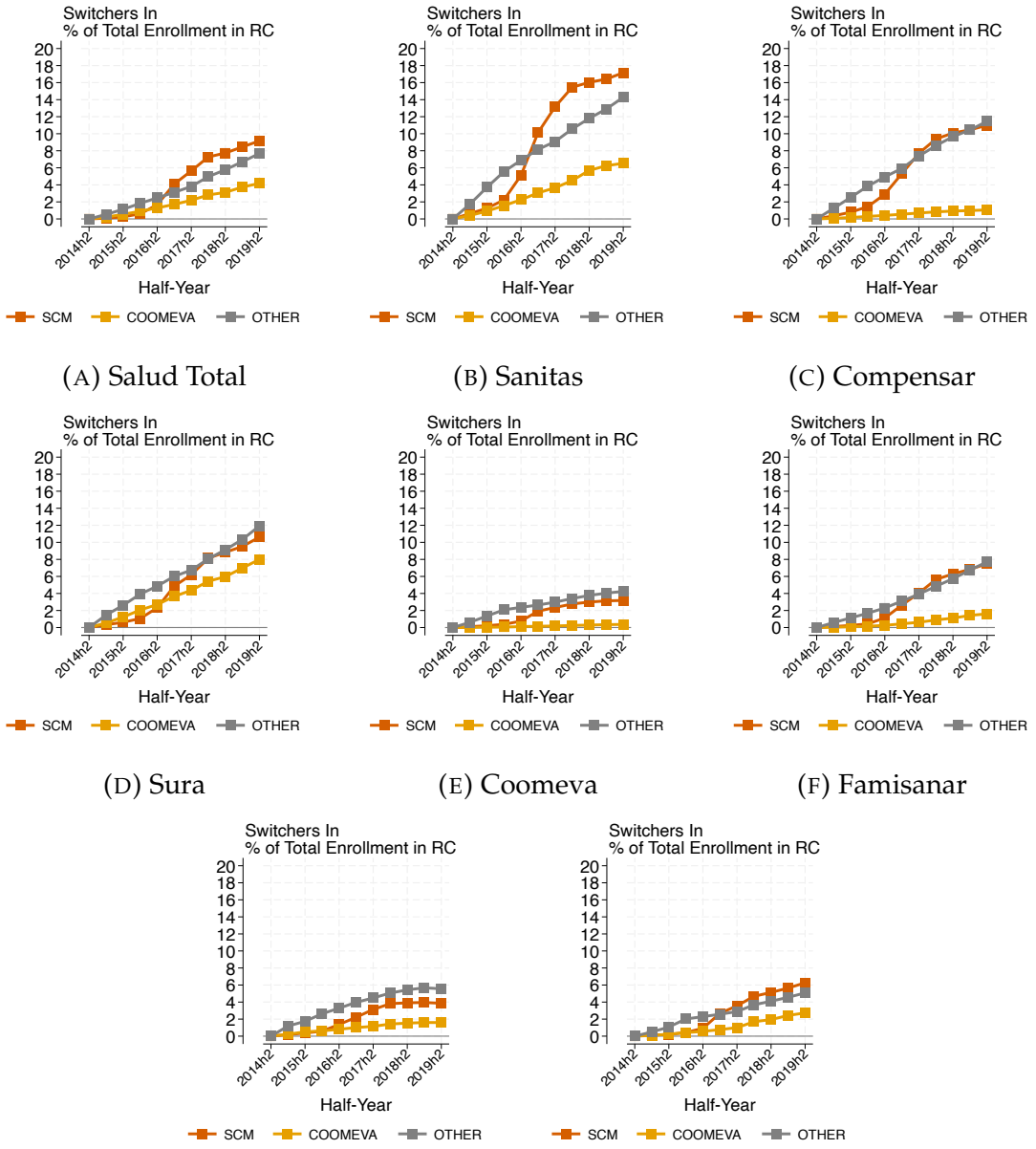
(A) Salud Total (B) Sanitas (C) Compensar

(D) Sura (E) Coomeva (F) Famisanar

(G) SOS (H) Nueva

FIGURE IV.5: Importance of Switchers to Each Plan's Enrollment (By Plan of Origin)

Note: This figure plots the contribution of Switchers In to the enrollment of each plan in the main sample for the period 2014-2019. Switchers are grouped by the program they are switching from (RS or RC). Each plot shows the number of "Switchers In" for each half year as a percentage of plan enrollment in that half-year.



(A) Salud Total (B) Sanitas (C) Compensar

(D) Sura (E) Coomeva (F) Famisanar

(G) SOS (H) Nueva

FIGURE IV.6: Cumulative Importance of Switchers to Each Plan's Enrollment (By Plan of Origin)

Note: This figure plots the contribution of Switchers In to the enrollment of each plan in the main sample for the period 2014-2019. Switchers are grouped by the plan they are switching from (Saludcoop-Medimas-Cafesalud, Coomeva, or Other). Each plot shows the cumulative number of "Switchers In" since 2014 as a percentage of plan enrollment in each half-year.

$$Y_{it} = \sum_{k=1}^{7} \beta_k \cdot \mathbb{1}\{\text{SwitchDate}_i - t = k\} + \alpha_{r(i,t),e(i,t),t} + \varepsilon_{it} \tag{IV.1}$$

$$Y_{it} = \sum_{g} \sum_{k=1}^{7} \beta_{gk} \cdot \mathbb{1}\{\text{SwitchDate}_i^g - t = k\} + \alpha_{r(i,t),e(i,t),t} + \varepsilon_{it} \tag{IV.2}$$

The outcome variable Y_{it} is a measure individual i's medical care utilization in half-year t. All specifications include capitation bin-by-insurer-by-time fixed effects, denoted by $\alpha_{r(i,t),e(i,t),t}$, where $r(i,t)$ denotes the capitation bin of i at time t and $e(i,t)$ the plan they are enrolled with. These fixed effects ensure that comparisons are made within plans, across individuals who generate the same revenue, while also controlling for plan- and time-specific shocks. All models include a standard mean-zero error term ε_{it}, and we assume that SwitchDate_i and SwitchDate_i^g are equal to infinity when i never switches in the data, or when i never switchers from group g, respectively. We also ignore any subsequent switches after the first.

The specifications are designed to test for the persistence of increased utilization. Equation IV.1 includes a series of indicators for each time horizon since switching. Each β_k captures the excess utilization of switchers k half-years after switching, relative to non-switchers in the same time period, capitation bin, insurer, and plan. On the other hand, Equation IV.2 allows these coefficients to vary by plan of origin. Following our approach in the previous section, we classify switchers into three mutually exclusive groups: those arriving from SCM, from Coomeva, and from other plans. The coefficients β_{gk} capture the excess utilization of switchers from group g at relative time k since they switched into their new plan.

The sample used to estimate these models spans the period 2016–2019 and is restricted to individual-period observations in which the enrollee is affiliated with one of the plans that reported claims data during this period. As a result, the estimation sample excludes enrollees while they are affiliated with non-reporting plans, including SCM. To avoid mechanically attenuated estimates of utilization, the switching indicators exclude the time period when the switching event occurs—e.g., where $\text{SwitchDate}_i = t$. Because the data

are aggregated at the half-year level, we do not observe the exact timing of the switch within each period. If a switch occurs in the middle of a semester, the individual is only partially enrolled, which mechanically lowers observed utilization in that period. This is a common issue in actuarial analyses of insurance data, where differences in time at risk—called exposure—must be accounted for when comparing utilization across enrollees.

Figure IV.7 presents the results from Equation IV.1. The results reveal a persistent gap: switchers use significantly more medical care than non-switchers, even several years after switching. The estimated coefficient for the first half-year after the switch is 0.9, which corresponds to a 146% increase in utilization relative to comparable non-switchers.[4] Although this excess declines over time, it remains substantial—around 0.45 in the seventh half-year—which corresponds to a 57% higher utilization rate. The point estimates are relatively stable after the second period.

To explore heterogeneity by plan of origin, Figure IV.8 shows the results from Equation IV.2, which allows excess utilization to vary across switchers from SCM, Coomeva, and other plans. The trajectories differ meaningfully. Switchers from SCM exhibit the highest and most persistent excess utilization: their utilization remains approximately 0.75–0.80 log units higher than that of comparable non-switchers for several periods, corresponding to a 112–123% increase in utilization. In contrast, switchers from Coomeva and other plans show a more pronounced decline in excess utilization over time. By the third half-year, their utilization levels are roughly 0.35–0.40 log units higher than those of comparable enrollees, equivalent to a 42–49% increase, and continue to fall thereafter—particularly for Coomeva switchers. These patterns suggest that the financial burden imposed by switchers varies not only in magnitude but also in duration, with switchers from SCM presenting the most persistent and costly profile.

Together, these results indicate that the higher utilization of switchers is not merely a short-run consequence of pent-up demand at the time of switching. While an initial spike in care use might be expected if individuals defer care while enrolled in low-performing

[4] Percentage differences are computed using $100 \cdot (\exp(\hat{\beta}) - 1)$.

plans, the fact that utilization remains significantly higher for at least 3.5 years after the switch points instead to persistent differences in underlying health risk. The magnitude and duration of the gap—particularly for switchers from SCM—suggest that these individuals are systematically sicker than incumbent enrollees. This interpretation reinforces the central claim of this chapter: increased switching after 2016 led to a redistribution of risk across plans.

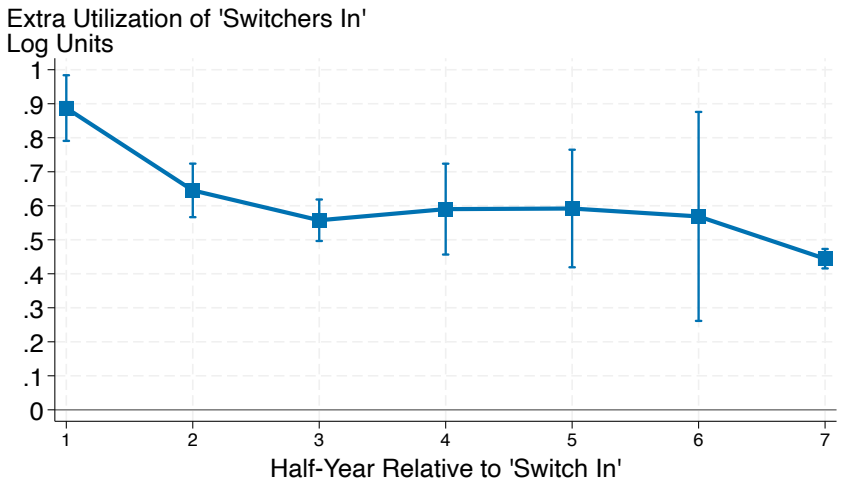


FIGURE IV.7: Extra Utilization of Switchers In

Note: This figure plots the OLS estimates of β_k for $k \in \{1, 2, ..., 7\}$ from equation IV.1, along with 95% confidence intervals. Standard errors are clustered twoway by individual and time. The sample used is the 10% random sample discussed in the main text.

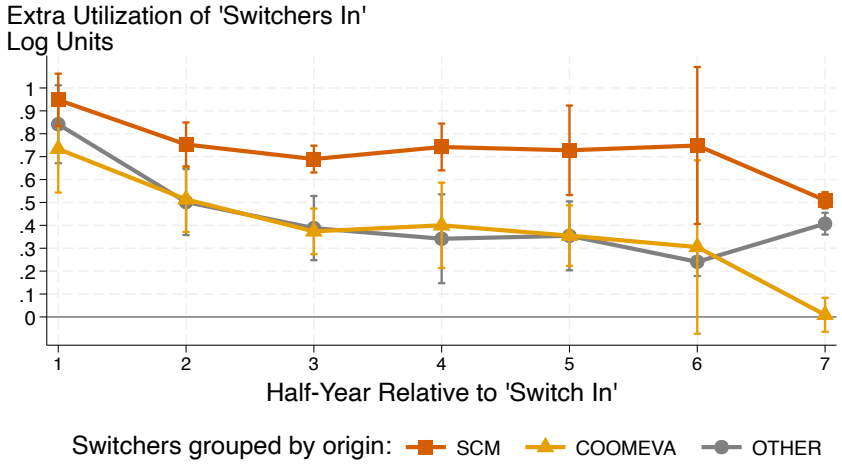


FIGURE IV.8: Extra Utilization of Switchers In (By Plan of Origin)

Note: This figure plots the OLS estimates of β_k for $k \in \{1, 2, ..., 7\}$ from equation IV.1, along with 95% confidence intervals. Standard errors are clustered twoway by individual and time. The sample used is the 10% random sample discussed in the main text.

We now turn to examine how the financial burden of switchers varies across plans. To do so, we estimate the dynamic specifications in Equations IV.1 and IV.2 separately for each plan and present the results in Figures IV.9 and IV.10, where each panel corresponds to one plan in the reporting sample.

Figure IV.9 pools across all switchers, regardless of their plan of origin. Several patterns emerge. First, in nearly all plans, switchers exhibit persistently higher utilization than non-switchers. The magnitude of this excess is particularly large for plans that, as shown in the previous section, absorbed a greater share of switchers—such as Sanitas, Salud Total, Compensar and Sura. In contrast, for plans like Coomeva and SOS, which saw limited switching inflows, the estimated effects are close to zero. Across plans, estimated coefficients in the first half-year after switching range from 0.4 (Nueva EPS) to around 1.75 (Sanitas), which correspond to approximately 49% to a staggering 475% higher utilization, respectively. Second, while the magnitude of excess utilization generally declines over time, it remains positive and statistically significant in most cases.

Figure IV.10 plots the dynamic results disaggregated by plan of origin. The patterns are remarkably consistent across plans: switchers from SCM exhibit both higher and more persistent excess utilization compared to those from Coomeva or other origin plans. In nearly all panels, SCM switchers show the largest utilization gap relative to non-switchers, and this gap persists at a high level throughout the post-switch period, whereas for Coomeva and Others it tends to decline to zero.

The results highlight substantial heterogeneity in the cost implications of switcher inflows. While nearly all plans experience some increase in realized utilization following the influx of new enrollees, a subset—particularly those that absorbed large numbers of switchers from SCM—face a double burden. Not only did these plans receive disproportionately high inflows relative to their enrollment base, but each switcher also imposed significantly higher-than-average medical costs. This compounding effect—greater volume and greater intensity of care—amplifies the financial pressure on receiving plans. These patterns are difficult to explain through short-run pent-up demand alone and instead suggest persistent differences in underlying health risk. The next section explores

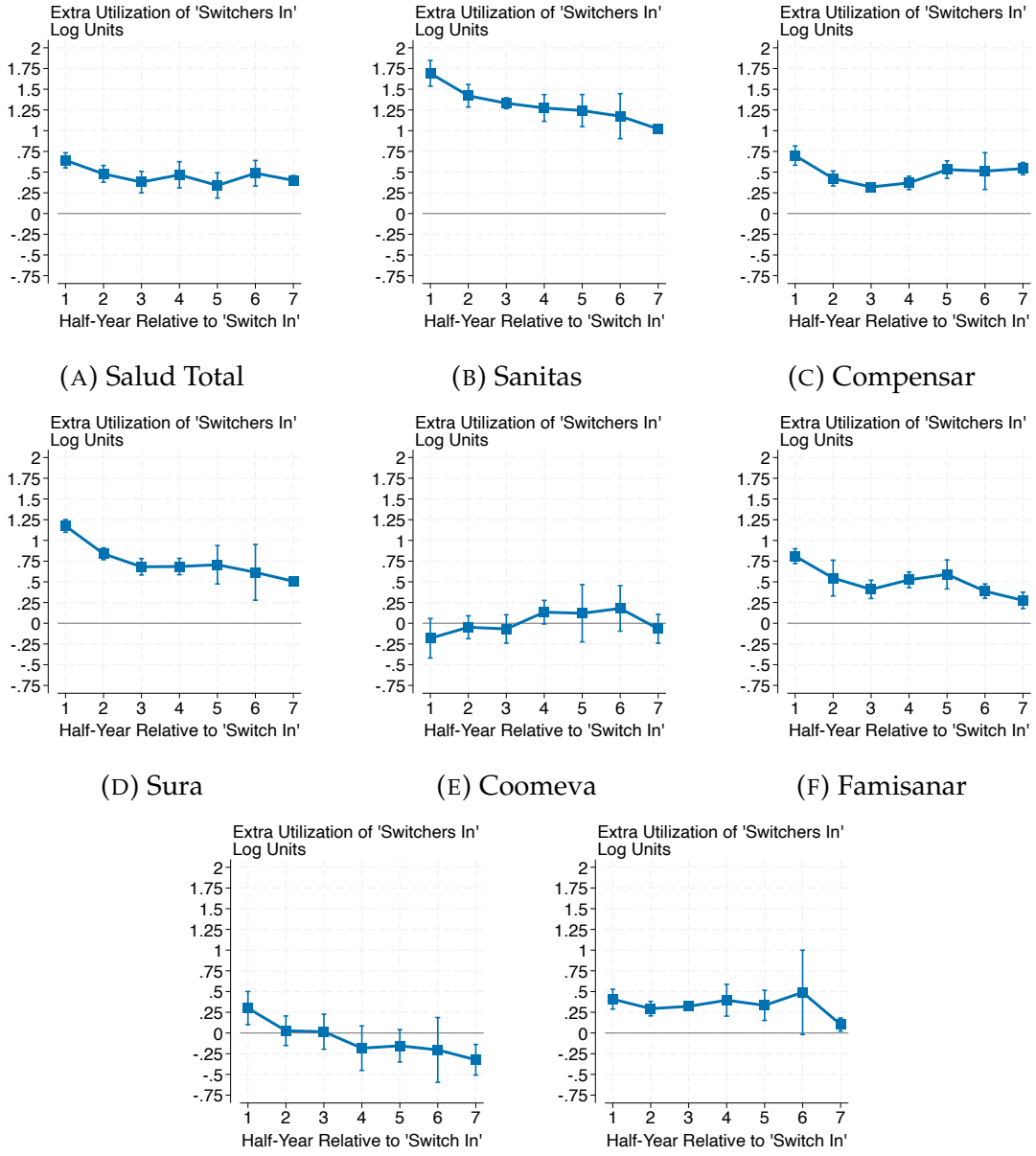
this interpretation more directly by assessing whether excess utilization can indeed be traced to differences in latent health status.

3 Evolution of Risk Composition

This section documents how the distribution of health risk evolved across plans during the period of substantial enrollee reallocation (2016–2019). It builds on the findings from Sections 1 and 2, which showed that a subset of plans received disproportionately large inflows of switchers, and that those switchers imposed persistently higher costs. We highlight three new empirical facts. First, consistent with the correlation between risk and choice persistence documented in Chapter II, average risk among enrollees who remained in the terminated plans increased steadily over time. Second, the plans that absorbed the largest volumes of switchers (relative to their enrollment) also experienced a rise in average risk, consistent with the patterns documented in Section 2. At first glance, these two findings appear to be in tension: how can both the terminated plans and the receiving plans grow riskier? The third empirical fact resolves this puzzle. The terminated plans began the period with substantially higher average risk than the plans that would later receive the bulk of their switchers. As a result, while the healthiest enrollees exited the terminated plans—raising average risk among those who remained—those who switched out were still riskier, on average, than the original enrollees in the receiving plans. These findings suggest that risk concentration, and its increase over time, was a key driver of the terminated plans' decline.

