Asymmetric Information with multiple risks: the case of the Chilean Private Health Insurance Market

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Abstract

Abstract: In the health insurance literature, the insured risk is assumed to be uni-dimensional, typically measured as medical expenditures. The uni-dimensional assumption, however, is not without its shortcomings as it fails to recognize the explicit distinction made in most health insurance contracts between at least two risks: the risk of needing inpatient care and the risk of needing outpatient care. The two risks are covered by different degrees of generosity as in, for example, Medicare parts A and B. In this paper we show, both theoretically and empirically, that the multi-dimensionality in health risk and coverage can affect the results of the "positive correlation test" (PCT) of Asymmetric Information in either direction. The recent literature argues that failure to find evidence of adverse selection may be due to the multidimensional heterogeneity of individuals' (unobserved) characteristics, such as risk aversion, cognitive ability, or misperceptions on risk. We instead analyze a different source of multidimensionality, namely, the presence of two sources of risk. Notice that, contrary to the ones proposed in the literature, these dimensions are not only directly relevant to the insuree but also to the insurer. This extension is important for at least four reasons: a) individuals may have different risk profiles along these dimensions, implying that risks may not be ranked; b) insurance contracts typically have different degrees of coverage along these two dimensions; c) there may be asymmetric information in one risk dimension and not in the other; (d) and most importantly, adverse selection may be underestimated if one takes aggregate measures of risk and coverage in order to reduce the analysis to one of single dimensional screening. We extend the competitive separating equilibrium of Rothschild and Stiglitz (1976) to allow for two sources of risk and two-dimensional coverage contracts and use its theoretical predictions to construct an alternative test of asymmetric information, which we apply to the universe of the privately insured in the Chilean health insurance market for the year 2007.

Keywords*: Testing for asymmetric information, adverse and advantageous selection, insurance markets„ Positive correlation test, Chilean health insurance, private health insurance, competitive multidimensional screening, Rothschild and Stiglitz.*

JEL Classification: *I13, L13, D82*

1 Introduction

In the health insurance literature, the insured risk is assumed to be uni-dimensional, typically measured as medical expenditures. This assumption is both practical and tractable. It is practical because medical expenditure data can be easily retrieved from surveys or insurance claims datasets and it is tractable because it facilitates theoretical scrutiny as well as empirical tests of insurance models such as the well-known "positive correlation test" (PCT) of asymmetric information (AI) developed by Chiappori and Salanié (2000).[1] The uni-dimensional assumption, however, is not without its shortcomings as it fails to recognize the explicit distinction made in most health insurance contracts between at least two risks: the risk of needing inpatient care and the risk of needing outpatient care. The two risks are typically covered to different degrees of generosity as in, for example, Medicare parts A and B. One obvious reason for having such differences in coverage is the existence of moral hazard, which has been shown to affect outpatient services significantly more than inpatient services;[2] but there may be other reasons. For example, it is conceivable that clients' private information regarding one of the risk sources is relatively more precise than the other, leading to a larger degree of adverse selection in the former dimension. Symmetrically, insurers may be able to condition on observables that are more informative of the actual risks in one dimension than in the other.

The weight of the empirical evidence favors the existence of adverse selection in health insurance markets (e.g. see the review in Cutler and Zechauser, 2000). Some recent papers, however, find a negative correlation between risk and coverage in some of these markets, a phenomenon that has been commonly referred to as "advantageous selection" in allusion to the higher insurance coverage amongst the lower risk individuals;[3] Inspired in part by the theoretical literature, this failure of the

[1] Rothschild and Stiglitz (1976) show that when individuals have privileged information about their risk of requiring medical services (asymmetric information), higher risk individuals buy insurance contracts with higher coverage in equilibrium. The PCT relies on the existence of such a positive correlation between (unobserved) risk and insurance coverage should the market exhibit AI. However, since higher coverage fosters higher usage, both adverse selection and moral hazard result in a positive correlation between risk and coverage. In the light of this concurrence, the findings of non-positive correlations in certain insurance markets were surprising (see the review in Cohen and Siegelman, NBER WP 2009). De Meza and Webb (2016) criticize the PCT as a test of asymmetric information since the absence of correlation is not identified with symmetric information.

[2] Finkelstein et al. (2012) is one exception where insurance was found to increase the usage of inpatient services. Considering the low social economic status of the population in their sample, however, it is possible that the increase in inpatient usage may at least in part reflect undertreatment or lack of access to inpatient services in the absence of insurance.

[3] The original term was *propitious selection* (Hemenway et al. 1990).

PCT is attributed to the multi-dimensionality of AI in the form of unobserved clients' characteristics, which affect positively the decision to buy (extra) coverage but tend to decrease the insured risk. A likely candidate for an extra dimension leading to advantageous selection is risk aversion but the latest evidence shows that it plays a minor role in health insurance markets (Fang et al. 2008, Einav et al. 2013, Handel 2013).

In this paper we show, both theoretically and empirically, that the multi-dimensionality in health risk and coverage can affect the results of the PCT. We start by extending the competitive separating equilibrium of Rothschild and Stiglitz (1976) to allow for two sources of risk and two-dimensional coverage contracts, reflecting the inpatient and outpatient dichotomy. We use the theoretical predictions to check for adverse/advantageous selection empirically in a way that exploits this multidimensionality; from simple regressions, to non-parametric methods, to a trivial extension of the PCT whereby each dimension of risk and coverage are treated separately. We use a unique dataset, provided to us by the Superintendencia de Salud del Gobierno de Chile, which consists of individual-level discharge data for the universe of the privately insured during the year 2007. Our model and data concerns exclusively with the private health insurance market, which covered around 2.7 million people or 16.6% of the population during 2007. We are, therefore, silent regarding a potential selection between the public and private parts of the health insurance market.

Our theoretical model assumes that the insurance market is perfectly competitive and, for simplicity, that the two sources of risk (inpatient care and outpatient care) are independent. As we will see, the Chilean data does not contradict the first assumption. The second assumption, while strong, allows us to restrict attention to four types of individuals: i) high risk in both sources, or type HH, ii) high risk in inpatient and low risk in outpatient, or type HL, iii) low risk in inpatient and high risk in outpatient, or type LH, and iv) low risk in both sources, or type LL. Even under the independence assumption, the model presents several technical difficulties. First, a contract in this setting must specify a premium, the coverage for inpatient services, and the coverage of outpatient services, hence a triplet of endogenous variables for each type, or 12 variables to be determined. Second, there is no natural ordering of the four types in the grounds of risk: types: HL and LH cannot be ordered. This implies that there is a large set of possible cases depending on which is the *subset* of incentive compatibility constraints that are binding. We therefore opt to characterize a fully separating competitive equilibrium where: (i) coverage is full for the service at which an

individual is a high risk (no distortion at the top); (ii) zero expected profits are obtained at each contract; and (iii) only four of the incentive compatibility constraints are binding, namely all the downward-adjacent. We prove that these properties lead to a (separating) candidate where all other incentive compatibility constraints are also satisfied. Our equilibrium candidate is therefore a fully separating equilibrium menu of four contracts that preserves the Rothschild and Stiglitz (1976) basic finding that higher risk types enjoy higher coverage. We also analize the case where the information is symmetric in one of the dimensions, that is, where the observables used to condition contracts are rich enough so that in the given cell there is no residual heterogeneity. In this case we find that all individuals obtain full coverage in the corresponding dimension. This finding is behind the tests of asymmetric information.

The model also yields a novel prediction that is relevant in explaining why the empirical literature has found mixed evidence on the presence of asymmetric information in insurance markets. We provide conditions under which the intermediate type HL enjoys less coverage than type LL in the second dimension i.e. outpatient care. Notice that when comparing the outpatient coverage of type HL and of type LL, one is comparing two types that have the same level of risk (low) in the outpatient dimension. Therefore, the positive correlation property is silent. This phenomenon, which we denominate by *coverage reversion*, occurs when the distance between types in the inpatient dimension is smaller than that in the outpatient dimension.[4]However, our finding implies that if one were to aggregate risk into a single index and coverage also into a single index, the correlation between this aggregate risk and coverage indices would underestimate the presence of adverse selection.

The intuition for this result is as follows. Due to the "no distortion at the top property", types HH and HL enjoy full coverage in the inpatient dimension. Therefore, the only way to avoid that HH mimic HL is to distort coverage in the outpatient dimension. It is true that the symmetric argument can be applied to types HH and LH. However, when types are very similar in the inpatient dimension, a distortion in the inpatient coverage offered to LH is not a very effective tool to induce separation. Since distortions come at a cost, the insurer will tend to reduce distortions (that is, provide relatively generous coverage) in LH's inpatient dimension. Now, when types LH and HL consider to mimic type LL, they look at the generosity of both the outpatient and the inpatient

[4]This result can also appear in the symmetric situation: if the distance between types in the outpatient dimension is sufficiently close to that in the inpatient dimension, then the intermediate type LH will enjoy less coverage than type LL in the inpatient dimension.

coverages, and hence distortions in any of these two dimensions serves as a screening device. Since HL's distortion in outpatient coverage was severe to start with, whereas LH's distortion in inpatient coverage was mild, there is more room in distorting inpatient coverage than the outpatient coverage at contract LL. Moreover, HL and LL have the same risk in the outpatient dimension so again it is more effective to distort the first dimension than the second. Hence the distortion is deposited on LL's inpatient coverage. This explains why distortions in outpatient coverage are larger for type HL than for type LL.

The Chilean private health insurance market is ideal for testing AI because not only regulation limits the number of variables affecting private premiums to only three, leaving ample room for adverse selection, but also because we can observe some of the unused variables which constitute potential sources of adverse or advantageous selection (Finkelstein and Poterba, 2014). With this in mind, we search for evidence of asymmetric information and coverage reversion using both one-dimensional, more naive or standard methods, and two-dimensional methods, which should acommodate the inpatient-outpatient dichotomy observed also in the data.

The main disadvantage of the one-dimensional methods is, of course, the separate treatment of the inpatient and outpatient components, hence, failing to account fully for the multi-dimensionality of the health insurance plans. Amongst one-dimensional methods, we include very parsymonious regression models of individual-level inpatient and outpatient coverages against individual risk —measured either in continuous form and in the form of type dummy variables— for each cell and insurer company. We define a cell as a unique combination of individual gender and age group. There are, thus, 14 such cells per insurer. The individual type dummy variables were constructed by assigning individuals to a risk type (HH, HL, LH, LL) according to their health care usage. These regressions may deliver biased results for at least two reasons. First, because moral hazard, particularly in the outpatient dimension, biases the estimated coefficients. Second, the assignment of individuals to types may be incorrect. For example, we may assign an individual to type HH when he/she is considered as a type HL by insurers.[5] Having these caveats in mind, our first two "tests" reveal considerable heterogeneity across insurers with some insurers having none or limited evidence of adverse selection while others having strong evidence. The evidence of adverse selection is slightly stronger in the

[5]Ideally, we should use past or ex-ante information to define types. This is, unfortunately, not available to us. We follow the literature in using ex-post usage to define ex-ante risk. This approach may be problematic in the presence of moral hazard since ex-post risk would depend on coverage.

outpatient dimension, perhaps fueled by biases coming from potential moral hazard. Interestingly, we find scarse evidence of advantageous selection. Regarding evidence of coverage reversion, it is only significant for one insurer and it concerns the contracts of types HL and LL.

We end the one-dimensional methods section with an application of the standard PCT to each dimension independently. Our application of the PCT consists in estimating separate correlations between measures of the beneficiaries' risk—such as frequency or usage of medical inpatient and outpatient services—and the average level of inpatient and outpatient insurance coverage, respectively, conditional on the individual characteristics that insurance companies are allowed to use for pricing and an insurer fixed effect (Chiappori and Salanié, 2000). We also show estimations which include the unused variables income and location in our PCT tests. The correlations are derived from bi-probit models and hence require the dichotomization of both the coverage and risk variables, which introduces a level of discretion into the method. We reproduce the results for different cutoffs used in the dichotomization. Another Results show that the risk-coverage correlation estimate is sensitive to the particular cutoffs used to dichotomize the coverage variables. Depending on the cuttoffs, there is some evidence of positive and statistically significant correlation for inpatient care, i.e. adverse selection, and no evidence of a positive correlation for outpatient care, despite the likely upper bias due to ex-post moral hazard in outpatient usage (this result is similar to the one obtained by Sappeli and Vial, 2003 for independent workers). As for the lack of evidence of adverse selection in the outpatient component of care, several possible explanations exist. The first one is that other dimensions of privately known heterogeneity lead to advantageous selection that compensates both the risk-based adverse selection and the moral hazard bias. The other is that insurers only use screening contracts along the inpatient component. A third explanation, and the motivation for our non-parametric approach, is that one needs a full-test of AI, rather than a partial test, where the inpatient and outpatient components are treated jointly. The approach assesses whether the empirical distribution of risks and contracts matches the theoretical counterpart conditional on the same variables.

In the two-dimension part of the empirical section, we implement a non-parametric check of adverse/advantageous selection based on the prevalence of observations of high(low) risk individuals with high(low) coverage contracts per insurer and gender-age cell. The idea is to assess whether the empirical distribution of risks and contracts matches the theoretical counterpart. This check

demands the dichotomization of the coverage variables. Because the choice of cutoffs used for the discretization of the coverage variables may affect results, we show results using several cutoffs. For some insurers we do find robust evidence of adverse selection in both inpatient and outpatient dimensions while for others results are more dispersed and may depend on the coverage cutoffs used leading to advantageous selection in inpatient or even evidence of full information. An advantage of this approach is that it fully accounts for the multidimensionality of the insurance contracts.

To sum up our extension to two sources of risk is important for at least four reasons: a) individuals may have different risk profiles along these dimensions, implying that risks may not be ranked e.g., the case of chronic diseases is typically characterized by low inpatient risk and high outpatient risk while women of childbearing age may have high inpatient risk (due to labor) and low outpatient risk; b) insurance contracts may have different degrees of coverage along these two dimensions; c) there may be asymmetric information in one risk dimension and not in the other, and, d) and most importantly adverse selection (and therefore the presence of asymmetric information) may be underestimated if one takes aggregate measures of risk and coverage in order to reduce the analysis to one of single-dimensional screening.

The rest of the paper is structured as follows: Section 2 reviews the most relevant literature. Section 3 describes the Chilean health system; Section 4 describes our dataset; Section 5 describes the theoretical model; Section 6 describes the empirical methodology; Section 7 concludes. The Appendix A contains all proofs and Appendix B contains additional tables and figures.