To study the evolution of risk composition, we focus on individuals enrolled in the RC in 2015 and follow them through 2019. We group plans into four categories: (i) SCM: Cafesalud and Medimás; (ii) Coomeva; (iii) SSCF: the set of receiving plans that absorbed the majority of switchers—Salud Total, Sura, Compensar, and Famisanar; and (iv) SN: Nueva EPS and SOS, which received fewer switchers relative to their enrollment. We exclude Sanitas from the analysis because it did not report utilization prior to 2016.

We measure each individual's risk using predicted healthcare utilization in 2015, based



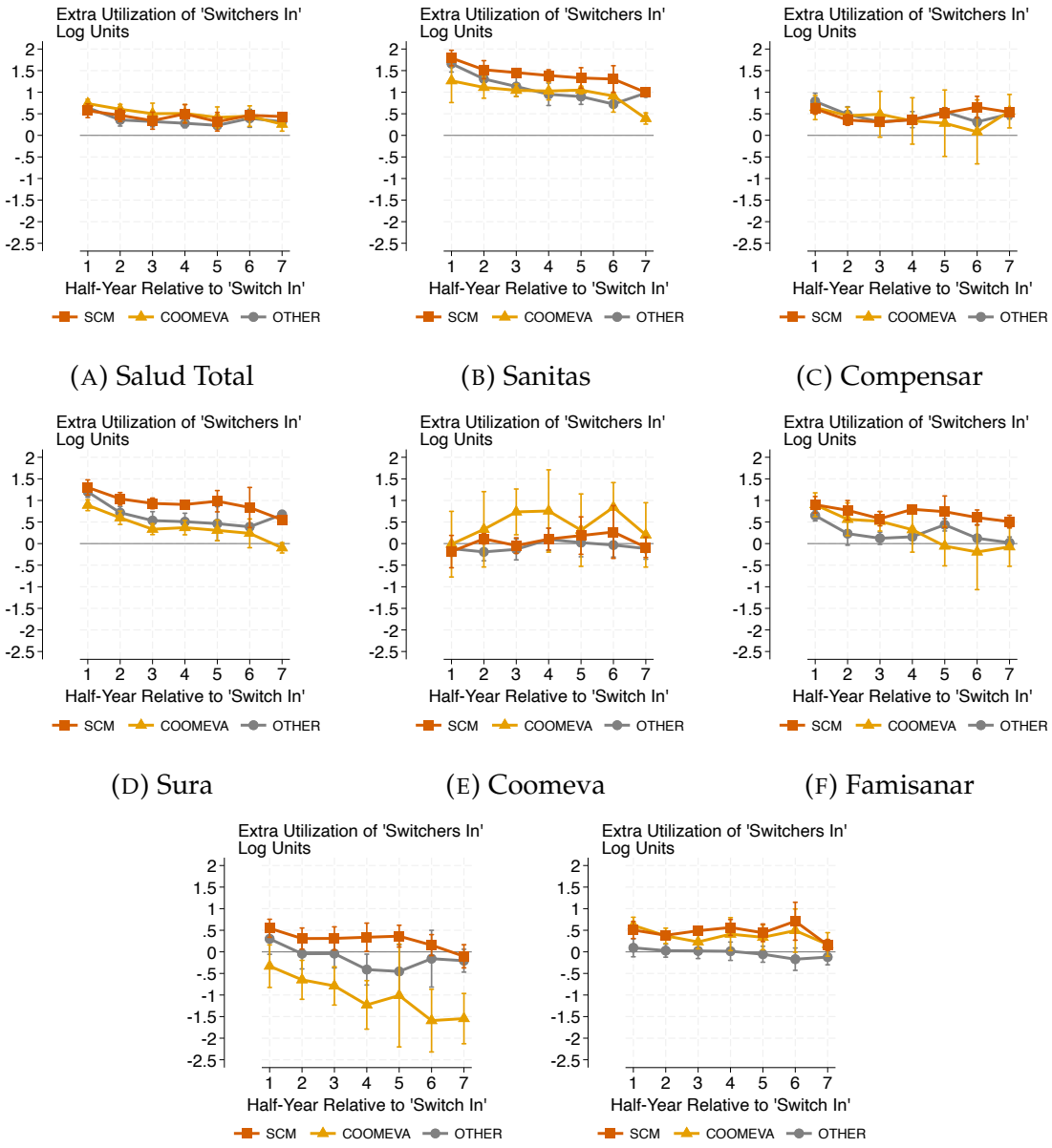
(A) Salud Total (B) Sanitas (C) Compensar

(D) Sura (E) Coomeva (F) Famisanar

(G) SOS (H) Nueva

FIGURE IV.9: Extra Utilization of Switchers In for Each Plan

Note: This figure plots the OLS estimates of β_k for $k \in \{1, 2, ..., 7\}$ from equation IV.1, along with 95% confidence intervals. Each panel is a separate regression for each plan. Standard errors are clustered twoway by individual and time. The sample used before restricting by plan is the 10% random sample discussed in the main text.



(A) Salud Total (B) Sanitas (C) Compensar

(D) Sura (E) Coomeva (F) Famisanar

(G) SOS (H) Nueva

FIGURE IV.10: Extra Utilization of Switchers In for Each Plan (By Plan of Origin)

Note: This figure plots the OLS estimates of β_{gk} for $g = \text{SCM}, \text{Coomeva}, \text{Other}$ and $k \in \{1, 2, ..., 7\}$ from equation IV.2, along with 95% confidence intervals. Each panel is a separate regression for each plan. Standard errors are clustered twoway by individual and time. The sample used before restricting by plan is the 10% random sample discussed in the main text.

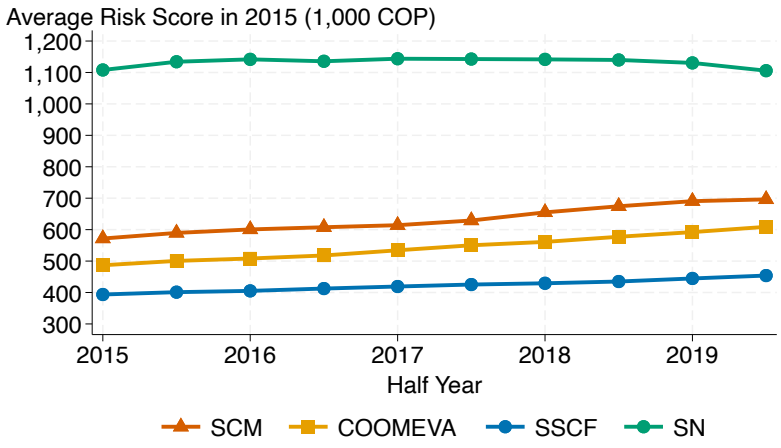
on the risk model developed in Chapter III for Nueva EPS. This model uses demographic characteristics, enrollment history, and past diagnostic information to predict future utilization. Importantly, by applying a model estimated on a single plan to all individuals, this approach controls for variation in the supply of care across plans, enabling a more standardized comparison of enrollee risk.

Figure IV.11 presents the main results of this section. Panel A shows average realized utilization in 2015 across the four plan groups. To ease the comparison, Panel B shows the same results but normalizing each line by its value in 2015. The first empirical fact is that average risk increased steadily for enrollees who remained in the terminated plans. For SCM, the average risk score rose from 572,000 COP in 2015h1 to 696,000 COP in 2019h2—an increase of 21.7%. Coomeva exhibited a similar trajectory, with average risk rising from 487,000 COP to 609,000 COP, a 25.1% increase over the same period. These results are consistent with the lock-in effect described in Chapter II: when the relatively healthier switch out at higher rates, they leave behind a riskier pool of enrollees.

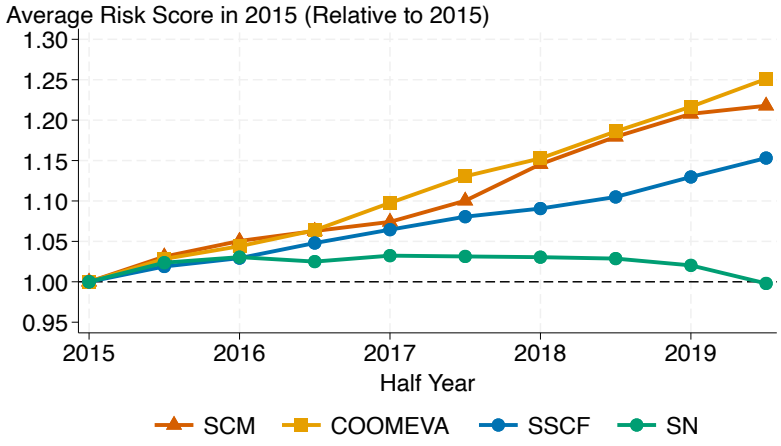
The second fact is that the plans that absorbed the largest inflows of switchers—those in the SSCF group—also experienced a meaningful increase in average risk, rising from 394,000 COP to 454,000 COP, or 15.4%. This is consistent with the results in 2 that document the higher cost profile of incoming enrollees relative to the original risk pool.

The third fact explains why average risk increased for both groups: SCM and Coomeva already had significantly higher average risk in 2015: (572,000 and 487,000 COP, respectively) than SSCF (394,000 COP). The SN group, which received fewer switchers, had an even higher baseline risk of over 1.1 million COP.[5] This initial imbalance helps reconcile the first two facts: although switchers were high-risk, they were still healthier than those who remained in the terminated plans, and riskier than those originally enrolled in the receiving plans.

[5]Their higher risk is explained by fact that Nueva disproportionally enrolls older enrolles. As shown in Table A.1, the average age of their enrollees in 2015 was 44, compared to 31-34 at other plans.



(A) Risk Score in 2015



(B) Risk Score in 2015 (Relative to 2015)

FIGURE IV.11: Evolution of Ex-Ante Risk Composition Across Plans

Note: This figure shows the evolution of average enrollee risk for three groups of health plans: (1) Coomeva, (2) Saludcoop–Cafesalud–Medimás (SCM), (3) Sura, Salud Total, Compensar, Famisanar, and (4) SOS, Nueva. Panel A shows average risk score in 2015, which is the prediction of utilization in 2015 based on the Nueva risk model from Chapter III. The sample is restricted to individuals who were enrolled in 2015. Panel B shows the same plots in Panel B where each line is relative to its value in 2015.

Chapter V

Conclusion

This dissertation provides novel evidence on the role of health status as a driver of consumer inertia in health insurance markets. The findings demonstrate that health risk increases choice persistence, even when health plans exhibit substantial differences in the amount of medical care they provide to their enrollees. While the correlation between health status and persistence may have limited implications when plan quality is homogeneous and switching rates are low, it can have much more serious consequences when plan quality diverges and switching becomes more frequent. In such cases, healthier enrollees tend to exit first, leaving behind a pool of higher-risk individuals. This shift raises the average risk profile of the plan, which can strain financial sustainability and further degrade quality, setting off a dynamic that resembles a death spiral.

Importantly, this is not a classic adverse selection death spiral. In the standard theory (e.g., Rothschild and Stiglitz, 1976), high-risk individuals are disproportionately attracted to higher-quality offerings, raising costs and potentially destabilizing those options. In contrast, this dissertation documents a dynamic in which higher-risk individuals remain in low-quality plans that continue to deteriorate over time, while lower-risk individuals exit early. This nuance echoes the insight in Polyakova (2016) that the impact of switching frictions on adverse selection depends critically on the direction of change in plan characteristics. When quality declines in some plans, frictions can amplify risk concentration in those plans, as illustrated by the case of Coomeva and Saludcoop–Cafesalud–Medimás. The consequences of this dynamic are particularly severe in settings with coarse risk ad-

justment, such as Colombia's Régimen Contributivo, where payments to insurers do not fully account for differences in enrollee risk.

Several important questions remain open. One immediate extension is to explore the link between risk composition, financial performance, and care provision more directly. Chapter IV shows that increased switching after 2015 had a substantial impact on the risk composition of plans, and that care utilization declined sharply in geographic markets highly exposed to the termination of Saludcoop. A more explicit analysis of how risk composition affects financial performance and, in turn, service provision would help assess the potential impact of improving the risk adjustment formula.

Another important area for further research is insurer entry. In principle, consumer inertia gives incumbents market power, while discouraging entry by making it difficult to attract enrollees. However, when risk and inertia are correlated, this logic may be inverted. Because sicker individuals are more likely to remain in their existing plans, new entrants may disproportionately attract healthier enrollees. Understanding this interplay is critical to evaluating the conditions under which entry can improve market outcomes. From a welfare perspective, it is also important to assess whether this creates incentives for excessive entry, as new plans may be able to cream-skim healthy enrollees by targeting those with lower switching costs.

Finally, the approach taken in this dissertation treats health status as exogenous to plan quality. In practice, however, prolonged enrollment in low-quality plans may worsen health outcomes, increase long-term healthcare costs, and create pent-up demand for care. When such plans are eventually terminated, this deferred care can spill over into receiving plans, straining their financial resources and potentially undermining their quality. Accounting for these longer-term consequences remains an important direction for future research.

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Appendix

A Tables

TABLE A.1: Summary Statistics by Plan

	Salud Total	Sanitas	Compensar	Sura	Famisanar	Nueva	Coomeva	Cafesalud	All
				A. December 2015					
Enrollment									
N	2,106,200	1,479,500	1,117,500	2,221,400	1,679,100	2,810,300	2,784,500	5,091,200	21,459,200
Market share	9.8	6.9	5.2	10.4	7.8	13.1	13.0	23.7	100.0
Age									
Mean	31	36	33	33	31	44	33	32	34
25th ptile	16	20	18	19	16	23	17	16	18
Median	29	34	32	31	29	47	32	31	32
75th ptile	44	51	47	46	44	63	48	46	49
90th ptile	57	65	60	58	57	75	61	59	63
IBC									
Mean	1,232	2,754	1,908	1,770	1,349	1,297	1,578	1,275	1,542
Std Dev	1,690	3,216	2,151	2,312	1,702	1,609	2,046	1,486	2,028
Median	771	1,449	1,100	917	830	770	860	800	849
75th ptile	1,169	3,458	2,184	1,759	1,309	1,295	1,600	1,300	1,509
90th ptile	2,163	6,600	4,267	3,985	2,600	2,550	3,401	2,488	3,264
				B. December 2019					
Enrollment									
N	2,973,600	3,175,500	1,648,400	3,527,000	2,127,100	3,782,800	1,476,900	1,673,100	22,628,200
Market share	13.1	14.0	7.3	15.6	9.4	16.7	6.5	7.4	100.0
Age									
Mean	32	35	35	35	33	42	36	36	35
25th ptile	32	35	35	35	33	42	36	36	35
Median	30	33	34	33	30	41	35	35	33
75th ptile	44	49	50	49	46	61	52	51	50
90th ptile	58	63	63	62	60	74	65	65	64
IBC									
Mean	1,387	2,488	2,168	2,084	1,486	1,428	1,865	1,484	1,787
Std Dev	1,456	2,881	2,235	2,363	1,509	1,413	2,184	1,506	2,093
Median	982	1,334	1,309	1,200	1,035	980	1,070	963	1,063
75th ptile	1,360	2,879	2,523	2,228	1,500	1,500	1,865	1,503	1,785
90th ptile	2,349	5,703	4,700	4,508	2,609	2,545	4,054	2,743	3,664

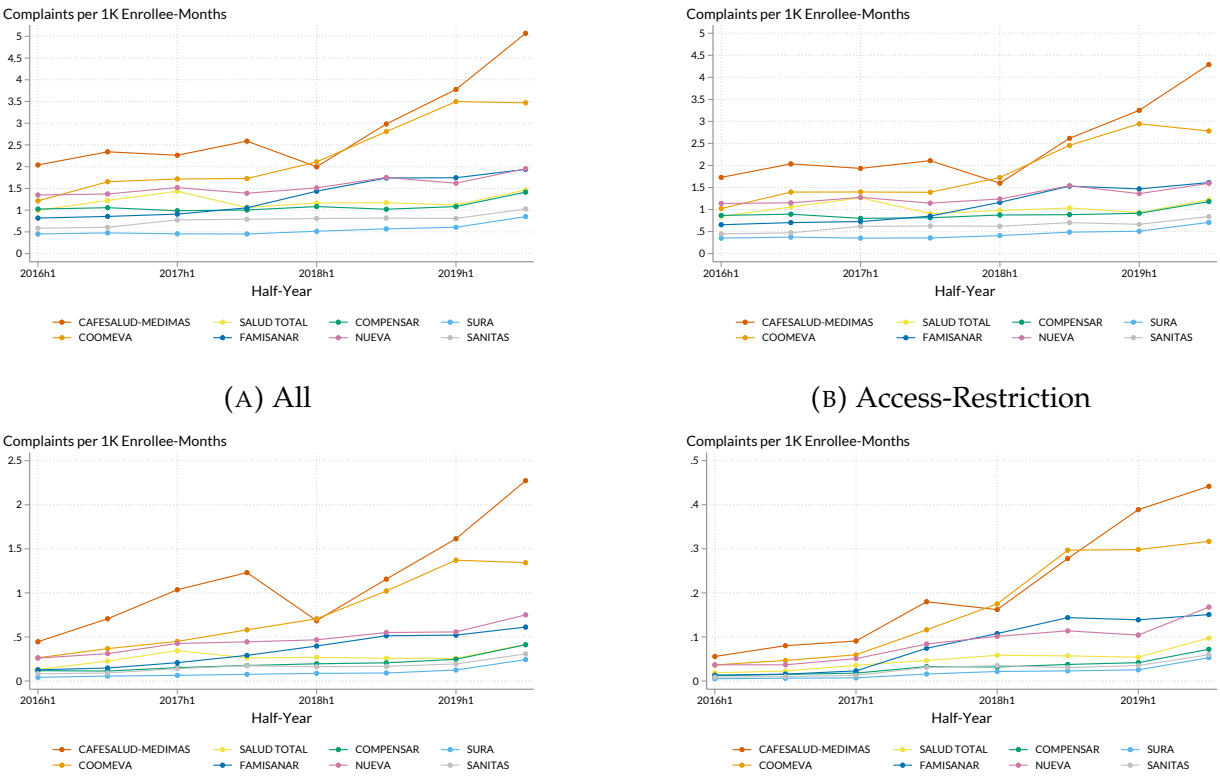
Notes: This table shows descriptive statistics for the main plans in the analysis. The sample is restricted to urban markets, as defined in the main text. Panel A shows statistics for 2015 and Panel B for 2019. The statistics for Cafesalud in 2019 correspond to Medimás, which is the new plan that was created when Cafesalud was sold. IBC refers to the monthly income used for calculating the payroll tax contributions and is measured in thousand of current Colombian pesos. Statistics are computed using a random sample of 10% of individuals, but the totals are adjusted to reflect the full sample.

TABLE A.2: Treatment Effect of Cancer Shock on the Probability of Switching Insurers

	(1)	(2)	(3)	(4)
Treatment Effect	-0.0430	-0.0333	-0.0448	-0.0347
	(0.001599)	(0.001766)	(0.001672)	(0.001864)
Counterfactual Mean (Treated)	0.1238	0.1248	0.1196	0.1203
	(0.001279)	(0.001396)	(0.001346)	(0.001484)
Ratio	-0.3477	-0.2672	-0.3744	-0.2879
	(0.0120)	(0.0136)	(0.0130)	(0.0149)
Number of Treated	28,027	23,027	23,875	19,195
Number of Control	310,646	305,548	310,646	305,548
Treatment Years	2016-2019	2016-2019	2016-2018	2016-2018
Treated Sample	All	Survivors	All	Survivors

Notes: Estimated treatment effect of cancer shock on the probability of switching out of 2015 insurer during the period 2015-2019. The first row presents treatment effect estimates, the second row the estimate of mean counterfactual switching out rates for the treated group, and the third presents the ratio between the first two. Columns (1) and (3) use the full sample, and columns (2) and (4) drop individuals in treatment and control group who passed away before the end of 2019. Columns (1) and (2) use all cancer shocks since 2016, and columns (3) and (4) drop cancer shocks happening in 2019. The construction of treatment and control groups is explained in detail in the appendix. Treatment effects are estimated using the Stata command `teffects ra`, which uses a regression adjustment approach. Model of mean is a linear function of metropolitan area by insurer dummies, and interactions of female indicator with age and age squared. Robust standard errors in parentheses.

B Figures



(A) All

(B) Access-Restriction

(C) Life-Threatening

(D) Cancer Complaints

FIGURE B.1: Average Complaints by Type and Insurer (Time Trends)

Note: Complaints data from SNS and enrollment data from Cubo Afiliados SISPRO. Data only for the 44 urban municipios that belong to the 23 Cities and Metropolitan Areas identified by DANE. Insurers are ordered by the results in Panel (A). Go back to main text.

C Reference Tables

Diagnostic Groups for Model of Care Provision

TABLE C.1: Diagnostic Groups Used in Model of Care Provision

ICD 10	Description
B20	HIV disease
C00	Malignant neoplasm of lip
C01	Malignant neoplasm of base of tongue
C02	Malignant neoplasm of other and unspecified parts of tongue
C03	Malignant neoplasm of gum
C04	Malignant neoplasm of floor of mouth
C05	Malignant neoplasm of palate
C06	Malignant neoplasm of other and unspecified parts of mouth
C07	Malignant neoplasm of parotid gland
C08	Malignant neoplasm of other and unspecified major salivary glands
C09	Malignant neoplasm of tonsil
C10	Malignant neoplasm of oropharynx
C11	Malignant neoplasm of nasopharynx
C12	Malignant neoplasm of pyriform sinus
C13	Malignant neoplasm of hypopharynx
C14	Malignant neoplasm of other and ill-defined sites in the lip, oral cavity and pharynx
C15	Malignant neoplasm of esophagus
C16	Malignant neoplasm of stomach
C18	Malignant neoplasm of colon
C22	Malignant neoplasm of liver and intrahepatic bile ducts
C23	Malignant neoplasm of gallbladder
C25	Malignant neoplasm of pancreas
C34	Malignant neoplasm of bronchus and lung
C40	Malignant neoplasm of bone and articular cartilage of limbs
C41	Malignant neoplasm of bone and articular cartilage of other and unspecified sites
C43	Malignant melanoma of skin
C44	Other malignant neoplasms of skin
C45	Mesothelioma
C46	Kaposi's sarcoma
C47	Malignant neoplasm of peripheral nerves and autonomic nervous system
C48	Malignant neoplasm of retroperitoneum and peritoneum
C49	Malignant neoplasm of other connective and soft tissue
C50	Malignant neoplasm of breast
C61	Malignant neoplasm of prostate
C64	Malignant neoplasm of kidney
C67	Malignant neoplasm of bladder
C70	Malignant neoplasm of meninges
C71	Malignant neoplasm of brain
C72	Malignant neoplasm of spinal cord, cranial nerves and other parts of central nervous system
C73	Malignant neoplasm of thyroid gland
C76	Malignant neoplasm of other and ill-defined sites
C77	Secondary and unspecified malignant neoplasm of lymph nodes
C78	Secondary malignant neoplasm of respiratory and digestive organs
C79	Secondary malignant neoplasm of other sites
C80	Malignant neoplasm without specification of site
C81	Hodgkin lymphoma
C82	Follicular lymphoma
C83	Diffuse non-Hodgkin lymphoma

ICD 10	Description
C84	Peripheral and cutaneous T-cell lymphomas
C85	Other and unspecified types of non-Hodgkin lymphoma
C90	Multiple myeloma
C91	Lymphoid leukemia
C97	Multiple independent primary neoplasms
D46	Myelodysplastic syndromes
D50	Iron deficiency anemia
D63	Anemia in chronic diseases
D64	Other anemias
D80	Immunodeficiency with predominantly antibody defects
D81	Combined immunodeficiencies
D82	Immunodeficiency associated with other major defects
D83	Common variable immunodeficiency
D84	Other immunodeficiencies
D89	Other disorders involving the immune mechanism
E10	Type 1 diabetes mellitus
E11	Type 2 diabetes mellitus
E66	Obesity
E78	Disorders of lipoprotein metabolism
F01	Vascular dementia
F03	Unspecified dementia
F06	Mental disorders due to brain damage
F07	Personality and behavioral disorders due to brain disease
F09	Unspecified mental disorder due to known physiological condition
F10	Alcohol-related disorders
F11	Opioid-related disorders
F20	Schizophrenia
F25	Schizoaffective disorders
F31	Bipolar disorder
F32	Depressive episode
F33	Recurrent depressive disorder
F41	Anxiety disorders
F84	Pervasive developmental disorders (autism, etc.)
F88	Other disorders of psychological development
F99	Mental disorder, not otherwise specified
G20	Parkinson's disease
G30	Alzheimer's disease
G31	Other degenerative CNS diseases
G35	Multiple sclerosis
G89	Pain disorders related to chronic conditions
I10	Essential (primary) hypertension
I11	Hypertensive heart disease
I20	Angina pectoris
I21	Acute myocardial infarction
I25	Chronic ischemic heart disease
I42	Cardiomyopathy
I50	Heart failure
I63	Cerebral infarction (ischemic stroke)
I69	Sequelae of cerebrovascular disease
J43	Emphysema
J44	Chronic obstructive pulmonary disease (COPD)
J45	Asthma
J96	Respiratory failure
K50	Crohn's disease
K51	Ulcerative colitis
K70	Alcoholic liver disease
K74	Fibrosis and cirrhosis of liver
K76	Other liver diseases
K92	Other diseases of digestive system

ICD 10	Description
L40	Psoriasis
L89	Pressure ulcers
M05	Rheumatoid arthritis with rheumatoid factor
M06	Other rheumatoid arthritis
M81	Osteoporosis without current pathological fracture
N18	Chronic kidney disease
N39	Urinary tract disorders
P07	Disorders related to prematurity
Q90	Down syndrome
R06	Abnormalities of breathing
R26	Abnormalities of gait and mobility
R29	Other symptoms involving nervous and musculoskeletal systems
R41	Cognitive symptoms
R53	Malaise and fatigue
R54	Age-related physical debility
S06	Intracranial injury
S72	Fracture of femur (hip fracture)
Z00	General examination and routine child health check
Z43	Attention to artificial openings
Z51	Encounter for chemotherapy or dialysis
Z72	Problems related to lifestyle
Z73	Problems related to life-management difficulty
Z74	Problems related to care provider dependency
Z75	Problems related to medical facility access and care
Z76	Persons encountering health services in other circumstances
Z79	Long-term (current) drug therapy
Z91	Personal risk factors, including fall history
Z94	Transplanted organ and tissue status
Z95	Presence of cardiac and vascular implants and grafts
Z96	Presence of other functional implants
Z98	Other postprocedural states
Z99	Dependence on enabling machines and devices

Notes: This table lists the diagnostic groups used as predictors in the model of care provision presented in Chapter III. The diagnostic codes follow the International Classification of Diseases, 10th Revision (ICD-10), for Colombia. Diagnoses are grouped using the first three characters of each code.

Cancer Shock Activities

TABLE C.2: Activities for Breast Cancer

Code	Name	Code Type	Activity Type	Purpose
L01AA01	CICLOFOSFAMIDA	ATC	Medication	Treatment
L01BC02	FLUOROURACILO	ATC	Medication	Treatment
L01BC05	GEMCITABINA	ATC	Medication	Treatment
L01BC06	CAPECITABINA	ATC	Medication	Treatment
L01CD01	PACLITAXEL	ATC	Medication	Treatment
L01CD02	DOCETAXEL	ATC	Medication	Treatment
L01DB01	DOXORUBICINA	ATC	Medication	Treatment
L01DB03	EPIRRUBICINA	ATC	Medication	Treatment
L01XA01	CISPLATINO	ATC	Medication	Treatment
L01XC03	TRASTUZUMAB	ATC	Medication	Treatment

Code	Name	Code Type	Activity Type	Purpose
L01XC07	BEVACIZUMAB	ATC	Medication	Treatment
L01XC14	TRASTUZUMAB EMTANSINA	ATC	Medication	Treatment
L01XC32	ATEZOLIZUMAB	ATC	Medication	Treatment
L01XE07	LAPATINIB	ATC	Medication	Treatment
L01XE33	PALBOCICLIB	ATC	Medication	Treatment
L01XE42	RIBOCICLIB	ATC	Medication	Treatment
L01XE50	ABEMACICLIB	ATC	Medication	Treatment
L02BA01	TAMOXIFENO	ATC	Medication	Treatment
L02BA03	FULVESTRANT	ATC	Medication	Treatment
L02BG03	ANASTRAZOL	ATC	Medication	Treatment
851	PROCEDIMIENTOS DIAGNOSTICOS EN MAMA	CUPS	Procedure	Diagnosis
8511	BIOPSIA CERRADA (PERCUTANEA) (AGUJA) DE MAMA	CUPS	Procedure	Diagnosis
851101	BIOPSIA POR PUNCION CON AGUJA FINA DE MAMA	CUPS	Procedure	Diagnosis
851102	BIOPSIA DE MAMA CON AGUJA (TRUCUT)	CUPS	Procedure	Diagnosis
851103	BIOPSIA DE MAMA POR ESTEREOTAXIA	CUPS	Procedure	Diagnosis
8512	BIOPSIA ABIERTA DE MAMA	CUPS	Procedure	Diagnosis
851200	BIOPSIA ABIERTA DE MAMA SOD	CUPS	Procedure	Diagnosis
8513	LOCALIZACION DE LESION NO PALPABLE DE MAMA	CUPS	Procedure	Diagnosis
851301	LOCALIZACION DE LESION NO PALPABLE DE MAMA CON ARPON U OTRO	CUPS	Procedure	Diagnosis
851302	LOCALIZACION DE LESION NO PALPABLE DE MAMA POR ESTEREOTAXIA	CUPS	Procedure	Diagnosis
851303	LOCALIZACION DE LESION NO PALPABLE DE MAMA RADIOGUIADA	CUPS	Procedure	Diagnosis
852002	ESCISION SELECTIVA DE CANAL GALACTOFORO	CUPS	Procedure	Diagnosis
852003	ESCISION EN BLOQUE DE CONDUCTOS GALACTOFOROS	CUPS	Procedure	Diagnosis
8768	MAMOGRAFIA	CUPS	Procedure	Diagnosis
876801	MAMOGRAFIA UNILATERAL O DE PIEZA QUIRURGICA	CUPS	Procedure	Diagnosis
876802	MAMOGRAFIA BILATERAL	CUPS	Procedure	Diagnosis
876803	TOMOSINTESIS	CUPS	Procedure	Diagnosis
8769	GALACTOGRAFIA DE CONTRASTE	CUPS	Procedure	Diagnosis
876901	GALACTOGRAFIA DE UN CONDUCTO	CUPS	Procedure	Diagnosis
876902	GALACTOGRAFIA DE MULTIPLES	CUPS	Procedure	Diagnosis
881201	ECOGRAFIA DE MAMA CON TRANSDUCTOR DE 7 MHZ O MAS	CUPS	Procedure	Diagnosis
906603	ANTIGENO DE CANCER DE MAMA SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
908432	BRCA 1 Y BRCA 2 PERFIL COLOMBIA	CUPS	Procedure	Diagnosis
908433	BRCA 1 Y BRCA 2 SECUENCIACION COMPLETA	CUPS	Procedure	Diagnosis
908434	BRCA 1 Y BRCA 2 MUTACION FAMILIAR CONOCIDA	CUPS	Procedure	Diagnosis
920215	GAMAGRAFIA DE GLANDULA MAMARIA	CUPS	Procedure	Diagnosis
852401	ESCISION DE PEZON ACCESORIO O SUPERNUMERARIO	CUPS	Procedure	Diagnosis and Treatment
8526	ESCISION DE AREOLA O PEZON	CUPS	Procedure	Diagnosis and Treatment
9213	GAMAGRAFIA DE VIABILIDAD TUMORAL (RASTREO GAMAGRAFICO)	CUPS	Procedure	Diagnosis and Treatment
921301	GAMAGRAFIA DE VIABILIDAD TUMORAL CON MIBI, TETROFOSMIN, TALI	CUPS	Procedure	Diagnosis and Treatment
921302	GAMAGRAFIA TUMORAL CON 18 FDG.	CUPS	Procedure	Diagnosis and Treatment
921303	GAMAGRAFIA TUMORAL CON 11 C. METIONINA	CUPS	Procedure	Diagnosis and Treatment
9216	GAMAGRAFIA DE ANTICUERPOS MONOCLONALES	CUPS	Procedure	Diagnosis and Treatment
921600	GAMAGRAFIA DE ANTICUERPOS MONOCLONALES SOD	CUPS	Procedure	Diagnosis and Treatment
9217	GAMAGRAFIA CON DMSA PENTAVALENTE	CUPS	Procedure	Diagnosis and Treatment
921700	GAMAGRAFIA CON DMSA. PENTAVALENTE SOD	CUPS	Procedure	Diagnosis and Treatment
402201	ESCISION DE GANGLIO LINFATICO MAMARIO INTERNO	CUPS	Procedure	Treatment
402301	ESCISION DE GANGLIO LINFATICO AXILAR VIA ABIERTA	CUPS	Procedure	Treatment
852	ESCISION DE TEJIDO DE LA MAMA	CUPS	Procedure	Treatment
8520	ESCISION O ABLACION DE TEJIDO DE MAMA	CUPS	Procedure	Treatment
8521	ESCISION LOCAL DE LESION DE MAMA	CUPS	Procedure	Treatment
852100	RESECCION LOCAL DE LESION DE MAMA SOD	CUPS	Procedure	Treatment
8522	RESECCION DE CUADRANTES DE MAMA	CUPS	Procedure	Treatment
852201	RESECCION DE CUADRANTE DE MAMA	CUPS	Procedure	Treatment
852202	RESECCION DE CUADRANTE DE MAMA CON CONDUCTOS TERMINALES	CUPS	Procedure	Treatment
8523	MASTECTOMIA SUBTOTAL	CUPS	Procedure	Treatment
852300	MASTECTOMIA SUBTOTAL SOD	CUPS	Procedure	Treatment
8534	PLASTIA ONCOLOGICA DE MAMA (MAMOPLASTIA ONCOLOGICA)	CUPS	Procedure	Treatment
853401	MAMOPLASTIA ONCOLOGICA UNILATERAL	CUPS	Procedure	Treatment

Code	Name	Code Type	Activity Type	Purpose
853402	MAMOPLASTIA ONCOLOGICA BILATERAL	CUPS	Procedure	Treatment
854	MASTECTOMIA	CUPS	Procedure	Treatment
8540	MASTECTOMIA SUBCUTANEA	CUPS	Procedure	Treatment
854101	MASTECTOMIA SIMPLE UNILATERAL	CUPS	Procedure	Treatment
854102	MASTECTOMIA SIMPLE UNILATERAL POR GLANDULA SUPERNUMERARIA	CUPS	Procedure	Treatment
854103	MASTECTOMIA SIMPLE UNILATERAL CON PRESERVACION DE PIEL O COM	CUPS	Procedure	Treatment
8542	MASTECTOMIAS SIMPLES BILATERALES	CUPS	Procedure	Treatment
854201	MASTECTOMIA SIMPLE BILATERAL	CUPS	Procedure	Treatment
854202	MASTECTOMIA SIMPLE BILATERAL POR GLANDULA SUPERNUMERARIA	CUPS	Procedure	Treatment
854203	MASTECTOMIA SIMPLE BILATERAL CON PRESERVACION DE PIEL O COMP	CUPS	Procedure	Treatment
8543	MASTECTOMIA SIMPLE AMPLIADA UNILATERAL	CUPS	Procedure	Treatment
854301	MASTECTOMIA SIMPLE CON ESCISION DE GANGLIOS LINFATICOS REGIO	CUPS	Procedure	Treatment
8544	MASTECTOMIA SIMPLE AMPLIADA BILATERAL	CUPS	Procedure	Treatment
854401	MASTECTOMIA SIMPLE AMPLIADA BILATERAL VIA ABIERTA	CUPS	Procedure	Treatment
8545	MASTECTOMIA RADICAL UNILATERAL	CUPS	Procedure	Treatment
854501	ESCISION DE MAMA, MUSCULOS PECTORALES Y GANGLIO LINFATICO RE	CUPS	Procedure	Treatment
854502	MASTECTOMIA RADICAL MODIFICADA UNILATERAL	CUPS	Procedure	Treatment
8546	MASTECTOMIA RADICAL BILATERAL	CUPS	Procedure	Treatment
854601	MASTECTOMIA RADICAL BILATERAL VIA ABIERTA	CUPS	Procedure	Treatment
8547	MASTECTOMIA RADICAL AMPLIADA UNILATERAL	CUPS	Procedure	Treatment
854701	ESCISION DE MAMA, MUSCULOS, GANGLIOS LINFATICOS (AXILARES, C	CUPS	Procedure	Treatment
8548	MASTECTOMIA RADICAL AMPLIADA BILATERAL	CUPS	Procedure	Treatment
854801	MASTECTOMIA RADICAL AMPLIADA BILATERAL VIA ABIERTA	CUPS	Procedure	Treatment
9224	TELETERAPIA CON ACELERADOR LINEAL CON FOTONES	CUPS	Procedure	Treatment
922441	TELETERAPIA CON ACELERADOR LINEAL (PLANEACION COMPUTARIZADA	CUPS	Procedure	Treatment
922442	TELETERAPIA CON ACELERADOR LINEAL (PLANEACION COMPUTARIZADA	CUPS	Procedure	Treatment
922443	TELETERAPIA CON ACELERADOR LINEAL (PLANEACION COMPUTARIZADA	CUPS	Procedure	Treatment
922444	TELETERAPIA CON ACELERADOR LINEAL (PLANEACION COMPUTARIZADA	CUPS	Procedure	Treatment
922445	TELETERAPIA CON ACELERADOR LINEAL (PLANEACION COMPUTARIZADA	CUPS	Procedure	Treatment
922446	TELETERAPIA CON ACELERADOR LINEAL (PLANEACION COMPUTARIZADA	CUPS	Procedure	Treatment
9225	TELETERAPIA CON ELECTRONES	CUPS	Procedure	Treatment
922504	TELETERAPIA CON ACELERADOR LINEAL DE ELECTRONES (PLANEACION	CUPS	Procedure	Treatment
922505	TELETERAPIA CON ACELERADOR LINEAL DE ELECTRONES (PLANEACION	CUPS	Procedure	Treatment
922506	RADIOTERAPIA INTRAOPERATORIA	CUPS	Procedure	Treatment
9226	BRAQUITERAPIA	CUPS	Procedure	Treatment
922605	BRAQUITERAPIA INTRACAVITARIA (PLANEACION COMPUTARIZADA BIDIM	CUPS	Procedure	Treatment
922606	BRAQUITERAPIA INTRACAVITARIA (PLANEACION COMPUTARIZADA BIDIM	CUPS	Procedure	Treatment
922607	BRAQUITERAPIA INTRACAVITARIA (PLANEACION COMPUTARIZADA TRIDI	CUPS	Procedure	Treatment
922608	BRAQUITERAPIA INTRACAVITARIA (PLANEACION COMPUTARIZADA TRIDI	CUPS	Procedure	Treatment
922615	BRAQUITERAPIA INTERSTICIAL (PLANEACION COMPUTARIZADA BIDIMEN	CUPS	Procedure	Treatment
922616	BRAQUITERAPIA INTERSTICIAL (PLANEACION COMPUTARIZADA TRIDIME	CUPS	Procedure	Treatment
992504	POLITERAPIA ANTINEOPLASICA DE BAJA TOXICIDAD	CUPS	Medication	Treatment
992505	POLITERAPIA ANTINEOPLASICA DE ALTA TOXICIDAD	CUPS	Medication	Treatment
992511	MONOTERAPIA ANTINEOPLASICA DE ALTA TOXICIDAD	CUPS	Medication	Treatment