2 Literature Review

There are very few theoretical works dealing with multiple sources of risk. Fluet and Pannequin (2002) study, as we do, a model with two sources of risk but they restrict their analysis to the two intermediate types (HL and LH in our notation). Moreover, their objectives are very different from ours, namely, to study whether the insurance market gains efficiency when the coverages for each of the two risks are offered by different firms rather than have the two coverages bundled in a single contract at all firms (as it is the case in our paper). Janssen and Karamychev (2008) also study two sources of risk but most of their results are derived for the case where there are no individuals that

are low risk in both dimensions. Incidentally, these authors prove that in such a market the zero profit condition as well as the "no distortion at the top" must hold in a competitive equilibrium. These conditions are instead imposed in our model, but we do not impose any limits on the set of types that are present.[6]

Regarding the existence of more than one source of heterogeneity, both the theoretical and empirical literature are abundant and growing. However, in this literature the additional source of heterogeneity is not another dimension of risk, but rather individuals' preferences over risk or over health as a good. Under perfect competition, Villeneuve (2003), Smart (2000), and Wambach (2000) deal with risk and risk attitude as the two dimensions of (privately known) heterogeneity. The prediction of these models is that the PCT remains valid: in the presence of asymmetric information in these two variables, riskier individuals purchase more generous coverage These results are generalized by Chiappori, Jullien, Salanie and Salanie (2006, henceforth CJSS). The only requirements for the PCT to hold is that consumers are rational and that profits per contract are (weakly) decreasing in coverage generosity. Perfect competition is a particular case where both conditions hold. CSSJ recognize that under sufficiently strong market power the PCT is no longer valid. de Meza and Webb (2001) present a model where individuals also differ in risk and preferences for risk, but add two novel ingredients: the possibility that less risk tolerant individuals engage in precautionary behavior and the presence of administrative costs in the implementation of contracts. They show that in equilibrium coverage and risk may be negatively correlated. Boone and Schottmüller (2017) analyze a setting where the single crossing condition fails to hold due to the combination of three forces: heterogeneity in wealth, heterogeneity in health and ex-post moral hazard. They also conclude that the correlation between risk and coverage can be negative in the presence of market power, a result that is consistent with CSSJ's predictions.

In our theoretical model the only sources of heterogeneity are the risks of needing each of the two health care services and we rule out moral hazard (although we informally discuss the consequences of adding this phenomenon on our results).

[6]We are not aware of any work dealing with the case where the two risks are not independent and the full set of types is analyzed. Notice that such setting would require the specification of the probabilities of four events: needing both services, needing only the outpatient service, needing only the inpatient service, not ill. This would require the specification of three parameters per type (the fourth being determined by the sum of probabilities equal to one), and therefore the existence of at least 8 types.

With respect to the empirical literature, although most evidence points to the existence of adverse selection on health insurance markets, both in the intensive margin—i.e. in the choice among plans with different levels of generosity in their coverage— and the extensive margin—i.e. in the choice between purchasing insurance and not (e.g. see the review in Cutler and Zechauser, 2000),[7] some recent papers find evidence of advantage selection.[8] The latter attribute the failure of the PCT to the multi-dimensionality of AI. As mentioned before, a likely candidate for an extra dimension leading to advantageous selection is risk aversion but the latest evidence shows that it plays a minor role in health insurance markets (Fang et al. 2008, Einav et al. 2013, Handel 2013). An exception where risk aversion seems to be important is the long-term care insurance market, as shown by Finkelstein and McGarry (2006). Another prominent candidate is cognitive ability (Fang et al. 2008) and Keane and Stavrunova (2016). In general, any private information could function as a source of advantageous selection if it is positively correlated with insurance coverage and at the same time negatively correlated with risk.

Although our multidimensional risk approach also provides reasons why the PCT may underestimate the presence of asymmetric information, our theoretical and empirical approach differs from the literature: our source of multidimensionality does not lie in individual's attitude towards risk, their preference for health, or their cognitive ability, but on the multidimensional nature of the sources of risk themselves. Notice that these dimensions are not only directly relevant to the insuree (like cognitive ability or risk preference are) but also to the insurer. In this sense one could say that while risk is a "common value", cognitive ability is a "private value". Our multidimensionality is more symmetric in the sense that both sources of risk are common value. This allows us to derive the separating equilibrium with tools that are closer to those in RS.

[7]Some more recent papers finding evidence of adverse selection in health insurance markets, among others, are: Olivella and Vera-Hernández, 2013, Sapelly and Vial, 2013, Panthöfer, 2016, Bolhaar et al. 2012.

[8]Cardon and Hendel (2000) do not find evidence in favor of adverse selection in their structural model. While Fang et al. 2008 and Keane and Stavrunova (2016) find advantageous selection in Medigap coverage (both papers test the extensive margin), Cutler, Finkelstein and McGarry find that although individuals involved in riskier activities (or not involved in risk-reducing activities) are less likely to hold private acute health insurance and Medigap coverage, there is not a clear relationship between risky behavior and medical expenses, with some risky behaviors associated with lower and others with higher medical expenses. Finkelstein and McGarry (2006) find evidence of advantageous selection in the long-term care insurance market.

3 The Chilean Health System

The Chilean Health Insurance market is characterized by the coexistence of public and private insurance. The public insurance provider is called FONASA. In 2007, there were 14 private insurers generally designated as "ISAPREs" (from the Spanish acronym of *Instituciones de Salud Previsional*). Insurance is mandatory for all employees and retirees but they may choose between public or private coverage. Individuals who are not employees, e.g. the self-employed, may choose to be uninsured or opt for either private or public insurance. Private and Public insurance are, therefore, substitutes in the Chilean market.[9] In 2007, FONASA covered $11,740,688$ individuals or 70.4% of the population whereas ISAPREs covered a total of $2,776,912$ individuals or 16.6% of the population.[10] Our data is restricted to beneficiaries from ISAPREs.

FONASA cannot deny coverage to any individual and its premiums do not depend on the number of dependents. Hence low income individuals are highly subsidized and over-represented in FONASA's pool of beneficiaries (Paraje and Vásquez, 2011). In contrast, ISAPRES can deny coverage to new clients based on pre-existing illnesses. Existing clients of ISAPREs cannot be denied coverage but may face premium increases.

There are two types of ISAPREs depending on whether their clients are drawn from the entire population or come from a job-related group. "Open" ISAPREs may accept any individual as beneficiary. "Closed" ISAPREs only accept beneficiaries that fulfill some condition, usually related to employment at a particular employer/institution or sector (e.g. coal production companies).[11] In 2007, there were 6 closed ISAPREs, and 8 open ISAPREs. Our model and empirical study only applies to open ISAPREs, i.e. those competing for clients. The closed ISAPREs account for only 4.2% of the beneficiaries.

The structure of copayments is described in the insurance plan and it may differ by medical

[9] Amongst private beneficiaries, it is common to hold a complementary private insurance. Women, for example, tend to contract insurance against breast cancer. These insurance plans, which are offered either by ISAPREs, banks, or insurance companies, are complementary private insurance that cover expenses such as copayments and hospitalizations, up to some amount, in the event of pre-specified *catastrophic* illnesses. This complementary coverage is in theory compatible with either private or public insurance but more common amongst private insurance holders.

[10] Source: (http://www.fonasa.cl/wps/wcm/connect/internet/sa-general/informacion+corporativa/estadisticas+institucionales/estadisticas+institucionales).

[11] Most, if not all, closed ISAPREs were created by worker unions that offered medical insurance to their affiliates before the approval of the law of ISAPREs in May 1981. After the enactment of the law, these organizations were restructured as ISAPREs to continue functioning.

procedure, by specialty of the physician, and by provider. The insurance plan also establishes a maximum amount covered for each service, implying that the beneficiary should pay out-of-pocket for the difference between this cap and the price charged by the provider. These caps, which are easily reached for the average plan, vary by procedure and specialty and are most common for ambulatory procedures and visits. Each contract also establishes a cap on the total annual expenditures that are covered by the ISAPRE but, contrary to service caps, these are usually sufficiently high and often not binding.

The pricing of private plans is subject to some legal, although mild, restrictions. ISAPRES set a "reference premium" (from the Spanish *precio base*) for each plan which they can modify annually and unilaterally. The premium paid by each beneficiary for a given plan depends directly on the reference premium and on a factor load which is a function of the policyholder's age, gender, and the inclusion of dependents in the policy.[12] These restrictions imply that two people with the same age, gender and codependants cannot be charged different premiums for the same plan. Although ISAPRES are free to establish the factor loads, regulation enacted in May 2005 limited the number of factor loads by ISAPRE to two, limiting the extent to which factor loads could be used to price discriminate. In 2007 alone there are $21,065$ plans chosen by at least one beneficiary.[13] By 2011, the number of plans exceed $52,000$ (Atal, 2016). This is feasible because in contrast to more regulated health insurance markets, such as the *individual mandate* in the US, there is no limit to the number of plans issued by each ISAPRE, and the menu of plans offered to any single (potential) client is not observed by outsiders.

4 Data and Descriptives

The data was provided to us by the Superintendencia de Salud del Gobierno de Chile. It constitutes a unique individual-level dataset compiled from administrative records from all private health insurers in Chile and contain all claims to ISAPREs during 2007. Claim data from insurance companies is typically viewed as a noisy measure of individual real risk since the decision to file a claim is

[12]Some exceptions are family plans that have lower effective premiums for dependents (children and spouses) when the dependents' income is below the minimum legal wage. Other examples are plans that offer reduced coverage for specific events such as childbirth, and physician visits. For these events the coinsurance rate may reach 75%, but the plan is cheaper than a comparable plan without reduced coverage.

[13]We count the number of plans with different IDs as are reported in the administrative records.

endogenous and depends on the the characteristics of the insurance contract and on the costs of filing claims (Chiappori and Salanié, 2000; Cohen and Siegelman, 2009). In Chile, however, claims are filed automatically and with zero cost when the service is provided, what has been denominated as *sistema de bonificacion inmediata*, which guarantees that everyone files a claim. The claim system also guarantees that the date of the claim, which we observe in our data set, coincides with the date the service was provived.

Crucial to our analysis, the dataset contains all the variables that ISAPREs are allowed to use for pricing i.e., age, gender, and the existence of dependents for each beneficiary. Additionally, other individual characteristics such as whether the individual is the main policyholder, the labor market status of the policyholder (e.g. employed, self-employed, retired, or voluntary contributor), the individual's region of residence and, in the case the individual is employed, income is also available in the dataset. Dependents can be matched with the main policyholder to reveal their relationship (e.g. spouse, children, etc.).

The dataset also provides for each claim the amount that is paid by the beneficiary and the amount paid by ISAPRE for each health service provided to the beneficiary or his/her dependents. There is also information on the main characteristics of the insurance contract such as the identity of the insurer (ISAPRE), the type of plan (individual, family, or collective), whether the plan has a preferred provider,[14] whether the plan has reduced coverage for birth delivery, and/or for physician fees, and the premium paid. When the plan specifies a preferred provider, we observe whether or not that provider is chosen for each service.

Finally, the dataset contains plan-specific summary information that the regulator requires from all ISAPREs such as the average copayment for outpatient care services, and the average copayment for inpatient care services. We use these two variables in the definition of our main dependent variables when searching for evidence of AI in Section 6.

Importantly, we lack information on: 1) the subset of plans or menus offered to each beneficiary, we can only observe the chosen plan; 2) the detailed copayment structure of the chosen plan; we

[14]When a provider is stated in the contract as "prefered provider" it generally implies that copayments when using that provider are lower. If the prefered provider is an expensive clinic, however, that may imply a higher premium for the given coverage. In this paper, we abstract from this characteristic although 55% of the plans include a preferred provider clause with some ISAPRES applying such a clause to 99% of their plans and other ISAPREs to none of their plans.

can only observe copayments for those services actually provided; 3) the prices negotiated by the ISAPREs with each health provider when the beneficiary does not use services.

We restrict our sample to employees between 25 and 60 years old, with no dependents, and who remain insured with the same individual plan throughout 2007 in an open ISAPRE. We restrict the sample to employees because they are the only group for whom insurance is mandatory. We drop individuals with dependents because we want to make sure individuals take their own risk into account, rather than that of others, when choosing an insurance plan. We further restrict the sample to individuals that did not change plans during 2007 in order to assign them a unique coverage level for the entire year. Finally, we only consider open ISAPREs since these are the ones among which individuals can freely choose their insurance plans.[15] By restricting the sample to be as homogenous as possible we are restricting the amount of asymmetric information in the sample.

4.1 The Market

There are eight Isapres in competition for clients in Chile in 2007. Table 4.1 shows the distribution of beneficiaries in our working sample by Isapre and region of the country in 2007. Around 61% of beneficiaries live in Santiago (region 13). Santiago also represents around 60% of the market for most Isapres which means that most Isapres have a proportional presence in Santiago. The exceptions are Isapre 2, which beneficiaries are mostly in region 2 (77%) with a negligible presence in Santiago (1%), and Isapres 1 and 8 with very strong presence in Santiago (80 and 78%, respectively) and almost zero presence in the rest of the country. The countrywide Herfindhal index (HHI) of 0.19 is 50% larger than the one obtained with equal-sized firms. Nonetheless, this countrywide index does not show a strong case of concentration. When computing HHIs for each regional market (see last column of Table 4.1), we find surprisingly similar values, between 0.18 and 0.24, and, hence, there is no strong evidence of market concentration in regional markets either.

[15] According to Atal (2016), individuals that switch plans or insurance company should be of a lower risk than non-switchers. The reason lies in the higher reclassification risk and probability of coverage denial amongst the latter. However his empirical results show that only 5% of the individuals are locked-in in the Chilean private health insurance i.e. fail to switch due to these risks. We, therefore, abstract from this potentially small selection in our sample.

Table 1: *Distribution of Beneficiaries by Isapre and Region*

	Isapre																	
Region	**1**		**2**		**3**		**4**		**5**		**6**		**7**		**8**		**%Pop**	**HHI**
	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	%	
1	6	0%	719	10%	21	0%	1,749	3%	7,154	6%	2,290	3%	3,154	3%	3,214	2%	3.1%	0.24
2	0	0%	5,575	77%	20	0%	3,775	6%	4,756	4%	3,917	5%	2,770	2%	2,673	2%	4.0%	0.18
3	0	0%	464	6%	11	0%	2,214	4%	1,150	1%	789	1%	597	0%	1,594	1%	1.2%	0.21
4	0	0%	433	6%	37	0%	1,431	2%	2,052	2%	1,446	2%	1,322	1%	2,929	2%	1.6%	0.20
5	324	9%	9	0%	4,348	12%	5,124	8%	7,489	6%	4,152	5%	9,244	8%	7,268	5%	6.4%	0.18
6	11	0%	2	0%	92	0%	5,224	8%	2,768	2%	2,028	3%	4,276	4%	2,875	2%	2.9%	0.22
7	8	0%	0	0%	592	2%	1,872	3%	2,556	2%	5,270	7%	2,405	2%	3,163	2%	2.7%	0.21
8	234	6%	5	0%	2,894	8%	15,214	24%	8,718	7%	3,118	4%	5,468	5%	4,812	3%	6.9%	0.23
9	136	4%	3	0%	1,811	5%	3,700	6%	4,280	4%	2,058	3%	3,048	3%	2,056	1%	2.9%	0.18
10	13	0%	0	0%	3,035	8%	8,900	14%	8,141	7%	3,588	5%	4,315	4%	3,063	2%	5.3%	0.20
11	3	0%	0	0%	8	0%	413	1%	670	1%	406	1%	686	1%	300	0%	0.4%	0.21
12	0	0%	6	0%	10	0%	2,056	3%	1,648	1%	949	1%	771	1%	1,186	1%	1.1%	0.22
13	2,986	80%	49	1%	24,648	66%	11,317	18%	68,455	57%	49,532	62%	81,741	68%	122,866	78%	61.4%	0.22
Total	3,721	100%	7,265	100%	37,527	100%	62,989	100%	119,837	100%	79,543	100%	119,797	100%	157,999	100%	100%	0.19
Share		0.6%		1.2%		6.4%		10.7%		20.4%		13.5%		20.3%		26.8%		

Table 2 presents descriptive statistics for the main variables of interest by ISAPRE. In the first panel, we show basic demographics of beneficiaries. The average beneficiary is 40 years old and male. A high proportion (61%) reside in the Metropolitan Region of Santiago. Figure 7 in Appendix B shows evidence that in our sample low risk individuals may be overrepresented, which is consistent with previous research by Sappeli and Vial (2003) whereby FONASA ends up absorving higher risks. Lacking data from FONASA, we take this selection as given and worry about the self-selection of contracts amongst clients of a given ISAPRE, what we refer to as the intensive margin adverse selection.