TABLE C.3: Activities for Lung Cancer

Code	Name	Code Type	Activity Type	Purpose
L01BA04	PEMETREXED	ATC	Medication	Treatment
L01EB03	AFATINIB	ATC	Medication	Treatment
L01ED03	ALECTINIB	ATC	Medication	Treatment
L01XC28	DURVALUMAB	ATC	Medication	Treatment
L01XE02	GEFITINIB	ATC	Medication	Treatment
L01XE03	ERLOTINIB	ATC	Medication	Treatment
L01XE35	OSIMERTINIB	ATC	Medication	Treatment
3321	BRONCOSCOPIAS A TRAVES DE ESTOMA ARTIFICIAL	CUPS	Procedure	Diagnosis
332101	BRONCOSCOPIA A TRAVES DE ESTOMA ARTIFICIAL	CUPS	Procedure	Diagnosis
3322	BRONCOSCOPIAS	CUPS	Procedure	Diagnosis
332201	BRONCOSCOPIA CON LAVADO BRONQUIAL	CUPS	Procedure	Diagnosis
332202	BRONCOSCOPIA	CUPS	Procedure	Diagnosis
332203	BRONCOSCOPIA CON LAVADO BRONCOALVEOLAR	CUPS	Procedure	Diagnosis
332204	BRONCOSCOPIA CON CEPILLADO	CUPS	Procedure	Diagnosis
332205	BRONCOSCOPIA CON APLICACION O RETIRO DE FUENTE RADIACTIVA	CUPS	Procedure	Diagnosis
332206	BRONCOSCOPIA CON PUNCION (ASPIRACION) TRANSTRAQUEAL	CUPS	Procedure	Diagnosis
332207	BRONCOSCOPIA CON PUNCION (ASPIRACION) TRANSBRONQUIAL	CUPS	Procedure	Diagnosis
332208	BRONCOSCOPIA CON AUTOFLUORESCENCIA	CUPS	Procedure	Diagnosis
332209	BRONCOSCOPIA CON TOMOGRAFIA DE COHERENCIA OPTICA	CUPS	Procedure	Diagnosis
3324	BIOPSIA BRONQUIAL VIA ENDOSCOPICA	CUPS	Procedure	Diagnosis
332401	BIOPSIA DE BRONQUIO VIA ENDOSCOPICA	CUPS	Procedure	Diagnosis
3325	BIOPSIA BRONQUIAL VIA ABIERTA	CUPS	Procedure	Diagnosis
332501	BIOPSIA DE BRONQUIO VIA ABIERTA	CUPS	Procedure	Diagnosis
3326	BIOPSIAS CERRADAS DE PULMON VIA PERCUTANEA	CUPS	Procedure	Diagnosis
332601	BIOPSIA CERRADA DE PULMON VIA PERCUTANEA	CUPS	Procedure	Diagnosis
3327	BIOPSIAS DE PULMON VIA ENDOSCOPICA	CUPS	Procedure	Diagnosis
332703	BIOPSIA DE PULMON VIA ENDOSCOPICA	CUPS	Procedure	Diagnosis
332704	BIOPSIA DE PULMON POR TORACOSCOPIA	CUPS	Procedure	Diagnosis
3328	BIOPSIAS DE PULMON VIA ABIERTA	CUPS	Procedure	Diagnosis
332801	BIOPSIA DE PULMON VIA ABIERTA	CUPS	Procedure	Diagnosis
341001	MEDIASTINOSCOPIA DIAGNOSTICA	CUPS	Procedure	Diagnosis
341201	BIOPSIA DE ORGANO O TEJIDO DE MEDIASTINO VIA PERCUTANEA	CUPS	Procedure	Diagnosis
341202	BIOPSIA DE ORGANO O TEJIDO DE MEDIASTINO VIA ABIERTA	CUPS	Procedure	Diagnosis
341203	BIOPSIA DE ORGANO O TEJIDO DE MEDIASTINO POR MEDIASTINOSCOPI	CUPS	Procedure	Diagnosis
341204	BIOPSIA DE ORGANO O TEJIDO DE MEDIASTINO POR TORACOSCOPIA	CUPS	Procedure	Diagnosis
341205	BIOPSIA DE ORGANO O TEJIDO DE MEDIASTINO POR BRONCOSCOPIA	CUPS	Procedure	Diagnosis
34201	TORACOTOMIA EXPLORATORIA	CUPS	Procedure	Diagnosis
345401	BIOPSIA DE PLEURA PERCUTANEA	CUPS	Procedure	Diagnosis
345402	BIOPSIAS DE PLEURA VIA ABIERTA	CUPS	Procedure	Diagnosis
345403	BIOPSIAS DE PLEURA POR TORACOSCOPIA	CUPS	Procedure	Diagnosis
32	PROCEDIMIENTOS EN BRONQUIO Y PULMON	CUPS	Procedure	Diagnosis and Treatment
33	OTROS PROCEDIMIENTOS EN BRONQUIO Y PULMON	CUPS	Procedure	Diagnosis and Treatment
332	PROCEDIMIENTOS EN PULMON Y BRONQUIO	CUPS	Procedure	Diagnosis and Treatment
890271	CONSULTA DE PRIMERA VEZ POR ESPECIALISTA EN NEUMOLOGIA	CUPS	Procedure	Diagnosis and Treatment
890371	CONSULTA DE CONTROL O DE SEGUIMIENTO POR ESPECIALISTA EN NEU	CUPS	Procedure	Diagnosis and Treatment
890471	INTERCONSULTA POR ESPECIALISTA EN NEUMOLOGIA	CUPS	Procedure	Diagnosis and Treatment
320	ESCISION O ABLACION DE LESION O TEJIDO BRONQUIAL	CUPS	Procedure	Treatment
3200	RESECCION O ABLACION DE LESION O TEJIDO EN BRONQUIO CON BRON	CUPS	Procedure	Treatment
320001	RESECCION O ABLACION DE LESION O TEJIDO EN BRONQUIO CON BRON	CUPS	Procedure	Treatment
320002	RESECCION O ABLACION DE LESION O TEJIDO EN BRONQUIO CON BRON	CUPS	Procedure	Treatment
320003	RESECCION O ABLACION DE LESION O TEJIDO EN BRONQUIO CON BRON	CUPS	Procedure	Treatment
3202	RESECCION O ABLACION DE LESION O TEJIDO BRONQUIAL VIA ENDOSC	CUPS	Procedure	Treatment
320201	RESECCION DE LESION EN BRONQUIO VIA ENDOSCOPICA	CUPS	Procedure	Treatment
320203	RECANALIZACION DE BRONQUIO VIA ENDOSCOPICA	CUPS	Procedure	Treatment
321	PROCEDIMIENTOS DE REPARACION EN PULMON Y BRONQUIO	CUPS	Procedure	Treatment
3210	CIERRE DE FISTULA BRONQUIAL	CUPS	Procedure	Treatment

Code	Name	Code Type	Activity Type	Purpose
321001	CIERRE DE FISTULA BRONCOCUTANEA O BRONCOPLEURAL VIA ABIERTA	CUPS	Procedure	Treatment
321002	CIERRE DE FISTULA BRONCOCUTANEA O BRONCOPLEURAL VIA ENDOSCOP	CUPS	Procedure	Treatment
321003	CIERRE DE FISTULA BRONCOCUTANEA O BRONCOPLEURAL POR TORACOSC	CUPS	Procedure	Treatment
321004	CIERRE DE BRONCOSTOMIA VIA ABIERTA	CUPS	Procedure	Treatment
321005	CIERRE DE BRONCOSTOMIA VIA ENDOSCOPICA	CUPS	Procedure	Treatment
321006	CIERRE DE BRONCOSTOMIA POR TORACOSCOPIA	CUPS	Procedure	Treatment
3211	BLOQUEO DE BRONQUIO	CUPS	Procedure	Treatment
321101	BLOQUEO DE BRONQUIO VIA ENDOSCOPICA	CUPS	Procedure	Treatment
3212	RECONSTRUCCION DE BRONQUIO [BRONCOPLASTIA]	CUPS	Procedure	Treatment
321201	BRONCOPLASTIA VIA ABIERTA	CUPS	Procedure	Treatment
321202	BRONCOPLASTIA VIA ENDOSCOPICA	CUPS	Procedure	Treatment
321203	BRONCOPLASTIA POR TORACOSCOPIA	CUPS	Procedure	Treatment
321204	RESECCION EN MANGUITO CON BRONCOPLASTIA VIA ABIERTA	CUPS	Procedure	Treatment
321205	RESECCION EN MANGUITO CON BRONCOPLASTIA POR TORACOSCOPIA	CUPS	Procedure	Treatment
3213	CIERRE DE LACERACION DE BRONQUIO Y PULMON	CUPS	Procedure	Treatment
321301	BRONCORRAFIA VIA ABIERTA	CUPS	Procedure	Treatment
321302	BRONCORRAFIA POR TORACOSCOPIA	CUPS	Procedure	Treatment
321303	NEUMORRAFIA VIA ABIERTA	CUPS	Procedure	Treatment
321304	NEUMORRAFIA POR TORACOSCOPIA	CUPS	Procedure	Treatment
3214	DILATACION BRONQUIAL	CUPS	Procedure	Treatment
321401	DILATACION DE BRONQUIO VIA ENDOSCOPICA	CUPS	Procedure	Treatment
3215	INSERCION DE DISPOSITIVOS EN BRONQUIO	CUPS	Procedure	Treatment
321501	IMPLANTE O SUSTITUCION DE DISPOSITIVO EN BRONQUIO VIA ENDOSC	CUPS	Procedure	Treatment
3216	EXTRACCION DE DISPOSITIVOS EN BRONQUIO	CUPS	Procedure	Treatment
321601	RETIRO DE DISPOSITIVO EN BRONQUIO VIA ENDOSCOPICA	CUPS	Procedure	Treatment
3217	INYECCION DE SUSTANCIA TERAPEUTICA EN BRONQUIO O PULMON	CUPS	Procedure	Treatment
321701	INYECCION DE SUSTANCIA TERAPEUTICA EN BRONQUIO O PULMON VIA	CUPS	Procedure	Treatment
322	ESCISION O ABLACION DE LESION O TEJIDO PULMONAR	CUPS	Procedure	Treatment
322201	REDUCCION DE VOLUMEN PULMONAR VIA ABIERTA	CUPS	Procedure	Treatment
322202	REDUCCION DE VOLUMEN PULMONAR VIA ENDOSCOPICA	CUPS	Procedure	Treatment
322203	REDUCCION DE VOLUMEN PULMONAR POR TORACOSCOPIA	CUPS	Procedure	Treatment
3228	RESECCION O ABLACION DE LESION O TEJIDO PULMONAR	CUPS	Procedure	Treatment
322801	RESECCION O ABLACION DE LESION O TEJIDO PULMONAR VIA ENDOSCO	CUPS	Procedure	Treatment
324	LOBECTOMIA DE PULMON	CUPS	Procedure	Treatment
3241	LOBECTOMIA SEGMENTARIA O RESECCION EN CUÑA	CUPS	Procedure	Treatment
324101	LOBECTOMIA SEGMENTARIA VIA ABIERTA	CUPS	Procedure	Treatment
324102	LOBECTOMIA SEGMENTARIA POR TORACOSCOPIA	CUPS	Procedure	Treatment
324103	RESECCION EN CUÑA VIA ABIERTA	CUPS	Procedure	Treatment
324104	RESECCION EN CUÑA POR TORACOSCOPIA	CUPS	Procedure	Treatment
324105	RESECCION DE METASTASIS PULMONARES VIA ABIERTA	CUPS	Procedure	Treatment
324106	RESECCION DE METASTASIS PULMONARES POR TORACOSCOPIA	CUPS	Procedure	Treatment
3242	LOBECTOMIA TOTAL PULMONAR	CUPS	Procedure	Treatment
324201	LOBECTOMIA TOTAL PULMONAR VIA ABIERTA	CUPS	Procedure	Treatment
324202	LOBECTOMIA TOTAL PULMONAR POR TORACOSCOPIA	CUPS	Procedure	Treatment
324203	BILOBECTOMIA PULMONAR VIA ABIERTA	CUPS	Procedure	Treatment
324204	BILOBECTOMIA PULMONAR POR TORACOSCOPIA	CUPS	Procedure	Treatment
324205	LOBECTOMIA TOTAL PULMONAR (DONANTE VIVO) VIA ABIERTA	CUPS	Procedure	Treatment
324206	LOBECTOMIA TOTAL PULMONAR (DONANTE VIVO) POR TORACOSCOPIA	CUPS	Procedure	Treatment
325	NEUMONECTOMIA	CUPS	Procedure	Treatment
3251	NEUMONECTOMIA SIMPLE	CUPS	Procedure	Treatment
325101	NEUMONECTOMIA SIMPLE VIA ABIERTA	CUPS	Procedure	Treatment
325102	NEUMONECTOMIA SIMPLE POR TORACOSCOPIA	CUPS	Procedure	Treatment
3252	NEUMONECTOMIA RADICAL	CUPS	Procedure	Treatment
325201	NEUMONECTOMIA RADICAL VIA ABIERTA	CUPS	Procedure	Treatment
325202	NEUMONECTOMIA RADICAL POR TORACOSCOPIA	CUPS	Procedure	Treatment
3253	NEUMONECTOMIA CON DECORTICACION CONCOMITANTE [PLEURONEUMONEC	CUPS	Procedure	Treatment
325301	NEUMONECTOMIA CON DECORTICACION CONCOMITANTE [PLEURONEUMONEC	CUPS	Procedure	Treatment
325302	PLEURONEUMOPERICARDIECTOMIA EXTRAPLEURAL CON RECONSTRUCCION	CUPS	Procedure	Treatment
325303	NEUMONECTOMIA CON DECORTICACION CONCOMITANTE [PLEURONEUMONEC	CUPS	Procedure	Treatment