Table 2: **Descriptive statistics by ISAPRE**

	Isapre								**All**
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**sample**
Demographics									
Male (%)	56.6	71.1	54.9	57.1	74.9	57.6	58.3	62.4	62.5
Age (years)	39.4	39.4	40.4	39.5	40.6	39.9	40.3	39.2	39.9
Women 25 to 40 (%)	24.0	15.2	25.4	23.9	11.9	24.0	21.6	20.6	20.1
Average income (in dollars)	1148	1508	1774	1653	1379	1863	1482	1464	1547
Income missing (%)	11.2	12.5	7.4	5.4	9.3	3.6	10.2	9.1	8.2
Residing in Metro Region (%)	80.2	0.7	65.7	18.0	57.1	62.3	68.2	77.8	61.4
Usage									
No usage of insurance in 2007	22.6	13.5	12.6	15.5	24.0	11.7	13.8	20.2	17.5
Average annual Health expenditure (in US dollars) conditional on usage	615	1347	1459	870	645	1357	1183	964	1033
Hospitalized in 2007 (%)	5.8	8.7	11.5	8.1	6.1	7.1	5.8	8.7	7.4
Average number of physician visits in 2007	3.7	6.3	4.0	3.7	2.9	4.1	4.7	3.4	3.8
Hospitalized for childbirth in 2007 (women, 25 to 40) (%)	6.6	3.1	9.0	6.7	5.4	8.5	5.6	8.1	7.1
Coverage									
Inpatient coverage (%)	100.0	100.0	92.0	99.0	92.	75.6	90.8	92.4	90.6
Outpatient coverage (%)	80.0	100.0	74.6	82.0	72.2	74.4	71.2	80.0	76.0
Inpatient coverage $\geq 90\%$	100.0	100.0	98.3	97.6	98.5	44.2	98.7	94.1	90.0
Outpatient coverage $\geq 80\%$	100.0	100.0	29.8	93.9	23.0	36.7	11.1	50.3	39.2

4.2 Usage

In the second panel of Table 2 we show basic statistics regarding overall usage and usage by ISAPRE. Most beneficiaries are users, i.e. they filed at least one claim in 2007, only 17.5% did not. Among users, the annual average health care expenditure is $1,033$ US dollars. Hospitalizations are a relatively

rare event with only 7.4% of the beneficiaries hospitalized during 2007. Among 25 to 40 year-old women, 7.1% were hospitalized for childbirth. Beneficiaries paid an average of 3.8 visits to physicians during 2007.

When comparing usage across ISAPREs, we verify that it is not evenly distributed, implying that neither is risk. The differences in risk composition across ISAPREs can be seen, for example, in the percentage of beneficiaries that have at least one hospitalization in 2007, which ranges from 5.8% to 11.5%, or on the noticeable differences in average total expenditures which vary from 615 to 1459 USD. On the contrary, the average number of physician visits is quite similar for most ISAPREs.

Based on this pattern of usage, our baseline measures of inpatient and outpatient risk are going to be an indicator function that takes value one if the individual has been hospitalized at least once during 2007 and zero otherwise, and an indicator function that either takes the value one if the individual has 5 or more visits to the physician during 2007 (the sample average is 4 visits) or has more visits than the average in his/her cell.

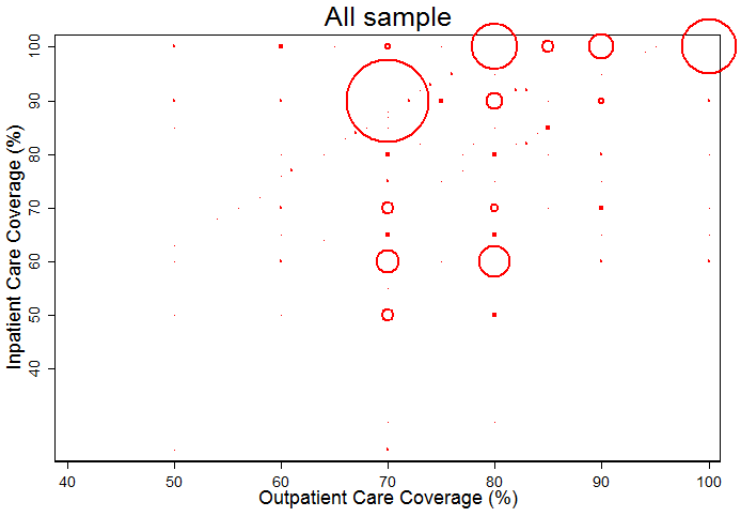
4.3 Coverage

ISAPREs must inform the regulator about each plan's average coverage for inpatient and outpatient care. The average contract is relatively more generous in the inpatient dimension, with 90.6% coverage, than in the outpatient dimension, with an average coverage of 76% (see statistics in the third panel of Table 2). There is also more heterogeneity across ISAPREs in the coverage generosity of outpatient services than of inpatient services.

Each ISAPRE offers a high number of different plans. Altogether, there are 21,065 different active plans in our sample although they correspond to only 88 different combinations of average inpatient and outpatient coverages.[16] Figure 1 shows the distribution of plans held by the beneficiaries in our sample according to the plan's average inpatient and outpatient coverages as reported by the ISAPREs to the regulator. The size of the bubble represents the percentage of plans with each combination of average inpatient and outpatient coverages. The most frequent plan covers on average 90% of inpatient care and 70% of outpatient care.

[16] Plans may differ in other dimensions such as preferred provider; copayment structure by service or provider, and service caps.

Figure 1: Distribution of plans by outpatient and inpatient care coverage



When we replicate Figure 1 by ISAPRE (see figure 2), there is still a considerable amount of heterogeneity across these dimensions for most Isapres with the noticeable exception of Isapres 1 and 2, however.

To help visualize the differences in coverage, we construct two discrete measures of coverage that reflect high or low coverage in the inpatient and outpatient care dimensions, respectively. The inpatient care coverage dummy variable takes the value of 1 when the plan has 90% or more inpatient coverage and takes the value zero otherwise. The outpatient dummy variable takes the value of 1 when the plan has 80% or more outpatient coverage, and zero otherwise. The thresholds of 90 and 80 percent for inpatient and outpatient coverage were selected based on the distribution observed in Figure 1.[17]

In the next Section, we derive a theoretical model that helps explain the equivalent to Figure 1 by cell. In particular, we want to know if such a configuration of plans can arise in equilibrium and which individuals choose each plan. As an example, in Table 3 we divide the coverage space of Figure 1 into four types of contracts (high and low coverage of inpatient and outpatient coverage, respectively) and compute the fraction of beneficiaries with at least one inpatient episode as well as the average

[17]In the bottom panel of Table 2, we show evidence of important differences across ISAPREs in terms of coverage generosity as defined by the dummy variables proposed; while all contracts offered by ISAPRES 1 and 2 have inpatient coverages above 90% and outpatient coverages above 80%, only 44.2% (36.7%) of the contracts offered by ISAPRE 6 have inpatient (outpatient) coverages above 90% (80%).