Code	Name	Code Type	Activity Type	Purpose
326	DISECCION DE ESTRUCTURAS TORACICAS	CUPS	Procedure	Treatment
3261	DISECCION EN BLOQUE DE ESTRUCTURAS TORACICAS	CUPS	Procedure	Treatment
326101	DISECCION EN (BLOQUE) DE BRONQUIO, LOBULO DE PULMON, PLEJO B	CUPS	Procedure	Treatment
327	TRASPLANTE DE PULMON	CUPS	Procedure	Treatment
3270	TRASPLANTE UNILATERAL DE PULMON	CUPS	Procedure	Treatment
327001	TRASPLANTE UNILATERAL DE PULMON VIA ABIERTA	CUPS	Procedure	Treatment
3271	TRASPLANTE BILATERAL DE PULMON	CUPS	Procedure	Treatment
327101	TRASPLANTE BILATERAL DE PULMON VIA ABIERTA	CUPS	Procedure	Treatment
328	TRASPLANTE COMBINADO DE PULMON CORAZON	CUPS	Procedure	Treatment
3280	TRASPLANTE DE PULMON CORAZON	CUPS	Procedure	Treatment
328001	TRASPLANTE DE PULMON CORAZON VIA ABIERTA	CUPS	Procedure	Treatment
332210	BRONCOSCOPIA CON TERMOPLASTIA BRONQUIAL	CUPS	Procedure	Treatment
3329	EXTRACCION DE CUERPO EXTRAÑO EN BRONQUIO O PULMON	CUPS	Procedure	Treatment
332901	EXTRACCION DE CUERPO EXTRAÑO DE BRONQUIO O PULMON VIA ABIERT	CUPS	Procedure	Treatment
332902	EXTRACCION DE CUERPO EXTRAÑO DE BRONQUIO O PULMON VIA ENDOSC	CUPS	Procedure	Treatment
332903	EXTRACCION DE CUERPO EXTRAÑO DE BRONQUIO O PULMON POR TORACO	CUPS	Procedure	Treatment
340501	BIOPSIA DE LESION DE PARED TORACICA VIA PERCUTANEA	CUPS	Procedure	Treatment
340502	BIOPSIA DE LESION DE PARED TORACICA VIA ABIERTA	CUPS	Procedure	Treatment
340601	ESCISION O ABLACION DE LESION DE PARED TORACICA POR TORACOTO	CUPS	Procedure	Treatment
340602	ESCISION O ABLACION RADICAL DE PARED TORACICA	CUPS	Procedure	Treatment
340905	TORACOPLASTIA CON CIERRE DE FISTULA BRONCOPLEURAL	CUPS	Procedure	Treatment
340906	TORACOPLASTIA EXTRAPLEURAL	CUPS	Procedure	Treatment
341101	EXPLORACION Y DRENAJE DE MEDIASTINO POR MEDIASTINOTOMIA	CUPS	Procedure	Treatment
341104	EXPLORACION Y DRENAJE DE MEDIASTINO POR ESTERNOTOMIA	CUPS	Procedure	Treatment
341105	EXPLORACION Y DRENAJE DE MEDIASTINO POR TORACOTOMIA	CUPS	Procedure	Treatment
341106	EXPLORACION Y DRENAJE DE MEDIASTINO POR TORACOSCOPIA	CUPS	Procedure	Treatment
341401	RESECCION DE TUMOR MALIGNO DEL MEDIASTINO POR TORACOTOMIA	CUPS	Procedure	Treatment
341402	RESECCION DE TUMOR MALIGNO DEL MEDIASTINO POR ESTERNOTOMIA	CUPS	Procedure	Treatment
341403	RESECCION DE TUMOR MALIGNO DEL MEDIASTINO POR TORACOSCOPIA	CUPS	Procedure	Treatment
342101	TORACOSCOPIA DIAGNOSTICA	CUPS	Procedure	Treatment
345001	TORACENTESIS DIAGNOSTICA	CUPS	Procedure	Treatment
345002	TORACENTESIS DE DRENAJE O DESCOMPRESIVA	CUPS	Procedure	Treatment
345101	PLEURECTOMIA PARIETAL VIA ABIERTA	CUPS	Procedure	Treatment
345102	PLEURECTOMIA PARIETAL POR TORACOSCOPIA	CUPS	Procedure	Treatment
345201	PLEURODESIS QUIMICA VIA ABIERTA	CUPS	Procedure	Treatment
345202	PLEURODESIS QUIMICA POR TORACOSCOPIA	CUPS	Procedure	Treatment
345203	PLEURODESIS QUIMICA POR TORACOSTOMIA CERRADA	CUPS	Procedure	Treatment
345204	PLEURODESIS MECANICA VIA ABIERTA	CUPS	Procedure	Treatment
345205	PLEURODESIS MECANICA POR TORACOSCOPIA	CUPS	Procedure	Treatment
3453	DECORTICACION PULMONAR	CUPS	Procedure	Treatment
345301	DECORTICACION PULMONAR VIA ABIERTA	CUPS	Procedure	Treatment
345302	DECORTICACION PULMONAR POR TORACOSCOPIA	CUPS	Procedure	Treatment
345501	RESECCION DE TUMOR DE PLEURA VIA ABIERTA	CUPS	Procedure	Treatment
345502	RESECCION DE TUMOR DE PLEURA POR TORACOSCOPIA	CUPS	Procedure	Treatment
345601	COLOCACION DE CATETER PLEURAL PERMANENTE	CUPS	Procedure	Treatment

TABLE C.4: Activities for Cervical Cancer

Code	Name	Code Type	Activity Type	Purpose
L01XA02	CARBOPLATINO	ATC	Medication	Treatment
671	PROCEDIMIENTOS DIAGNOSTICOS EN CUELLO UTERINO (CERVIX)	CUPS	Procedure	Diagnosis
6712	BIOPSIA DE CUELLO UTERINO	CUPS	Procedure	Diagnosis
671201	BIOPSIA EN SACABOCADO DE CUELLO UTERINO	CUPS	Procedure	Diagnosis
671202	BIOPSIA DE CUELLO UTERINO CIRCUNFERENCIAL	CUPS	Procedure	Diagnosis
672	CONIZACIONES	CUPS	Procedure	Diagnosis
6720	CONIZACION	CUPS	Procedure	Diagnosis
672001	CONIZACION CERVICAL	CUPS	Procedure	Diagnosis
681	PROCEDIMIENTOS DIAGNOSTICOS EN UTERO Y ESTRUCTURRAS DE SOPOR	CUPS	Procedure	Diagnosis
6811	BIOPSIAS DE UTERO	CUPS	Procedure	Diagnosis
681101	BIOPSIAS DE UTERO POR LAPAROTOMIA	CUPS	Procedure	Diagnosis
681102	BIOPSIAS DE UERO POR LAPAROSCOPIA	CUPS	Procedure	Diagnosis
681103	BIOPSIAS DE LIGAMENTOS UTERINOS POR LAPAROTOMIA	CUPS	Procedure	Diagnosis
681104	BIOPSIAS DE LIGAMENTOS UTERINOS POR LAPAROSCOPIA	CUPS	Procedure	Diagnosis
681105	BIOPSIA DE ENDOMETRIA	CUPS	Procedure	Diagnosis
7022	COLPOSCOPIAS	CUPS	Procedure	Diagnosis
702203	COLPOSCOPIA	CUPS	Procedure	Diagnosis
8814	ECOGRAFIA DE PELVIS Y DE GENITALES FEMENINOS	CUPS	Procedure	Diagnosis
881401	ECOGRAFIA PELVICA GINECOLOGICA TRANSVAGINAL	CUPS	Procedure	Diagnosis
67	PROCEDIMIENTOS EN CUELLO UTERINO	CUPS	Procedure	Diagnosis and Treatment
68	PROCEDIMIENTOS EN UTERO	CUPS	Procedure	Diagnosis and Treatment
6812	HISTEROSCOPIAS	CUPS	Procedure	Diagnosis and Treatment
681201	HISTEROSCOPIA	CUPS	Procedure	Diagnosis and Treatment
670	DILATACION DEL CANAL CERVICAL	CUPS	Procedure	Treatment
6701	DILATACION Y CURETAJE DE MUÑON CERVICAL	CUPS	Procedure	Treatment
670101	DILATACION Y CURETAJE DE MUÑON CERVICAL	CUPS	Procedure	Treatment
673	RESECCION O ESCISION O ABLACION DE TEJIDOS DE CUELLO UTERINO	CUPS	Procedure	Treatment
6731	RESECCION DE LESION EN CUELLO UTERINO	CUPS	Procedure	Treatment
673101	ESCISION DE POLIPO EN CUELLO UTERINO	CUPS	Procedure	Treatment
673102	RESECCION DE LESION EN CUELLO UTERINO	CUPS	Procedure	Treatment
6732	ABLACION DE LESION O TEJIDO EN CUELLO UTERINO	CUPS	Procedure	Treatment
673201	ABLACION DE LESION O TEJIDO EN CUELLO UTERINO	CUPS	Procedure	Treatment
674	AMPUTACION EN CUELLO UTERINO (CERVIX)	CUPS	Procedure	Treatment
6740	AMPUTACION DEL CUELLO UTERINO	CUPS	Procedure	Treatment
674002	AMPUTACION DE CUELLO O TRAQUELECTOMIA POR LAPAROTOMIA	CUPS	Procedure	Treatment
674003	AMPUTACION DE CUELLO O TRAQUELECTOMIA POR LAPAROSCOPIA	CUPS	Procedure	Treatment
674004	AMPUTACION DE CUELLO O TRAQUELECTOMIA POR VIA VAGINAL	CUPS	Procedure	Treatment
6741	ESCISION DE MUÑON CERVICAL VIA VAGINAL O ABDOMINAL	CUPS	Procedure	Treatment
674101	ESCISION DE MUÑON CERVICAL POR LAPAROTOMIA	CUPS	Procedure	Treatment
674102	ESCISION DE MUÑON CERVICAL POR LAPAROSCOPIA	CUPS	Procedure	Treatment
674103	ESCISION DE MUÑON CERVICAL POR VIA VAGINAL	CUPS	Procedure	Treatment
6745	TRAQUELECTOMIA RADICAL	CUPS	Procedure	Treatment
674501	TRAQUELECTOMIA RADICAL POR LAPAROTOMIA	CUPS	Procedure	Treatment
674511	TRAQUELECTOMIA RAADICAL POR LAPAROSCOPIA	CUPS	Procedure	Treatment
675	REPARACION DE ORIFCIO INTERNO DE CUELLO UTERINO	CUPS	Procedure	Treatment
6761	SUTURA DE LACERACION O DESGARRO DE CUELLO UTERINO	CUPS	Procedure	Treatment
676101	SUTURA DE LACERACION O DESGARRO DE CUELLO UTERINO (CERVIX) V	CUPS	Procedure	Treatment
6762	CORRECCION DE FISTULA EN CUELLO UTERINO (CERVIX)	CUPS	Procedure	Treatment
676210	FISTULECTOMIA CERVICOSIGMOIDAL	CUPS	Procedure	Treatment
6769	OTRAS CORRECIONES O PLASTIAS DE CUELLO UTERINO (CERVIX)	CUPS	Procedure	Treatment
682	ESCISION O ABLACION DE LESIONES DE TEJIDO UTERINO	CUPS	Procedure	Treatment
684	HISTERECTOMIA TOTAL	CUPS	Procedure	Treatment
6840	HISTERECTOMIA TOTAL ABDOMINAL	CUPS	Procedure	Treatment
684001	HISTERECTOMIA TOTAL ABDOMINAL CON REMOCION DE MOLA O FETO MU	CUPS	Procedure	Treatment
684003	HISTERECTOMIA TOTAL POR LAPAROTOMIA	CUPS	Procedure	Treatment
684020	HISTERECTOMIA TOTAL POR LAPAROSCOPIA	CUPS	Procedure	Treatment

Code	Name	Code Type	Activity Type	Purpose
6841	HISTERECTOMIA TOTAL ABDOMINAL AMPLIADA	CUPS	Procedure	Treatment
684103	HISTERECTOMIA TOTAL ABDOMINAL AMPLIADA POR LAPAROTOMIA	CUPS	Procedure	Treatment
684104	HISTERECTOMIA TOTAL ABDOMINAL AMPLIADA POR LAPAROSCOPIA	CUPS	Procedure	Treatment
9223	TELETERAPIA CON RADIOISOTOPOS	CUPS	Procedure	Treatment
922321	TELETERAPIA CON COBALTO (PLANEACION COMPUTARIZADA BIDIMENSIO	CUPS	Procedure	Treatment
922322	TELETERAPIA CON COBALTO (PLANEACION COMPUTARIZADA TRIDIMENSI	CUPS	Procedure	Treatment
92241	TELETERAPIA CON ACELERADOR LINEAL (PLANEACION COMPUTARIZADA	CUPS	Procedure	Treatment
92242	TELETERAPIA CON ACELERADOR LINEAL (PLANEACION COMPUTARIZADA	CUPS	Procedure	Treatment
92243	TELETERAPIA CON ACELERADOR LINEAL (PLANEACION COMPUTARIZADA	CUPS	Procedure	Treatment
922603	BRAQUITERAPIA INTRALUMINAL (PLANEACION COMPUTARIZADA BIDIMEN	CUPS	Procedure	Treatment
922606	BRAQUITERAPIA INTRACAVITARIA (PLANEACION COMPUTARIZADA TRIDI	CUPS	Procedure	Treatment

TABLE C.5: Activities for Stomach Cancer

Code	Name	Code Type	Activity Type	Purpose
441	PROCEDIMIENTOS DIAGNOSTICOS EN EL ESTOMAGO	CUPS	Procedure	Diagnosis
4411	GASTROSCOPIA TRANSABDOMINAL	CUPS	Procedure	Diagnosis
441101	GASTROSCOPIA TRANSABDOMINAL (INTRAQUIRURGICA) VIA ABIERTA	CUPS	Procedure	Diagnosis
441102	GASTROSCOPIA TRANSABDOMINAL (INTRAQUIRURGICA) VIA LAPAROSCOP	CUPS	Procedure	Diagnosis
4412	GASTROSCOPIA A TRAVES DE ESTOMA ARTIFICIAL	CUPS	Procedure	Diagnosis
441200	GASTROSCOPIA A TRAVES DE ESTOMA ARTIFICIAL SOD	CUPS	Procedure	Diagnosis
4413	ESOFAGOGASTRODUODENOSCOPIA	CUPS	Procedure	Diagnosis
441303	ESOFAGOGASTRODUODENOSCOPIA [EGD] CON MAGNIFICACION O CROMOEN	CUPS	Procedure	Diagnosis
441304	MARCACION DE LESION EN ESOFAGO ESTOMAGO O DUODENO VIA ENDOSC	CUPS	Procedure	Diagnosis
4415	BIOPSIA ABIERTA DEL ESTOMAGO	CUPS	Procedure	Diagnosis
441501	BIOPSIA DE ESTOMAGO VIA ABIERTA	CUPS	Procedure	Diagnosis
441502	BIOPSIA DE ESTOMAGO VIA LAPAROSCOPICA	CUPS	Procedure	Diagnosis
893909	ELECTROGASTROGRAFIA TRANSCUTANEA O INTRAGASTRICA	CUPS	Procedure	Diagnosis
920506	MEDICION DE ABSORCION GASTROINTESTINAL DE VITAMINA B12	CUPS	Procedure	Diagnosis
9206	GAMAGRAFIA, ESTUDIOS ISOTOPICOS FUNCIONALES Y MORFOLOGICOS D	CUPS	Procedure	Diagnosis
920606	GAMAGRAFIA DE REFLUJO GASTROESOFAGICO	CUPS	Procedure	Diagnosis
920607	GAMAGRAFIA DE VACIAMIENTO GASTRCO EN FASE SOLIDA	CUPS	Procedure	Diagnosis
920608	GAMAGRAFIA DE VACIAMIENTO GASTRICO EN FASE LIQUIDA	CUPS	Procedure	Diagnosis
9213	GAMAGRAFIA DE VIABILIDAD TUMORAL (RASTREO GAMAGRAFICO)	CUPS	Procedure	Diagnosis
9216	GAMAGRAFIA DE ANTICUERPOS MONOCLONALES	CUPS	Procedure	Diagnosis
9217	GAMAGRAFIA CON DMSA PENTAVALENTE	CUPS	Procedure	Diagnosis
921700	GAMAGRAFIA CON DMSA. PENTAVALENTE SOD	CUPS	Procedure	Diagnosis
43	PROCEDIMIENTOS DE ESTOMAGO	CUPS	Procedure	Diagnosis and Treatment
434	ESCISION LOCAL ENDOSCOPICA DE LESION O TEJIDO DE ESTOMAGO	CUPS	Procedure	Diagnosis and Treatment
4340	ESCISION ENDOSCOPICA DE POLIPOS GASTRICOS	CUPS	Procedure	Diagnosis and Treatment
434001	ESCISION DE POLIPOS GASTRICOS VIA ENDOSCOPICA	CUPS	Procedure	Diagnosis and Treatment
434102	CONTROL ENDOSCOPICO DE HEMORRAGIA GASTRICA MEDIANTE ESCLEROT	CUPS	Procedure	Diagnosis and Treatment
434103	CONTROL ENDOSCOPICO DE HEMORRAGIA GASTRICA MEDIANTE CORRIENT	CUPS	Procedure	Diagnosis and Treatment
441302	ESOFAGOGASTRODUODENOSCOPIA [EGD] CON O SIN BIOPSIA	CUPS	Procedure	Diagnosis and Treatment
430	INCISION Y ESCICION DE ESTOMAGO	CUPS	Procedure	Treatment
4301	GASTROTOMIA	CUPS	Procedure	Treatment
430102	EXTRACCION DE CUERPO EXTRAÑO MULTIPLE (BEZOARD) POR GASTROTO	CUPS	Procedure	Treatment
430103	EXTRACCION DE CUERPO EXTRAÑO MULTIPLE (BEZOARD) POR GASTROTO	CUPS	Procedure	Treatment
431	GASTROTOMIA	CUPS	Procedure	Treatment
4310	GASTROTOMIAS	CUPS	Procedure	Treatment
431001	GASTROTOMIA VIA ABIERTA	CUPS	Procedure	Treatment
431002	GASTROTOMIA VIA PERCUTANEA (ENDOSCOPIA)	CUPS	Procedure	Treatment
431003	GASTROTOMIA VIA LAPAROSCOPIA	CUPS	Procedure	Treatment
433	PILORMIOTOMIA	CUPS	Procedure	Treatment
4331	PILOROMIOTOMIAS	CUPS	Procedure	Treatment
433101	PILOROMIOTOMIA VIA ABIERTA	CUPS	Procedure	Treatment
433102	PILOROMIOTOMIA VIA LAPAROSCOPIA	CUPS	Procedure	Treatment
4341	ABORDAJE ENDOSCOPICO DE VARICES GASTRICAS	CUPS	Procedure	Treatment
434101	LIGADURA ENDOSCOPICA DE VARICES GASTRICAS	CUPS	Procedure	Treatment
4342	RESECCION ENDOSCOPICA DE LESION O TUMOR SUBMUCOSO GASTRICO	CUPS	Procedure	Treatment
434201	RESECCION DE LESION O TUMOR SUBMUCOSO GASTRICO VIA ENDOSCOPI	CUPS	Procedure	Treatment
4345	MUCOSECTOMIA ENDOSCOPICA GASTRICA	CUPS	Procedure	Treatment
434500	MUCOSECTOMIA ENDOSCOPICA GASTRICA SOD	CUPS	Procedure	Treatment
436	GASTRECTOMIA PARCIAL CON ANASTOMOSIS AL DUODENO	CUPS	Procedure	Treatment
4361	GASTRODUODENOSTOMIA	CUPS	Procedure	Treatment
436101	GASTRODUODENOSTOMIA VIA ABIERTA	CUPS	Procedure	Treatment
436102	GASTRODUODENOSTOMIA VIA LAPAROSCOPIA	CUPS	Procedure	Treatment
437	GASTRECTOMIA PARCIAL CON ANASTOMOSIS AL YEYUNO	CUPS	Procedure	Treatment
4371	GASTROYEYUNOSTOMIA	CUPS	Procedure	Treatment
437101	GASTROYEYUNOSTOMIA VIA ABIERTA	CUPS	Procedure	Treatment
437102	GASTROYEYUNOSTOMIA VIA LAPAROSCOPICA	CUPS	Procedure	Treatment