Figure 2:

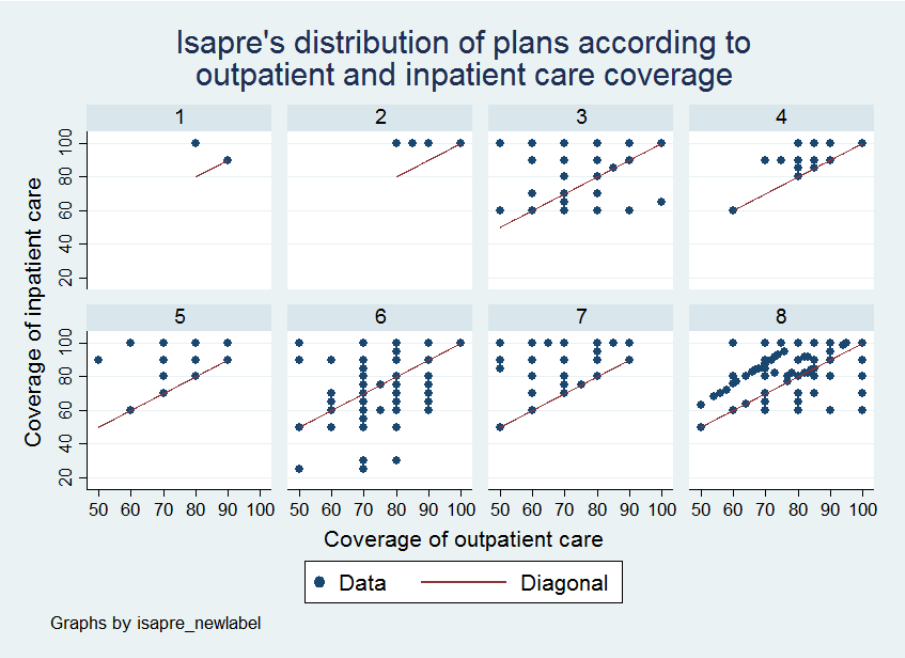


Figure 3: Distribution of plans by gender

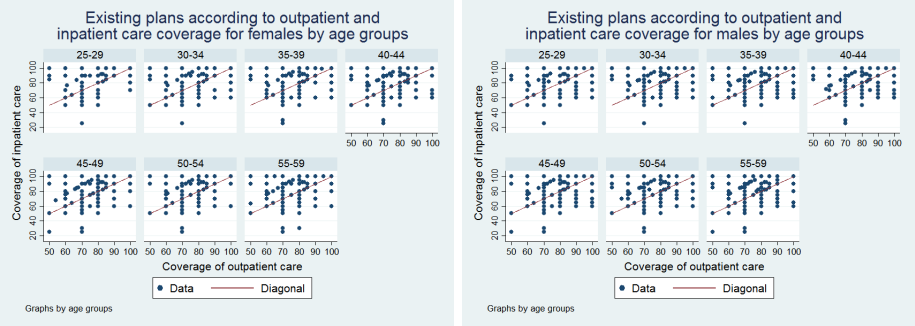


Table 3: Distribution of risks by insurance coverage

	MALE, 40-45 years old	
	High I-Low O	High I - High O
	$\#visits = 2.7$ $hosp = 0.05$ $Obs = 47,650$	$\#visits = 3.1$ $hosp = 0.06$ $Obs = 7,882$
	Low I-Low O	Low I- High O
	$\#visits = 2.4$ $hosp = 0.03$ $Obs = 4,720$	$\#visits = 3.1$ $hosp = 0.04$ $Obs = 242$

Note: "High" and "Low" stand for high and low (<90% inpatient and <80% outpatient) coverage, respectively of "I" (inpatienty) and "O" (outpatient) services.

number of outpatient episodes for $40-45$ year-old males. The individuals that choose high coverage of inpatient and/or outpatient services have a higher number of inpatient and/or outpatient episodes compared to those that choose lower coverages.

5 The model

5.1 The players

The players are a set of insurers and a set of individuals. There are 4 states of the world: Not ill, ill needing inpatient services only, ill needing outpatient service only, and ill needing both types of services. We represent these 4 states of the world as, respectively, $\Omega = \{\emptyset, i, o, b\}$. We assume for simplicity that the needs of inpatient and outpatient services are independent. This means that it suffices to define two probabilities: the probability that the individual needs inpatient services, p_i and the probability that the individual needs outpatient services, p_o.[18] The distribution of probabilities in the set Ω is given by $\{1-p_o-p_i+p_ip_o, p_i(1-p_o), p_o(1-p_i), p_ip_o\}$. Also for simplicity, we assume that insurance companies offer coverage in each of the services, inpatient and outpatient, independently. This is indeed the case for the Chilean case. For instance, they do not condition the

[18]Suppose that the two events were not independent. This would require establishing 3 distinct probabilities, and therefore $2^3 = 8$ types exist even in the simplest case of dichotomous types: LLL, LLH, LHL,...,HHH. No theoretical model exists dealing with a similar situation at the time of writing this paper..

coverage of outpatient services on whether the patient also uses inpatient services.[19] Therefore, an insurance contract establishes a premium P, a fixed level of coverage for inpatient services c_i, and a fixed level of coverage for outpatient services c_o. All individuals have the same initial wealth w. The expected utility of an individual who accepts an insurance package (P, c_i, c_o) and has probabilities p_i and p_o is given by:

$$\begin{aligned} Eu\left(P, c_i, c_o\right) = \left(1 - p_o - p_i + p_i p_o\right) u\left(w - P\right) + p_i \left(1 - p_o\right) u\left(w - P - \ell_i + c_i\right) + \\ p_o \left(1 - p_i\right) u\left(w - P - \ell_o + c_o\right) + p_i p_o u\left(w - P - \ell_i - \ell_o + c_i + c_o\right). \end{aligned} \tag{1}$$

where l_i and l_o describe the value of the inpatient and outpatient loss, respectively.

Individuals are characterized by two sets of variables. The first set contains the variables that are publicly observable and the law allows insurers to use for underwriting (in the case of Chile, gender and age). The second set contains the variables that are either not observable (like private information on unavoidable risks) or are observable but insurers are not allowed to condition either premia or coverage (in Chile, income and location). We conduct our theoretical analysis for a fixed class where class is defined by the first set of observables, for example, for 37 years old women. We also assume that any heterogeneity across individuals except for differences in risk (that is, in p_o and p_i) is completely eliminated once one conditions on class.[20]

For simplicity, we assume that there are two possible levels in the probability of inpatient (respectively, outpatient) services, $p_i^L < p_i^H$ (respectively, $p_o^L < p_o^H$). Therefore there are four types: (p_i^H, p_o^H), (p_i^H, p_o^L), (p_i^L, p_o^H), and (p_i^L, p_o^L); which we simply denote by HH, HL, LH, and LL, respectively. Let $T = HH, HL, LH, LL$ represent the set of all possible types. We use $x = HH, HL, LH, LL$ to represent a generic type. Hence the first letter in IJ refers to inpatient risk and the second to outpatient risk.

We can define the expected utility of an agent of type $x \in T$ accepting the contract aimed at the

[19] Jensen and Karamichev (2007) show that this assumption is inoquous if only tyes HH, HL,and LH are assumed to exist.