Code	Name	Code Type	Activity Type	Purpose
438	OTRA GASTRECTOMIA PARCIAL	CUPS	Procedure	Treatment
43803	GASTROENTEROANASTOMOSIS DERIVATIVA (DUODENO O YEYUNO) SIN EX	CUPS	Procedure	Treatment
4381	GASTRECTOMIA SUBTOTAL RADICAL	CUPS	Procedure	Treatment
438101	GASTRECTOMIA SUBTOTAL RADICAL VIA ABIERTA	CUPS	Procedure	Treatment
438102	GASTRECTOMIA SUBTOTAL RADICAL VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
4382	GASTRECTOMIA PARCIAL, CON RECONSTRUCCION CON O SIN VAGOTOMIA	CUPS	Procedure	Treatment
438201	GASTRECTOMIA PARCIAL CON RECONSTRUCCION CON VAGOTOMIA VIA LA	CUPS	Procedure	Treatment
438202	GASTRECTOMIA PARCIAL CON RECONSTRUCCION SIN VAGOTOMIA VIA LA	CUPS	Procedure	Treatment
438203	GASTRECTOMIA PARCIAL CON RECONSTRUCCION SIN VAGOTOMIA VIA AB	CUPS	Procedure	Treatment
438204	GASTRECTOMIA PARCIAL CON RECONSTRUCCION SIN VAGOTOMIA VIA LA	CUPS	Procedure	Treatment
4383	GASTROENTEROANASTOMOSIS DERIVATIVA (DUODENO O YEYUNO) CON EX	CUPS	Procedure	Treatment
438301	GASTROENTEROANASTOMOSIS DERIVATIVA (DUODENO O YEYUNO) CON EX	CUPS	Procedure	Treatment
438302	GASTROENTEROANASTOMOSIS DERIVATIVA (DUODENO O YUYUNO) CON EX	CUPS	Procedure	Treatment
438304	GASTROENTEROANASTOMOSIS DERIVATIVA (DUODENO O YEYUNO) SIN EX	CUPS	Procedure	Treatment
4384	GASTRECTOMIA VERTICAL	CUPS	Procedure	Treatment
438401	GASTRECTOMIA VERTICAL (MANGA GASTRICA) VIA ABIERTA	CUPS	Procedure	Treatment
4384102	GASTRECTOMIA VERTICAL (MANGA GASTRICA) POR LAPAROSCOPIA	CUPS	Procedure	Treatment
4385	REINVITENCION GASTRECTOMIA VERTICAL	CUPS	Procedure	Treatment
438501	REINTERVENCION O REVISION DE GASTRECTOMIA VERTICAL (MANGA GA	CUPS	Procedure	Treatment
438503	CONVERSION DE GASTRECTOMIA VERTICAL (MANGA GASTRICA) A OTRA	CUPS	Procedure	Treatment
438504	CONVERSION DE GASTRECTOMIA VERTICAL (MANGA GASTRICA) A OTRA	CUPS	Procedure	Treatment
4385102	REINTERVENCION O REVISION DE GASTRECTOMIA VERTICAL (MANGA GA	CUPS	Procedure	Treatment
439	GASTRECTOMIA TOTAL	CUPS	Procedure	Treatment
4390	GASTRECTOMIA TOTAL O TOTAL RADICAL	CUPS	Procedure	Treatment
439001	GASTRECTOMIA TOTAL VIA ABIERTA	CUPS	Procedure	Treatment
439002	GASTRECTOMIA TOTAL VIA LAPAROSCOPIA	CUPS	Procedure	Treatment
439003	GASTRECTOMIA TOTAL RADICAL VIA ABIERTA	CUPS	Procedure	Treatment
439004	GASTRECTOMIA TOTAL RADICAL VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
4391	RECONSTRUCCION GASTRICA CON INTERPOSICION INTESTINAL	CUPS	Procedure	Treatment
439101	RECOSTRUCCION GASTRICA CON INTERPOSICION INTESTINAL VIA ABIE	CUPS	Procedure	Treatment
439102	RECOSTRUCCION GASTRICA CON INTERPOSICION INTESTINAL VIA LAPA	CUPS	Procedure	Treatment
4392	RECONSTRUCCION GASTROINTESTINAL EN Y DE ROUX	CUPS	Procedure	Treatment
439201	RECONSTRUCCION GASTROINTESTINAL EN Y DE ROUX VIA ABIERTA	CUPS	Procedure	Treatment
439202	RECONSTRUCCION GASTROINTESTINAL EN Y DE ROUX VIA LAPAROSCOPI	CUPS	Procedure	Treatment
4393	ESOFAGOGASTRECTOMIA	CUPS	Procedure	Treatment
439301	ESOFAGOGASTRECTOMIA VIA ABIERTA	CUPS	Procedure	Treatment
439302	ESOFAGOGASTRECTOMIA VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
441301	ESOFAGOGASTRODUODENOSCOPIA [EGD] CON EXTRACCION DE CUERPO EX	CUPS	Procedure	Treatment
442	PILOROPLASTIA	CUPS	Procedure	Treatment
4421	DILATACION DE PILORO MEDIANTE INCISION	CUPS	Procedure	Treatment
442101	DILATACION DE PILORO MEDIANTE INCISION VIA ABIERTA	CUPS	Procedure	Treatment
442102	DILATACION DE PILORO MEDIANTE INCISION VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
4422	DILATACION ENDOSCOPICA DE PILORO O ANASTOMOSIS GASTROENTERIC	CUPS	Procedure	Treatment
442201	DILATACION DE PILORO VIA ENDOSCOPICA	CUPS	Procedure	Treatment
442202	DILATACION DE ANASTOMOSIS GASTROENTERICA VIA ENDOSCOPICA	CUPS	Procedure	Treatment
4424	PILOROPLASTIAS	CUPS	Procedure	Treatment
442401	PILOROPLASTIAS VIA ABIERTA	CUPS	Procedure	Treatment
442402	PILOROPLASTIAS VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
444	CONTROL DE HEMORRAGIA Y SUTURA DE ULCERA GASTRICA O DUODENAL	CUPS	Procedure	Treatment
4440	SUTURA DE ULCERA PERFORADA CON O SIN VAGOTOMIA CON EPIPLOPLA	CUPS	Procedure	Treatment
444001	SUTURA DE ULCERA PERFORADA CON O SIN VAGOTOMIA CON EPIPLOPLA	CUPS	Procedure	Treatment
444002	SUTURA DE ULCERA PERFORADA CON O SIN VAGOTOMIA CON EPIPLOPLA	CUPS	Procedure	Treatment
4441	SUTURA DE ULCERA GASTRICA	CUPS	Procedure	Treatment
444101	SUTURA DE ULCERA GASTRICA VIA ABIERTA	CUPS	Procedure	Treatment
444102	SUTURA DE ULCERA GASTRICA VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
4443	CONTROL DE HEMORRAGIA GASTRICA O DUODENAL (ENDOSCOPICA)	CUPS	Procedure	Treatment
444305	CONTROL DE HEMORRAGIA GASTRICA O DUODENAL CON DISPOSITIVO VI	CUPS	Procedure	Treatment
445	REVISION DE ANASTOMOSIS GASTRICA	CUPS	Procedure	Treatment
4451	REANASTOMOSIS DEL ESTOMAGO POR DEHISCENCIA DE LA SUTURA	CUPS	Procedure	Treatment

Code	Name	Code Type	Activity Type	Purpose
445101	REANASTOMOSIS DEL ESTOMAGO POR DEHISCENCIA DE LA SUTURA VIA	CUPS	Procedure	Treatment
445102	REANASTOMOSIS DEL ESTOMAGO POR DEHISCENCIA DE LA SUTURA VIA	CUPS	Procedure	Treatment
446	OTRA REPARACION DE ESTOMAGO	CUPS	Procedure	Treatment
4461	SUTURA DE DESGARRO O HERIDA DE ESTOMAGO [GASTRORRAFIA]	CUPS	Procedure	Treatment
446101	SUTURA DE DESGARRO O HERIDA DE ESTOMAGO [GASTRORRAFIA] VIA A	CUPS	Procedure	Treatment
446102	SUTURA DE DESGARRO O HERIDA DE ESTOMAGO [GASTRORRAFIA] VIA L	CUPS	Procedure	Treatment
4462	CIERRE DE GASTROSTOMIA	CUPS	Procedure	Treatment
446201	CIERRE DE GASTROSTOMIA VIA ABIERTA	CUPS	Procedure	Treatment
446202	CIERRE DE GASTROSTOMIA VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
4463	CIERRE DE OTRA FISTULA GASTRICA	CUPS	Procedure	Treatment
446301	CIERRE DE OTRA FISTULA GASTRICA VIA ABIERTA	CUPS	Procedure	Treatment
446302	CIERRE DE OTRA FISTULA GASTRICA VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
446303	CIERRE DE PERFORACION O FISTULA GASTRICA VIA ENDOSCOPICA	CUPS	Procedure	Treatment
4464	GASTROPEXIA	CUPS	Procedure	Treatment
446401	GASTROPEXIA VIA ABIERTA	CUPS	Procedure	Treatment
446402	GASTROPEXIA VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
4465	ESOFAGOGASTROPLASTIA	CUPS	Procedure	Treatment
446501	ESOFAGOGASTROPLASTIA VIA ABIERTA	CUPS	Procedure	Treatment
446502	ESOFAGOGASTROPLASTIA VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
4466	OTROS PROCEDIMIENTOS PARA CREACION DE COMPETENCIA ESFINTERIA	CUPS	Procedure	Treatment
446601	CIRUGIA ANTIRREFLUJO GASTRESOFAGICO CON RECONSTRUCCION DEL E	CUPS	Procedure	Treatment
446602	CIRUGIA ANTIRREFLUJO GASTRESOFAGICO CON RECONSTRUCCION DEL E	CUPS	Procedure	Treatment
446603	REINTERVENCION EN ANTIRREFLUJO GASTRESOFAGICO CON RECONSTRUC	CUPS	Procedure	Treatment
446604	CIRUGIA ANTIRREFLUJO GASTRESOFAGICO MAS RECONSTRUCCION DE ES	CUPS	Procedure	Treatment
449	OTROS PROCEDIMIENTOS EN ESTOMAGO	CUPS	Procedure	Treatment
4490	ABLACION DE LESION GASTRICA	CUPS	Procedure	Treatment
449001	ABLACION DE LESION GASTRICA VIA ENDOSCOPICA	CUPS	Procedure	Treatment
4491	LIGADURA DE VARICES GASTRICAS	CUPS	Procedure	Treatment
449101	LIGADURA DE VARICES GASTRICAS VIA ABIERTA	CUPS	Procedure	Treatment
449102	LIGADURA DE VARICES GASTRICAS VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
4492	MANIPULACION INTRAOPERATORIA DE ESTOMAGO (REDUCCION DE VOLVU	CUPS	Procedure	Treatment
449201	MANIPULACION INTRAOPERATORIA DE ESTOMAGO (REDUCCION DE VOLVU	CUPS	Procedure	Treatment
449202	MANIPULACION INTRAOPERATORIA DE ESTOMAGO (REDUCCION DE VOLVU	CUPS	Procedure	Treatment
4493	INSERCION O REVISION DE DISPOSITIVO GASTRICO	CUPS	Procedure	Treatment
449301	INSERCION DE DISPOSITIVO INTRAGASTRICO RESTRICTIVO POR ENDOS	CUPS	Procedure	Treatment
449302	INSERCION DE DISPOSITIVO PERIGASTRICO RESTRICTIVO (FIJO O AJ	CUPS	Procedure	Treatment
449303	INSERCION DE DISPOSITIVO PERIGASTRICO RESTRICTIVO (FIJO O AJ	CUPS	Procedure	Treatment
449304	REVISION DE DISPOSITIVO PERIGASTRICO RESTRICTIVO (FIJO O AJU	CUPS	Procedure	Treatment
449305	REVISION DE DISPOSITIVO PERIGASTRICO RESTRICTIVO (FIJO O AJU	CUPS	Procedure	Treatment
449306	CONVERSION DE CIRUGIA CON DISPOSITIVO PERIGASTRICO RESTRICTI	CUPS	Procedure	Treatment
449307	CONVERSION DE CIRUGIA CON DISPOSITIVO PERIGASTRICO RESTRICTI	CUPS	Procedure	Treatment
4494	EXTRACCION DE DISPOSITIVO GASTRICO	CUPS	Procedure	Treatment
449401	EXTRACCION DE DISPOSITIVO INTRAGASTRICO RESTRICTIVO POR ENDO	CUPS	Procedure	Treatment
449402	EXTRACCION DE DISPOSITIVO PERIGASTRICO RESTRICTIVO (FIJO O A	CUPS	Procedure	Treatment
449403	EXTRACCION DE DISPOSITIVO PERIGASTRICO RESTRICTIVO (FIJO O A	CUPS	Procedure	Treatment
4495	BAIPAS O DERIVACION O PUENTE DUODENAL PARA REFLUJO DUODENOGA	CUPS	Procedure	Treatment
449501	BAIPAS O DERIVACION O PUENTE DUODENAL PARA REFLUJO DUODENOGA	CUPS	Procedure	Treatment
449502	BAIPAS O DERIVACION O PUENTE DUODENAL PARA REFLUJO DUODENOGA	CUPS	Procedure	Treatment
4496	BAIPAS O DERIVACION O PUENTE GASTRICO	CUPS	Procedure	Treatment
449601	BAIPAS O DERIVACION O PUENTE GASTRICO VIA ABIERTA	CUPS	Procedure	Treatment
449602	BAIPAS O DERIVACION O PUENTE GASTRICO POR LAPAROSCOPIA	CUPS	Procedure	Treatment
922	RADIOTERAPIA	CUPS	Procedure	Treatment
9222	TELETERAPIA ORTOVOLTAJE	CUPS	Procedure	Treatment
922201	TELETERAPIA CON ORTOVOLTAJE	CUPS	Procedure	Treatment
922604	BRAQUITERAPIA INTRALUMINAL CON BAJA TASA DE DOSIS	CUPS	Procedure	Treatment

TABLE C.6: Diagnostic Groups for Model of Care Provision

ICD 10	Description
B20	HIV disease
C00	Malignant neoplasm of lip
C01	Malignant neoplasm of base of tongue
C02	Malignant neoplasm of other and unspecified parts of tongue
C03	Malignant neoplasm of gum
C04	Malignant neoplasm of floor of mouth
C05	Malignant neoplasm of palate
C06	Malignant neoplasm of other and unspecified parts of mouth
C07	Malignant neoplasm of parotid gland
C08	Malignant neoplasm of other and unspecified major salivary glands
C09	Malignant neoplasm of tonsil
C10	Malignant neoplasm of oropharynx
C11	Malignant neoplasm of nasopharynx
C12	Malignant neoplasm of pyriform sinus
C13	Malignant neoplasm of hypopharynx
C14	Malignant neoplasm of other and ill-defined sites in the lip, oral cavity and pharynx
C15	Malignant neoplasm of esophagus
C16	Malignant neoplasm of stomach
C18	Malignant neoplasm of colon
C22	Malignant neoplasm of liver and intrahepatic bile ducts
C23	Malignant neoplasm of gallbladder
C25	Malignant neoplasm of pancreas
C34	Malignant neoplasm of bronchus and lung
C40	Malignant neoplasm of bone and articular cartilage of limbs
C41	Malignant neoplasm of bone and articular cartilage of other and unspecified sites
C43	Malignant melanoma of skin
C44	Other malignant neoplasms of skin
C45	Mesothelioma
C46	Kaposi's sarcoma
C47	Malignant neoplasm of peripheral nerves and autonomic nervous system
C48	Malignant neoplasm of retroperitoneum and peritoneum
C49	Malignant neoplasm of other connective and soft tissue
C50	Malignant neoplasm of breast
C61	Malignant neoplasm of prostate
C64	Malignant neoplasm of kidney
C67	Malignant neoplasm of bladder
C70	Malignant neoplasm of meninges
C71	Malignant neoplasm of brain
C72	Malignant neoplasm of spinal cord, cranial nerves and other parts of central nervous system
C73	Malignant neoplasm of thyroid gland
C76	Malignant neoplasm of other and ill-defined sites
C77	Secondary and unspecified malignant neoplasm of lymph nodes
C78	Secondary malignant neoplasm of respiratory and digestive organs
C79	Secondary malignant neoplasm of other sites
C80	Malignant neoplasm without specification of site
C81	Hodgkin lymphoma
C82	Follicular lymphoma
C83	Diffuse non-Hodgkin lymphoma
C84	Peripheral and cutaneous T-cell lymphomas
C85	Other and unspecified types of non-Hodgkin lymphoma
C90	Multiple myeloma
C91	Lymphoid leukemia
C97	Multiple independent primary neoplasms
D46	Myelodysplastic syndromes
D50	Iron deficiency anemia
D63	Anemia in chronic diseases
D64	Other anemias

ICD 10	Description
D80	Immunodeficiency with predominantly antibody defects
D81	Combined immunodeficiencies
D82	Immunodeficiency associated with other major defects
D83	Common variable immunodeficiency
D84	Other immunodeficiencies
D89	Other disorders involving the immune mechanism
E10	Type 1 diabetes mellitus
E11	Type 2 diabetes mellitus
E66	Obesity
E78	Disorders of lipoprotein metabolism
F01	Vascular dementia
F03	Unspecified dementia
F06	Mental disorders due to brain damage
F07	Personality and behavioral disorders due to brain disease
F09	Unspecified mental disorder due to known physiological condition
F10	Alcohol-related disorders
F11	Opioid-related disorders
F20	Schizophrenia
F25	Schizoaffective disorders
F31	Bipolar disorder
F32	Depressive episode
F33	Recurrent depressive disorder
F41	Anxiety disorders
F84	Pervasive developmental disorders (autism, etc.)
F88	Other disorders of psychological development
F99	Mental disorder, not otherwise specified
G20	Parkinson's disease
G30	Alzheimer's disease
G31	Other degenerative CNS diseases
G35	Multiple sclerosis
G89	Pain disorders related to chronic conditions
I10	Essential (primary) hypertension
I11	Hypertensive heart disease
I20	Angina pectoris
I21	Acute myocardial infarction
I25	Chronic ischemic heart disease
I42	Cardiomyopathy
I50	Heart failure
I63	Cerebral infarction (ischemic stroke)
I69	Sequelae of cerebrovascular disease
J43	Emphysema
J44	Chronic obstructive pulmonary disease (COPD)
J45	Asthma
J96	Respiratory failure
K50	Crohn's disease
K51	Ulcerative colitis
K70	Alcoholic liver disease
K74	Fibrosis and cirrhosis of liver
K76	Other liver diseases
K92	Other diseases of digestive system
L40	Psoriasis
L89	Pressure ulcers
M05	Rheumatoid arthritis with rheumatoid factor
M06	Other rheumatoid arthritis
M81	Osteoporosis without current pathological fracture
N18	Chronic kidney disease
N39	Urinary tract disorders
P07	Disorders related to prematurity
Q90	Down syndrome

ICD 10	Description
R06	Abnormalities of breathing
R26	Abnormalities of gait and mobility
R29	Other symptoms involving nervous and musculoskeletal systems
R41	Cognitive symptoms
R53	Malaise and fatigue
R54	Age-related physical debility
S06	Intracranial injury
S72	Fracture of femur (hip fracture)
Z00	General examination and routine child health check
Z43	Attention to artificial openings
Z51	Encounter for chemotherapy or dialysis
Z72	Problems related to lifestyle
Z73	Problems related to life-management difficulty
Z74	Problems related to care provider dependency
Z75	Problems related to medical facility access and care
Z76	Persons encountering health services in other circumstances
Z79	Long-term (current) drug therapy
Z91	Personal risk factors, including fall history
Z94	Transplanted organ and tissue status
Z95	Presence of cardiac and vascular implants and grafts
Z96	Presence of other functional implants
Z98	Other postprocedural states
Z99	Dependence on enabling machines and devices

Notes: This table lists the diagnostic groups from the RIPS database used as predictors in the model of care provision presented in Chapter III. The diagnostic codes follow the International Classification of Diseases, 10th Revision (ICD-10) for Colombia. Diagnoses are grouped using the first three characters of the ICD-10 codes.

TABLE C.7: Activities for Prostate Cancer

Code	Name	Code Type	Activity Type	Purpose
L01CD04	CABAZITAXEL	ATC	Medication	Treatment
L01XX46	OLAPARIB	ATC	Medication	Treatment
L02AE02	LEUPROLINA ACETATO	ATC	Medication	Treatment
L02BB04	ENZALUTAMIDA	ATC	Medication	Treatment
L02BB05	APALUTAMIDA	ATC	Medication	Treatment
L02BX03	ABIRATERONA	ATC	Medication	Treatment
601	PROCEDIMIENTOS DIAGNOSTICOS EN PROSTATA Y VESICULAS SEMINALE	CUPS	Procedure	Diagnosis
6011	BIOPSIA CERRADA (PERCUTANEA) (AGUJA) DE PROSTATA	CUPS	Procedure	Diagnosis
601101	BIOPSIA CERRADA DE PROSTATA POR ABORDAJE TRANSRECTAL	CUPS	Procedure	Diagnosis
601102	BIOPSIA CERRADA DE PROSTATA POR ABORDAJE PERINEAL	CUPS	Procedure	Diagnosis
601103	BIOPSIA CERRADA DE PROSTATA POR SATURACION ABORDAJE TRANSREC	CUPS	Procedure	Diagnosis
601104	BIOPSIA CERRADA DE PROSTATA POR SATURACION ABORDAJE PERINEAL	CUPS	Procedure	Diagnosis
6012	BIOPSIAS DE PROSTATA VIA ABIERTA	CUPS	Procedure	Diagnosis
601201	BIOPSIA DE PROSTATA VIA ABIERTA	CUPS	Procedure	Diagnosis
6013	BIOPSIA CERRADA (PERCUTANEA) (AGUJA) DE VESICULAS SEMINALES	CUPS	Procedure	Diagnosis
601301	BIOPSIA CERRADA (PERCUTANEA) (AGUJA) DE VESICULAS SEMINALES	CUPS	Procedure	Diagnosis
601311	BIOPSIA DE VESICULA SEMINAL POR LAPAROSCOPIA	CUPS	Procedure	Diagnosis
6014	BIOPSIA ABIERTA DE VESICULAS SEMINALES	CUPS	Procedure	Diagnosis
601401	BIOPSIA DE VESICULA SEMINAL VIA ABIERTA	CUPS	Procedure	Diagnosis
6015	BIOPSIA DE TEJIDO PERIPROSTATICO	CUPS	Procedure	Diagnosis
601501	BIOPSIA DE TEJIDO PERIPROSTATICO VIA ABIERTA	CUPS	Procedure	Diagnosis
601502	BIOPSIA DE TEJIDO PERIPROSTATICO VIA LAPAROSCOPICA	CUPS	Procedure	Diagnosis
6071	ASPIRACION (PERCUTANEA) CON (AGUJA) DE VESICULAS SEMINALES	CUPS	Procedure	Diagnosis
607100	ASPIRACION (PERCUTANEA) CON (AGUJA) DE VESICULAS SEMINALES S	CUPS	Procedure	Diagnosis
6091	ASPIRACION PERCUTANEA DE PROSTATA	CUPS	Procedure	Diagnosis
609100	ASPIRACION PERCUTANEA DE PROSTATA SOD	CUPS	Procedure	Diagnosis
879431	UROGRAFIA CON TOMOGRAFIA COMPUTADA	CUPS	Procedure	Diagnosis
890294	CONSULTA DE PRIMERA VEZ POR ESPECIALISTA EN UROLOGIA	CUPS	Procedure	Diagnosis
908435	PCA3 PARA CANCER DE PROSTATA	CUPS	Procedure	Diagnosis
922830	TERAPIA DE METASTASIS CON ESTRONCIO	CUPS	Procedure	Diagnosis
609	OTROS PROCEDIMIENTOS EN PROSTATA	CUPS	Procedure	Diagnosis and Treatment
890394	CONSULTA DE CONTROL O DE SEGUIMIENTO POR ESPECIALISTA EN URO	CUPS	Procedure	Diagnosis and Treatment
890494	INTERCONSULTA POR ESPECIALISTA EN UROLOGIA	CUPS	Procedure	Diagnosis and Treatment
60	PROCEDIMIENTOS EN PROSTATA Y VESICULAS SEMINALES	CUPS	Procedure	Treatment
600	INCISION EN PROSTATA	CUPS	Procedure	Treatment
6001	DRENAJE DE COLECCION PROSTATICA	CUPS	Procedure	Treatment
600110	DRENAJE DE COLECCION EN PROSTATA VIA ABIERTA	CUPS	Procedure	Treatment
600112	DRENAJE DE COLECCION EN PROSTATA VIA ENDOSCOPICA	CUPS	Procedure	Treatment
6002	PROSTATOLITOTOMIA	CUPS	Procedure	Treatment
600201	PROSTATOLITOTOMIA VIA ABIERTA	CUPS	Procedure	Treatment
600202	PROSTATOLITOTOMIA VIA PERCUTANEA	CUPS	Procedure	Treatment
600203	PROSTATOLITOTOMIA VIA ENDOSCOPICA	CUPS	Procedure	Treatment
602	PROSTATECTOMIAS TRANSURETRALES O ADENOMECTOMIAS	CUPS	Procedure	Treatment
6020	ADENOMECTOMIAS O PROSTATECTOMIAS TRANSURETRALES	CUPS	Procedure	Treatment
602001	RESECCCION O ENUCLEACION TRANSURETRAL DE ADENOMA DE PROSTATA	CUPS	Procedure	Treatment
602002	ADENOMECTOMIA O PROSTATECTOMIA TRANSVESICAL	CUPS	Procedure	Treatment
602003	ADENOMECTOMIA O PROSTATECTOMIA RETROPUBICA O TRANSVESICOCAPS	CUPS	Procedure	Treatment
602004	ADENOMECTOMIA O PROSTATECTOMIA POR LAPAROSCOPIA	CUPS	Procedure	Treatment
602005	ADENOMECTOMIA POR ABLACION DE PROSTATA	CUPS	Procedure	Treatment
605	PROSTATECTOMIA RADICAL	CUPS	Procedure	Treatment
6051	PROSTATECTOMIA RADICAL [PROSTATOVESICULECTOMIA]	CUPS	Procedure	Treatment
605101	RESECCION DE PROSTATA [PROSTATECTOMIA] RADICAL [PROSTATOVESI	CUPS	Procedure	Treatment
605111	PROSTATECTOMIA RADICAL POR LAPAROSCOPIA	CUPS	Procedure	Treatment
605112	PROSTATECTOMIA RADICAL POR ABLACION	CUPS	Procedure	Treatment
607	PROCEDIMIENTOS EN VESICULAS SEMINALES	CUPS	Procedure	Treatment
6073	ESCISION DE VESICULAS SEMINALES	CUPS	Procedure	Treatment

Code	Name	Code Type	Activity Type	Purpose
607301	VESICULECTOMIA O ESPERMATOCISTECTOMIA	CUPS	Procedure	Treatment
607311	VESICULECTOMIA O ESPERMATOCISTECTOMIA UNILATERAL POR LAPAROS	CUPS	Procedure	Treatment
607312	VESICULECTOMIA O ESPERMATOCISTECTOMIA BILATERAL POR LAPAROSC	CUPS	Procedure	Treatment
6082	ESCISION DE TEJIDO PERIPROSTATICO	CUPS	Procedure	Treatment
608201	ESCISION DE LESION DE TEJIDO PERIPROSTATICO	CUPS	Procedure	Treatment

TABLE C.8: Activities for Leukemia

Code	Name	Code Type	Activity Type	Purpose
H02AB07	PREDNISONA	ATC	Medication	Treatment
L01AA09	BENDAMUSTINE	ATC	Medication	Treatment
L01BA01	METROTEXATE	ATC	Medication	Treatment
L01BB02	MERCAPTOPURINA	ATC	Medication	Treatment
L01BB05	FLUDARABINA	ATC	Medication	Treatment
L01BC01	CITARABINA	ATC	Medication	Treatment
L01BC07	AZACITIDINA	ATC	Medication	Treatment
L01CB01	ETOPOSIDO	ATC	Medication	Treatment
L01DB02	DAUNORRUBICINA	ATC	Medication	Treatment
L01DB06	IDARRUBICINA	ATC	Medication	Treatment
L01XC02	RITUXIMAB	ATC	Medication	Treatment
L01XE01	IMATINIB	ATC	Medication	Treatment
L01XE06	DASATINIB	ATC	Medication	Treatment
L01XE14	BOSUTINIB	ATC	Medication	Treatment
L01XE24	PONATINIB	ATC	Medication	Treatment
L01XE27	IBRUTINIB	ATC	Medication	Treatment
L01XE39	MIDOSTAURINA	ATC	Medication	Treatment
L01XX05	HIDROXICARBAMIDA	ATC	Medication	Treatment
L01XX52	VENETOCLAX	ATC	Medication	Treatment
L04AX03	METOTREXATE	ATC	Medication	Treatment
S01AA11	GENTAMICINA	ATC	Medication	Treatment
4010	BIOPSIA DE GANGLIO LINFATICO CENTINELA	CUPS	Procedure	Diagnosis
401001	BIOPSIA DE GANGLIO LINFATICO CENTINELA CON TINCION	CUPS	Procedure	Diagnosis
401002	BIOPSIA DE GANGLIO LINFATICO CENTINELA CON RADIOMARCACION	CUPS	Procedure	Diagnosis
4011	BIOPSIA DE ESTRUCTURA LINFATICA	CUPS	Procedure	Diagnosis
401101	BIOPSIA DE GANGLIO LINFATICO SUPERFICIAL	CUPS	Procedure	Diagnosis
401102	BIOPSIA DE GANGLIO LINFATICO PROFUNDO	CUPS	Procedure	Diagnosis
401201	BUSQUEDA DE LESION OCULTA RADIOGUIADA	CUPS	Procedure	Diagnosis
4131	BIOPSIA DE MEDULA OSEA	CUPS	Procedure	Diagnosis
413101	BIOPSIA POR ASPIRACION DE MEDULA OSEA	CUPS	Procedure	Diagnosis
4132	BIOPSIAS DE BAZO	CUPS	Procedure	Diagnosis
413201	BIOPSIA DE BAZO VIA PERCUTANEA	CUPS	Procedure	Diagnosis
413202	BIOPSIA DE BAZO VIA ABIERTA	CUPS	Procedure	Diagnosis
413204	BIOPSIA DE BAZO VIA LAPAROSCOPICA	CUPS	Procedure	Diagnosis
4191	ASPIRACION DE MEDULA OSEA DE DONANTE	CUPS	Procedure	Diagnosis
419100	ASPIRACION DE MEDULA OSEA DE DONANTE SOD	CUPS	Procedure	Diagnosis
902206	EXTENDIDO DE SANGRE PERIFERICA ESTUDIO DE MORFOLOGIA	CUPS	Procedure	Diagnosis
902210	HEMOGRAMA IV (HEMOGLOBINA HEMATOCRITO RECUENTO DE ERITROCITO	CUPS	Procedure	Diagnosis
902216	LEUCOGRAMA (RECUENTO TOTAL Y DIFERENCIAL)	CUPS	Procedure	Diagnosis
902220	RECUENTO DE PLAQUETAS AUTOMATIZADO	CUPS	Procedure	Diagnosis
906501	TIPIFICACION ANTIGENO LEUCOCITARIO HUMANO CLASE I (A B C)	CUPS	Procedure	Diagnosis
906502	TIPIFICACION ANTIGENO LEUCOCITARIO HUMANO CLASE I Y II (A B	CUPS	Procedure	Diagnosis
906503	TIPIFICACION ANTIGENO LEUCOCITARIO HUMANO LOCUS B	CUPS	Procedure	Diagnosis
906504	TIPIFICACION ANTIGENO LEUCOCITARIO HUMANO LOCUS DR	CUPS	Procedure	Diagnosis
906506	TIPIFICACION ANTIGENO LEUCOCITARIO HUMANO CLASE II (DR DQ DP	CUPS	Procedure	Diagnosis
906509	ANTICUERPOS CITOTOXICOS ANTI HLA	CUPS	Procedure	Diagnosis
906512	TIPIFICACION DE ANTIGENO LEUCOCITARIO HUMANO LOCUS A	CUPS	Procedure	Diagnosis
906520	ANTICUERPOS DONANTE ESPECIFICO (DONANTE - RECEPTOR TRASPLANT	CUPS	Procedure	Diagnosis
906521	ANTICUERPOS ANTI HLA CLASE I (P.R.A) CUALITATIVO	CUPS	Procedure	Diagnosis
906522	ANTICUERPOS ANTI HLA CLASE II (P.R.A) CUALITATIVO	CUPS	Procedure	Diagnosis
906523	ANTICUERPOS ANTI HLA CLASE I (P.R.A) CUANTITATIVO	CUPS	Procedure	Diagnosis
906524	ANTICUERPOS ANTI HLA CLASE II (P.R.A) CUANTITATIVO	CUPS	Procedure	Diagnosis
906525	ANTICUERPOS ANTI HLA ANTIGENO AISLADO CLASE I	CUPS	Procedure	Diagnosis
906526	ANTICUERPOS ANTI HLA ANTIGENO AISLADO CLASE II	CUPS	Procedure	Diagnosis
906527	PRUEBA DE QUIMERISMO	CUPS	Procedure	Diagnosis
906701	CULTIVO MIXTO DE LINFOCITOS	CUPS	Procedure	Diagnosis

Code	Name	Code Type	Activity Type	Purpose
906702	LEUCOCITOS CD14 MONOCITOS GRANULOCITOS SEMIAUTOMATIZADO O AU	CUPS	Procedure	Diagnosis
906703	LEUCOCITOS CD14 MONOCITOS GRANULOCITOS POR INMUNOHISTOQUIMIC	CUPS	Procedure	Diagnosis
906704	LEUCOCITOS CD33 MONOCITOS GRANULOCITOS SEMIAUTOMATIZADO O AU	CUPS	Procedure	Diagnosis
906705	LEUCOCITOS CD33 MONOCITOS GRANULOCITOS POR INMUNOHISTOQUIMIC	CUPS	Procedure	Diagnosis
906706	LEUCOCITOS CD34 CELULAS PROGENITORAS SEMIAUTOMATIZADO O AUTO	CUPS	Procedure	Diagnosis
906707	LEUCOCITOS CD34 CELULAS PROGENITORAS POR INMUNOHISTOQUIMICA	CUPS	Procedure	Diagnosis
906708	LEUCOCITOS CD45 LEUCOCITOS TOTALES SEMIAUTOMATIZADO O AUTOMA	CUPS	Procedure	Diagnosis
906709	LEUCOCITOS CD45 LEUCOCITOS TOTALES POR INMUNOHISTOQUIMICA	CUPS	Procedure	Diagnosis
906710	LEUCOCITOS MPO SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906710	LINFOCITOS B (CD19 Y CD20) SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906711	LINFOCITOS B (CD19 Y CD20) SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906711	LINFOCITOS T CD3 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906712	LINFOCITOS T CD3 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906712	LINFOCITOS T CD3 POR INMUNOFLUORESCENCIA	CUPS	Procedure	Diagnosis
906713	LINFOCITOS T CD4 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906714	LINFOCITOS T CD4 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906716	LINFOCITOS CD5 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906718	LINFOCITOS CD7 LINFOCITOS T Y NK SEMIAUTOMATIZADO O AUTOMATI	CUPS	Procedure	Diagnosis
906719	LINFOCITOS T CD8 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906720	LINFOCITOS T CD8 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906721	LINFOCITOS CD10 LINFOCITOS PRE-B [CALLA] SEMIAUTOMATIZADO O	CUPS	Procedure	Diagnosis
906722	LINFOCITOS CD10 LINFOCITOS PRE-B [CALLA] SEMIAUTOMATIZADO O	CUPS	Procedure	Diagnosis
906723	LINFOCITOS CD11 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906724	LINFOCITOS CD11 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906725	LINFOCITOS CD11 POR INMUNOHISTOQUIMICA	CUPS	Procedure	Diagnosis
906725	LINFOCITOS CD13 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906726	LINFOCITOS CD13 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906727	LINFOCITOS CD13 POR INMUNOHISTOQUIMICA	CUPS	Procedure	Diagnosis
906727	LINFOCITOS CD15 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906728	LINFOCITOS CD15 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906729	LINFOCITOS CD15 POR INMUNOHISTOQUIMICA	CUPS	Procedure	Diagnosis
906729	LINFOCITOS CD16 LINFOCITOS NK SEMIAUTOMATIZADO O AUTOMATIZAD	CUPS	Procedure	Diagnosis
906730	LINFOCITOS CD16 LINFOCITOS NK SEMIAUTOMATIZADO O AUTOMATIZAD	CUPS	Procedure	Diagnosis
906731	LINFOCITOS CD22 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906732	LINFOCITOS CD22 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906733	LINFOCITOS CD23 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906734	LINFOCITOS CD23 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906735	LINFOCITOS CD38 LINFOCITOS T ACTIVADOS Y B LINFOCITOS NK SEM	CUPS	Procedure	Diagnosis
906736	LINFOCITOS CD38 LINFOCITOS T ACTIVADOS Y B LINFOCITOS NK SEM	CUPS	Procedure	Diagnosis
906737	LINFOCITOS CD56 LINFOCITOS NK SEMIAUTOMATIZADO O AUTOMATIZAD	CUPS	Procedure	Diagnosis
906738	LINFOCITOS CD56 LINFOCITOS NK SEMIAUTOMATIZADO O AUTOMATIZAD	CUPS	Procedure	Diagnosis
906739	LINFOCITOS CD79A SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906740	LINFOCITOS CD79A SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906743	LINFOCITOS T CUANTIFICACION CD3 CD4 CD8 SEMIAUTOMATIZADO O A	CUPS	Procedure	Diagnosis
906744	LINFOCITOS T CUANTIFICACION CD3 CD4 CD8 SEMIAUTOMATIZADO O A	CUPS	Procedure	Diagnosis
906746	MONOCITOS CD45 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906747	MONOCITOS CD45 POR INMUNOHISTOQUIMICA	CUPS	Procedure	Diagnosis
906748	MONOCITOS CD64 SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906749	MONOCITOS CD64 POR INMUNOHISTOQUIMICA	CUPS	Procedure	Diagnosis
906762	LINFOCITOS T (CD3 CD4 CD8 RELACION CD4/CD8) Y LINFOCITOS B (	CUPS	Procedure	Diagnosis
906763	LINFOCITOS T (CD3 CD4 CD8 RELACION CD4/CD8) Y LINFOCITOS B (	CUPS	Procedure	Diagnosis
906763	LINFOCITOS T SUBPOBLACIONES PRINCIPALES: CD45 CD3 CD4 CD8 SE	CUPS	Procedure	Diagnosis
906764	LINFOCITOS T SUBPOBLACIONES PRINCIPALES: CD45 CD3 CD4 CD8 SE	CUPS	Procedure	Diagnosis
906765	MARCADOR TCR ALFA BETA (TCRAB) SEMIAUTOMATIZADO O AUTOMATIZA	CUPS	Procedure	Diagnosis
906766	MONOCITOS CD14	CUPS	Procedure	Diagnosis
906766	SUBPOBLACIONES DE LINFOCITOS T B NK Y MONOCITOS EN LEUCOCITO	CUPS	Procedure	Diagnosis
906768	SUBPOBLACIONES DE LINFOCITOS T B NK Y MONOCITOS EN LEUCOCITO	CUPS	Procedure	Diagnosis
906769	SUBPOBLACIONES EXTENDIDAS DE LINFOCITOS B (VIRGENES Y DE MEM	CUPS	Procedure	Diagnosis
906770	SUBPOBLACIONES EXTENDIDAS DE LINFOCITOS T (AYUDADORES Y CITO	CUPS	Procedure	Diagnosis