[20] There are three reasons why differences in income within a class are not of special concern. First, differences in income across the whole sample are relatively small since only individuals in the upper two deciles in the income distribution opt out of the public system. Second, ISAPRES may circumvent the law and condition their offers on income, for instance through selective advertising. Since we have data on income we can also condition our empirical analysis on income. Finally, the evidence on the sign of the effects of income on both risk and the willingness to pay for coverage is far from clear (see Panthofer, 2016, for example).

agent of type $y \in T$ as follows

$$\begin{aligned} Eu\left(x,y\right) = \left(1-p_o^x-p_i^x+p_i^x p_o^x\right) u\left(w-P^y\right) + p_i^x\left(1-p_o^x\right) u\left(w-P^y-\ell_i+c_i^y\right) + \\ p_o^x\left(1-p_i^x\right) u\left(w-P^y-\ell_o+c_o^y\right) + p_i^x p_o^x u\left(w-P^y-\ell_i-\ell_o+c_i^y+c_o^y\right) \end{aligned} \tag{2}$$

To simplify notation, we let $Eu\left(x,x\right) = [x]$ and $Eu(x,y) = [x,y]$.

A generic incentive compatibility constraint requires that a type x is not willing to pick the contract aimed to type y. It can be written as $[x] \geq [x,y]$. Whenever this expression is satisfied with equality, that is, if this incentive compatibility constraint is binding, we will represent the ensuing equation with the symbol $x \to y$.

Notice that the equilibrium needs to specify a contract for each type. Since there are 3 components in each contract and we have 4 types, we have 12 endogenous variables, namely $P^{IJ}, c_o^{IJ}, {c_i^{IJ}}_{i,j=L,H}$. We concentrate in the fully separating equilibrium candidate. We will show that this candidate is fully determined by the zero profit condition; no distortion at the top; and four (downward adjacent) incentive compatibility constraints.[21]

5.2 Constructing a fully separating equilibrium

In a fully separating equilibrium, all variables $P^{IJ}, c_o^{IJ}, {c_i^{IJ}}_{i,j=L,H}$ are potentially different. Hence we need 12 equations to determine the equilibrium. The conditions that we impose (developed in full below) to obtain the 12 equations are:

1) No distortion at the top in each dimension (4 equations);

2) Zero profits per contract when all individuals are truthful (4 equations);

3) The downward adjacent incentive compatibility constraints are binding, namely, $HH \to HL$, $HH \to LH$, $HL \to LL$, and $LH \to LL$ (4 equations).

[21] As in Wambach (2000), which also addresses a multidimensional screening model, we content ourselves in characterizing the necessary conditions for a separating equilibrium. Whether such equilibrium is robust to deviations is left for further research. Let us point out however, that, as shown by Olivella and Vera-Hernández (2007), introducing a slight horizontal differentiation does not alter the characterization of the separating contracts whereas deviations (i.e. by offering less distorted insurance contracts) cease to be profitable.

No distortion at the top

a) As usual, the individual who is risky in all dimensions will face no risk in either dimension:

$$c_i^{HH} = \ell_i \tag{3}$$

and

$$c_o^{HH} = \ell_o \tag{4}$$

b) Type HL cares a lot about inpatient coverage and little about outpatient coverage. Hence, more efficient separation is accomplished by having

$$c_i^{HL} = \ell_i \tag{5}$$

whereas $c_o^{HL} < \ell_o$.

c) Symmetrically, type LH cares little about inpatient coverage and a lot about outpatient coverage. Hence, more efficient separation is accomplished by having

$$c_o^{LH} = \ell_o \tag{6}$$

whereas $c_i^{LH} < \ell_i$.

The assumption that $c_i^{HH} = c_i^{HL} = \ell_i$ and $c_o^{HH} = c_o^{LH} = \ell_o$ has several implications. Consider contract $(P^{HH}, c_i^{HH}, c_o^{HH}) = (P^{HH}, \ell_i, \ell_o)$. All states of the world lead to the same final wealth $w - P^{HH}$. Therefore, the expected utility when accepting such contact is the same for all types, since probabilities cease to matter. In other words, $[HH] = [HL, HH] = [LH, HH] = [LL, HH] = u(w - P^{HH})$.

Consider now contract $(P^{HL}, c_i^{HL}, c_o^{HL}) = (P^{HL}, \ell_i, c_o^{HL})$. Notice that states $\emptyset$ and i lead to the same final wealth $w - P^{HL}$ since outpatient services are not required in either state and inpatient services are fully insured at this contract. The total probability of these two states is, for all types $IJ \in LL, LH, HL, HH$, given by $1 - p_o^J - p_i^I + p_i^I p_o^J + p_i^I(1 - p_o^J) = 1 - p_o^J$. Analogously, states o

and b also lead to the same final utility $w - P^{HL} - \ell_o + c_o^{HL}$ since outpatient services are required in both states (although not fully covered) and again inpatient services are fully insured. The total probability of these states is, for each type $IJ \in LL, LH, HL, HH$, given by $p_o^J(1-p_i^I) + p_i^I p_o^J = p_o^J$. Therefore, we can write $[IJ, HL] = (1-p_o^J)u(w - P^{HL}) + p_o^J u(w - P^{HL} - \ell_o + c_o^{HL})$. Notice that the right hand of the last expression is independent of the first type dimension I . Therefore:

$$[HH, HL] = [LH, HL] = (1-p_o^H)u(w - P^{HL}) + p_o^H u(w - P^{HL} - \ell_o + c_o^{HL}) \tag{7}$$

$$[HL] = [LL, HL] = (1-p_o^L)u(w - P^{HL}) + p_o^L u(w - P^{HL} - \ell_o + c_o^{HL}). \tag{8}$$

Symmetrically, consider contract $(P^{LH}, c_i^{LH}, c_o^{LH})$. States $\emptyset$ and o lead to the same final wealth $w - P^{LH}$, with total probability of these two states given by $1-p_i^I$; and states i and b also lead to the same final utility $w - P^{LH} - \ell_i + c_i^{LH}$, with total probability of these states given by p_i^I. Therefore, we can write

$[HH, LH] = [HL, LH] = (1-p_i^H)u(w - P^{LH}) + p_i^H u(w - P^{LH} - \ell_i + c_i^{LH})$ and

$[LH] = [LL, LH] = (1-p_i^L)u(w - P^{LH}) + p_i^L u(w - P^{LH} - \ell_i + c_i^{LH})$.

5.2.1 Zero profits (fair premia)

The zero profit conditions can be simply stated as follows[22]

$$P^{HH} = p_i^H c_i^{HH} + p_o^H c_o^{HH} = p_i^H \ell_i + p_o^H \ell_o \tag{9}$$

$$P^{HL} = p_i^H c_i^{HL} + p_o^L c_o^{HL} = p_i^H \ell_i + p_o^L c_o^{HL} \tag{10}$$

$$P^{LH} = p_i^L c_i^{LH} + p_o^H c_o^{LH} = p_i^L c_i^{LH} + p_o^H \ell_o \tag{11}$$

$$P^{LL} = p_i^L c_i^{LL} + p_o^L c_o^{LL} \tag{12}$$

[22]For $i = L, H$, $j = L, H$, we can write $P^{ij} = p_i^i(1-p_o^j)c_i^{ij} + p_o^j(1-p_i^i)c_o^{ij} + p_i^i p_o^j c_i^{ij} + p_i^i p_o^j c_o^{ij} = p_i^i c_i^{ij} - p_o^j p_i^i c_i^{ij} + p_o^j c_o^{ij} - p_i^i p_o^j c_o^{ij} + p_i^i p_o^j c_i^{ij} + p_i^i p_o^j c_o^{ij} = p_i^i c_i^{ij} + p_o^j c_o^{ij}$.
In sum, $P^{ij} = p_i^i c_i^{ij} + p_o^j c_o^{ij}$.