Code	Name	Code Type	Activity Type	Purpose
906776	LINFOPROLIFERACION A ANTI-CD3+ ANTI-CD28	CUPS	Procedure	Diagnosis
906777	LINFOPROLIFERACION A MITOGENO	CUPS	Procedure	Diagnosis
906778	APOPTOSIS DE LINFOCITOS T	CUPS	Procedure	Diagnosis
906784	LINFOCITOS T REGULADORES	CUPS	Procedure	Diagnosis
906801	BETA 2 GLICOPROTEINA I SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906805	COMPLEJOS INMUNES CIRCULANTES SEMIAUTOMATIZADO O AUTOMATIZAD	CUPS	Procedure	Diagnosis
906806	COMPLEMENTO C1Q SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
906808	ELECTROFORESIS DE HEMOGLOBINA SEMIAUTOMATIZADO	CUPS	Procedure	Diagnosis
906809	ELECTROFORESIS DE HEMOGLOBINA AUTOMATIZADO	CUPS	Procedure	Diagnosis
906822	HISTAMINA SEMIAUTOMATIZADO O AUTOMATIZADO	CUPS	Procedure	Diagnosis
9084	BCL-2 TRANSLOCACION (14;18)	CUPS	Procedure	Diagnosis
908402	BCL-2 TRANSLOCACION (14;18)	CUPS	Procedure	Diagnosis
908402	BCR/ABL TRANSLOCACION DE GENES CUALITATIVO	CUPS	Procedure	Diagnosis
908403	BCR/ABL TRANSLOCACION DE GENES CUALITATIVO	CUPS	Procedure	Diagnosis
908403	CARIOTIPO CON BANDEO G	CUPS	Procedure	Diagnosis
908404	CARIOTIPO CON BANDEO G	CUPS	Procedure	Diagnosis
908404	CARIOTIPO CON BANDEO Q	CUPS	Procedure	Diagnosis
908405	CARIOTIPO CON BANDEO Q	CUPS	Procedure	Diagnosis
908405	CARIOTIPO CON BANDEO C	CUPS	Procedure	Diagnosis
908406	CARIOTIPO CON BANDEO C	CUPS	Procedure	Diagnosis
908406	CARIOTIPO CON BANDEO RT	CUPS	Procedure	Diagnosis
908407	CARIOTIPO CON BANDEO RT	CUPS	Procedure	Diagnosis
908407	CARIOTIPO DE INTERCAMBIO DE CROMATIDES HERMANAS [SCE]	CUPS	Procedure	Diagnosis
908408	CARIOTIPO DE INTERCAMBIO DE CROMATIDES HERMANAS [SCE]	CUPS	Procedure	Diagnosis
908408	CARIOTIPO PARA CROMOSOMA X FRAGIL	CUPS	Procedure	Diagnosis
908409	CARIOTIPO PARA CROMOSOMA X FRAGIL	CUPS	Procedure	Diagnosis
908409	CARIOTIPO PARA CROMOSOMA FILADELFIA	CUPS	Procedure	Diagnosis
908410	CARIOTIPO PARA CROMOSOMA FILADELFIA	CUPS	Procedure	Diagnosis
908410	CARIOTIPO PARA ESTADOS LEUCEMICOS	CUPS	Procedure	Diagnosis
908411	CARIOTIPO PARA ESTADOS LEUCEMICOS	CUPS	Procedure	Diagnosis
908411	ESTUDIO MOLECULAR DE ENFERMEDADES	CUPS	Procedure	Diagnosis
908412	ERBB2 [HER-2/NEU] (ONCOGEN) HIBRIDACION ”IN SITU” CON FLUORE	CUPS	Procedure	Diagnosis
908412	ESTUDIO MOLECULAR DE ENFERMEDADES	CUPS	Procedure	Diagnosis
908414	REORGANIZACION DE GENE BCR/ABL	CUPS	Procedure	Diagnosis
908415	REORGANIZACION DE GENE BCR/ABL	CUPS	Procedure	Diagnosis
908416	BCR/ABL TRANSLOCACION DE GENES CUANTITATIVO	CUPS	Procedure	Diagnosis
908417	BCR/ABL TRANSLOCACION DE GENES CUANTITATIVO	CUPS	Procedure	Diagnosis
908417	ESTUDIOS GENETICOS DE CROMOSOMAS (ESPECIFICOS)	CUPS	Procedure	Diagnosis
908418	ESTUDIOS GENETICOS DE ADN MITOCONDRIAL (ESPECIFICO)	CUPS	Procedure	Diagnosis
908418	ESTUDIOS GENETICOS DE CROMOSOMAS (ESPECIFICOS)	CUPS	Procedure	Diagnosis
908419	ESTUDIOS MOLECULARES DE GENES (ESPECIFICOS)	CUPS	Procedure	Diagnosis
908420	ESTUDIOS MOLECULARES DE GENES (ESPECIFICOS)	CUPS	Procedure	Diagnosis
908420	ESTUDIO MOLECULAR DE REARREGLOS (ESPECIFICOS)	CUPS	Procedure	Diagnosis
908421	ESTUDIO MOLECULAR DE REARREGLOS (ESPECIFICOS)	CUPS	Procedure	Diagnosis
908423	ESTUDIO MOLECULAR DE MUTACIONES (ESPECIFICAS)	CUPS	Procedure	Diagnosis
908425	ESTUDIOS GENETICOS DE LOS CROMOSOMAS 14 23 Y 1	CUPS	Procedure	Diagnosis
908429	ABL MUTACION DE LA REGION TIROSINA KINASA	CUPS	Procedure	Diagnosis
908430	ABL MUTACION DE LA REGION TIROSINA KINASA	CUPS	Procedure	Diagnosis
908437	EXTRACCION DE ACIDOS NUCLEICOS	CUPS	Procedure	Diagnosis
908438	EXTRACCION DE ACIDOS NUCLEICOS	CUPS	Procedure	Diagnosis
908439	CARIOTIPO CON FRAGILIDAD CROMOSOMICA	CUPS	Procedure	Diagnosis
920502	GAMAGRAFIA DE MEDULA OSEA	CUPS	Procedure	Diagnosis
890251	CONSULTA DE PRIMERA VEZ POR ESPECIALISTA EN HEMATOLOGIA	CUPS	Procedure	Diagnosis and Treatment
890278	CONSULTA DE PRIMERA VEZ POR ESPECIALISTA EN ONCOLOGIA	CUPS	Procedure	Diagnosis and Treatment
890351	CONSULTA DE CONTROL O DE SEGUIMIENTO POR ESPECIALISTA EN HEM	CUPS	Procedure	Diagnosis and Treatment
890378	CONSULTA DE CONTROL O DE SEGUIMIENTO POR ESPECIALISTA EN ONC	CUPS	Procedure	Diagnosis and Treatment
890451	INTERCONSULTA POR ESPECIALISTA EN HEMATOLOGIA	CUPS	Procedure	Diagnosis and Treatment
890478	INTERCONSULTA POR ESPECIALISTA EN ONCOLOGIA	CUPS	Procedure	Diagnosis and Treatment
902210	HEMOGRAMA IV	CUPS	Procedure	Diagnosis and Treatment

Code	Name	Code Type	Activity Type	Purpose
4021	ESCISION O ABLACION DEL GANGLIO LINFATICO CERVICAL PROFUNDO	CUPS	Procedure	Treatment
402101	ESCISION DE GANGLIO LINFATICO CERVICAL PROFUNDO	CUPS	Procedure	Treatment
402102	ABLACION DE GANGLIO LINFATICO CERVICAL PROFUNDO VIA PERCUTAN	CUPS	Procedure	Treatment
4023	ESCISION DE GANGLIO LINFATICO AXILAR	CUPS	Procedure	Treatment
4024	ESCISION DE GANGLIO LINFATICO INGUINAL	CUPS	Procedure	Treatment
402400	ESCISION DE GANGLIO LINFATICO INGUINAL SOD	CUPS	Procedure	Treatment
4030	ESCISION DE GANGLIO LINFATICO REGIONAL EXTENDIDA AL AREA DE	CUPS	Procedure	Treatment
403000	ESCISION DE GANGLIO LINFATICO REGIONAL EXTENDIDA AL AREA DE	CUPS	Procedure	Treatment
4051	VACIAMIENTO RADICAL LINFATICO AXILAR	CUPS	Procedure	Treatment
405101	VACIAMIENTO RADICAL LINFATICO AXILAR VIA ABIERTA	CUPS	Procedure	Treatment
405102	VACIAMIENTO RADICAL LINFATICO AXILAR VIA ENDOSCOPICA	CUPS	Procedure	Treatment
4052	VACIAMIENTO RADICAL LINFATICO (LINFADENECTOMIA) TORACICO O M	CUPS	Procedure	Treatment
405201	VACIAMIENTO RADICAL LINFATICO (LINFADENECTOMIA) DE MEDIASTIN	CUPS	Procedure	Treatment
405202	VACIAMIENTO RADICAL LINFATICO (LINFADENECTOMIA) DE MEDIASTIN	CUPS	Procedure	Treatment
405203	VACIAMIENTO RADICAL LINFATICO (LINFADENECTOMIA) DE MEDIASTIN	CUPS	Procedure	Treatment
4053	VACIAMIENTO RADICAL LINFATICO INGUINO ILIACO	CUPS	Procedure	Treatment
405304	LINFADENECTOMIA RADICAL INGUINOFEMORAL, UNILATERAL VIA ABIER	CUPS	Procedure	Treatment
405305	LINFADENECTOMIA RADICAL INGUINOFEMORAL, UNILATERAL VIA LAPAR	CUPS	Procedure	Treatment
405306	LINFADENECTOMIA RADICAL INGUINOILIACO BILATERAL VIA ABIERTA	CUPS	Procedure	Treatment
405307	LINFADENECTOMIA RADICAL INGUINOILIACO BILATERAL VIA LAPAROSC	CUPS	Procedure	Treatment
4054	VACIAMIENTO RADICAL LINFATICO ABDOMINO PELVICO	CUPS	Procedure	Treatment
405404	LINFADENECTOMIA RADICAL ABDOMINAL VIA ABIERTA	CUPS	Procedure	Treatment
405405	LINFADENECTOMIA RADICAL ABDOMINAL VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
405406	LINFADENECTOMIA RADICAL PELVICA VIA ABIERTA	CUPS	Procedure	Treatment
405407	LINFADENECTOMIA RADICAL PELVICA VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
405408	LINFADENECTOMIA RADICAL EXTRAPERITONEAL VIA ABIERTA	CUPS	Procedure	Treatment
405409	LINFADENECTOMIA RADICAL EXTRAPERITONEAL VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
405411	LINFADENECTOMIA RADICAL ABDOMINO INGUINAL VIA ABIERTA	CUPS	Procedure	Treatment
405412	LINFADENECTOMIA RADICAL ABDOMINO INGUINAL VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
4055	ESCISION RADICAL DE GANGLIOS LINFATICOS RETROPERITONEALES	CUPS	Procedure	Treatment
405502	RESECCION RADICAL DE GANGLIOS LINFATICOS RETROPERITONEALES V	CUPS	Procedure	Treatment
405503	LINFADENECTOMIA RETROPERITONEAL VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
4056	VACIAMIENTO RADICAL LINFATICO DE MIEMBROS SUPERIORES O INFER	CUPS	Procedure	Treatment
405601	VACIAMIENTO RADICAL EPITROCLEAR VIA ABIERTA	CUPS	Procedure	Treatment
405602	VACIAMIENTO RADICAL POPLITEO VIA ABIERTA	CUPS	Procedure	Treatment
4074	TRASPLANTE DE LINFATICOS AUTOGENOS	CUPS	Procedure	Treatment
407400	TRASPLANTE DE LINFATICOS AUTOGENOS SOD	CUPS	Procedure	Treatment
410	TRASPLANTE DE MEDULA OSEA O DE CELULAS PROGENITORAS	CUPS	Procedure	Treatment
4105	TRASPLANTE AUTOLOGO	CUPS	Procedure	Treatment
410501	TRASPLANTE AUTOLOGO DE MEDULA OSEA	CUPS	Procedure	Treatment
410502	TRASPLANTE AUTOLOGO DE CELULAS MADRES HEMATOPOYETICAS DE SAN	CUPS	Procedure	Treatment
4106	TRASPLANTE ALOGENICO	CUPS	Procedure	Treatment
410601	TRASPLANTE ALOGENICO DE MEDULA OSEA	CUPS	Procedure	Treatment
410602	TRASPLANTE ALOGENICO DE CELULAS MADRES HEMATOPOYETICAS DE SA	CUPS	Procedure	Treatment
410603	TRASPLANTE ALOGENICO DE CELULAS MADRES HEMATOPOYETICAS DE CO	CUPS	Procedure	Treatment
4143	ESPLENECTOMIA PARCIAL	CUPS	Procedure	Treatment
414301	ESPLENECTOMIA PARCIAL VIA ABIERTA	CUPS	Procedure	Treatment
414302	ESPLENECTOMIA PARCIAL VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
4151	ESPLENECTOMIA TOTAL	CUPS	Procedure	Treatment
415102	ESPLENECTOMIA TOTAL VIA ABIERTA	CUPS	Procedure	Treatment
415103	ESPLENECTOMIA TOTAL VIA LAPAROSCOPICA	CUPS	Procedure	Treatment
4192	INYECCION DE MEDULA OSEA	CUPS	Procedure	Treatment
419200	INYECCION O INFUSION DE MEDULA OSEA SOD	CUPS	Procedure	Treatment
4194	TRASPLANTE DE BAZO	CUPS	Procedure	Treatment
419400	TRASPLANTE DE BAZO SOD	CUPS	Procedure	Treatment
920505	GAMAGRAFIA CON LEUCOCITOS MARCADOS	CUPS	Procedure	Treatment
921301	GAMAGRAFIA DE VIABILIDAD TUMORAL CON MIBI, TETROFOSMIN, TALI	CUPS	Procedure	Treatment
922447	IRRADIACION CORPORAL TOTAL	CUPS	Procedure	Treatment
922614	BRAQUITERAPIA METABOLICA	CUPS	Procedure	Treatment

Code	Name	Code Type	Activity Type	Purpose
992509	MONOTERAPIA ANTINEOPLASICA DE BAJA TOXICIDAD	CUPS	Medication	Treatment

TABLE C.9: Urban Markets

Municipio	City or Metropolitan Area
05001 - MEDELLÍN	05001 - MEDELLÍN
05079 - BARBOSA	05001 - MEDELLÍN
05088 - BELLO	05001 - MEDELLÍN
05129 - CALDAS	05001 - MEDELLÍN
05212 - COPACABANA	05001 - MEDELLÍN
05266 - ENVIGADO	05001 - MEDELLÍN
05308 - GIRARDOTA	05001 - MEDELLÍN
05360 - ITAGÜÍ	05001 - MEDELLÍN
05380 - LA ESTRELLA	05001 - MEDELLÍN
05631 - SABANETA	05001 - MEDELLÍN
08001 - BARRANQUILLA	08001 - BARRANQUILLA
08758 - SOLEDAD	08001 - BARRANQUILLA
11001 - BOGOTÁ, D.C.	11001 - BOGOTÁ, D.C.
13001 - CARTAGENA DE INDIAS	13001 - CARTAGENA DE INDIAS
15001 - TUNJA	15001 - TUNJA
17001 - MANIZALES	17001 - MANIZALES
17873 - VILLAMARÍA	17001 - MANIZALES
18001 - FLORENCIA	18001 - FLORENCIA
19001 - POPAYÁN	19001 - POPAYÁN
20001 - VALLEDUPAR	20001 - VALLEDUPAR
23001 - MONTERÍA	23001 - MONTERÍA
27001 - QUIBDÓ	27001 - QUIBDÓ
41001 - NEIVA	41001 - NEIVA
44001 - RIOHACHA	44001 - RIOHACHA
47001 - SANTA MARTA	47001 - SANTA MARTA
50001 - VILLAVICENCIO	50001 - VILLAVICENCIO
52001 - PASTO	52001 - PASTO
54001 - SAN JOSÉ DE CÚCUTA	54001 - SAN JOSÉ DE CÚCUTA
54261 - EL ZULIA	54001 - SAN JOSÉ DE CÚCUTA
54405 - LOS PATIOS	54001 - SAN JOSÉ DE CÚCUTA
54553 - PUERTO SANTANDER	54001 - SAN JOSÉ DE CÚCUTA
54874 - VILLA DEL ROSARIO	54001 - SAN JOSÉ DE CÚCUTA
63001 - ARMENIA	63001 - ARMENIA
66001 - PEREIRA	66001 - PEREIRA
66170 - DOSQUEBRADAS	66001 - PEREIRA
66400 - LA VIRGINIA	66001 - PEREIRA
68001 - BUCARAMANGA	68001 - BUCARAMANGA
68276 - FLORIDABLANCA	68001 - BUCARAMANGA
68307 - GIRÓN	68001 - BUCARAMANGA
68547 - PIEDECUESTA	68001 - BUCARAMANGA
70001 - SINCELEJO	70001 - SINCELEJO
73001 - IBAGUÉ	73001 - IBAGUÉ
76001 - CALI	76001 - CALI
76892 - YUMBO	76001 - CALI

Notes: This table lists the 44 municipios included in the urban markets sample. For each municipio in the left column, we also list the corresponding city or metropolitan area it belongs to in the right column. The codes preceding the municipio and city names are the ones defined by DANE in DIVIPOLA.