Notice that $P^{HH} = p_i^H \ell_i + p_o^H \ell_o > \left\{ \begin{array}{l} p_i^H \ell_i + p_o^L c_o^{HL} = P^{HL} \\ p_i^L c_i^{LH} + p_o^H \ell_o = P^{LH} \end{array} \right\}$. However, as we will see later on, c_o^{HL} can be lower than c_o^{LL} under some conditions. Therefore it is not necessarily the case that $P^{HL} > P^{LL} = p_i^L c_i^{LL} + p_o^L c_o^{LL}$. Symmetrically, c_i^{LH} can be lower than c_i^{LL} under some conditions and therefore it is not necessarily true that $P^{LH} > P^{LL}$.

Only the downward adjacent constraints are binding

We use the equations $HH \to HL$, $HH \to LH$, $HL \to LL$, and $LH \to LL$. Using previous results, we can rewrite $HH \to HL$ as:

$$[HH] \equiv u(w - P^{HH}) = (1 - p_o^H) u(w - P^{HL}) + p_o^H u(w - P^{HL} - \ell_o + c_o^{HL}) \equiv [HH, HL]$$

Using the zero profit conditions (9) and (10), we get

$$u(w - p_i^H \ell_i - p_o^H \ell_o) = (1 - p_o^H) u(w - p_i^H \ell_i - p_o^L c_o^{HL}) + p_o^H u(w - p_i^H \ell_i - p_o^L c_o^{HL} - \ell_o + c_o^{HL}) \quad (13)$$

or, letting $w_i' = w - p_i^H \ell_i$,

$$u(w_i{}' - p_o^H \ell_o) = (1 - p_o^H) u(w_i{}' - p_o^L c_o^{HL}) + p_o^H u(w_i{}' - \ell_o + c_o^{HL}(1 - p_o^L)). \quad (14)$$

Notice that this is an equation with a single unknown, c_o^{HL} and that the equation is independent of p_i^L. Moreover, we can prove that the equation is satisfied for some (partial) coverage c_o^{HL*}. Formally,

Lemma 1: The binding ICC $HH \to HL$ uniquely determines coverage c_o^{HL}. There exists $0 \leq c_o^{HL*} \leq \ell_o$ such that $HH \to HL$ is satisfied. This coverage is independent of p_i^L. By symmetry, using $w_o{}' = w - p_o^H \ell_o$ and the zero profit conditions (9) and (11), we can rewrite $HH \to LH$ as

$$u(w_o{}' - p_i^H \ell_i) = (1 - p_i^H) u(w_o{}' - p_i^L c_i^{LH}) + p_i^H u(w_o{}' - \ell_i + c_i^{LH}(1 - p_i^L)). \quad (15)$$

We also obtain a lemma that is also symmetric to the previous one:

Lemma 2: The binding ICC $HH \rightarrow LH$ uniquely determines coverage c_i^{LH}. There exists $0 \leq c_i^{LH*} \leq \ell_i$ where $HH \rightarrow LH$ is satisfied. This coverage is independent of p_o^L.

The only remaining unknowns to be determined are c_i^{LL}, c_o^{LL}. For this we use binding constraints $HL \rightarrow LL$ and $LH \rightarrow LL$, as well as the zero profit condition (12). This leads to a system of two equations and two unknowns, as we show next. We first define $P^{HL*} = p_i^H \ell_i + p_o^L c_o^{HL*}$, $P^{LH*} = p_o^H \ell_o + p_i^L c_i^{LH*}$, and $P^{LL} = p_i^L c_i^{LL} + p_o^L c_o^{LL}$. Then, $HL \rightarrow LL$ implies:

$$\begin{aligned}
[HL] \equiv & (1-p_o^L)u(w-P^{HL*}) + p_o^L u(w-P^{HL*}-\ell_o+c_o^{HL*}) = \\
& (1-p_o^L-p_i^H+p_i^H p_o^L)u(w-P^{LL}) + p_i^H(1-p_o^L)u(w-P^{LL}-\ell_i+c_i^{LL}) + \\
& p_o^L(1-p_i^H)u(w-P^{LL}-\ell_o+c_o^{LL}) + p_i^H p_o^L u(w-P^{LL}-\ell_i-\ell_o+c_i^{LL}+c_o^{LL}) \equiv [HL, LL],
\end{aligned} \tag{16}$$

Similarly, $LH \rightarrow LL$ implies

$$\begin{aligned}
[LH] \equiv & (1-p_i^L)u(w-P^{LH*}) + p_i^L u(w-P^{LH*}-\ell_i+c_i^{LH*}) = \\
& (1-p_o^H-p_i^L+p_i^L p_o^H)u(w-P^{LL}) + p_i^L(1-p_o^H)u(w-P^{LL}-\ell_i+c_i^{LL}) + \\
& p_o^H(1-p_i^L)u(w-P^{LL}-\ell_o+c_o^{LL}) + p_i^L p_o^H u(w-P^{LL}-\ell_i-\ell_o+c_i^{LL}+c_o^{LL}) \equiv [LH, LL].
\end{aligned} \tag{17}$$

Coinsurances c_i^{LL}, c_o^{LL} are then computed by solving the system of equations given by (16) and (17) for c_i^{LL}, c_o^{LL}.

To sum up, the equilibrium candidate can be computed in three steps. In step 1, substitute the premia defined in (9) and (12) and the (top) coverages $c_i^{HH} = c_i^{HL} = \ell_i$ and $c_o^{HH} = c_o^{LH} = \ell_o$. In step 2, compute c_i^{LH} using the incentive compatibility constraint ensuring that type HH does not wish to mimic type LH; and compute c_o^{HL} using the incentive compatibility constraint ensuring that type HH does not wish to mimic type HL. These two equations are independent. In step 3, obtain c_o^{LL} and c_i^{LL} by solving the system of incentive compatibility constraints ensuring that the intermediate types HL and LH do not wish to mimic type LL. It is possible to sign some comparative statics that will be useful in deriving our main result. The first result is that, dimension by dimension, an increase in the low type's risk brings about an increase in the corresponding intermediate type coverage. In other words, the inpatient coverage for type LH increases with p_i^L and the outpatient coverage for type HL increases with p_o^L. Mathematically,

Proposition 1. $\frac{\delta c_i^{LH}}{\delta p_i^L} > 0$ and $\frac{\delta c_o^{HL}}{\delta p_o^L} > 0$.

In order to establish our main result, it is useful to study the special case where there is full information in one of the dimensions. Due to the symmetry of our model, without loss of generality we address the case where this occurs in the inpatient dimension.

5.2.2 A special case: Symmetric information in the inpatient dimension

We are now assuming that $p_i^L = p_i^H = p_i$. Unsurprisingly, we obtain that all types receive full insurance in the inpatient dimension. A perhaps less foreseeable result is that types LL and HL obtain the same (partial) coverage in the outpatient dimension. However, the intuition is simple: notice that these two types coincide in this dimension. Formally,

Proposition 2: If $p_i^L = p_i^H = p_i$ but $p_o^L < p_o^H$, then $c_i^{IJ*} = \ell_i$ for all $IJ \in T$ and $c_o^{LL*} = c_o^{HL*} < \ell_o$.

Let us now depart from this special case by slightly reducing the low type's risk in the inpatient dimension. this will lead to our main result.

5.2.3 Almost symmetric information in the inpatient dimension

We know from Lemma 1 that type HL's outpatient coverage does not depend on p_i^L. Therefore, even if p_i^L is reduced, this coinsurance stays the same. In the next proposition we establish that LL's outpatient coverage locally increases when his inpatient risk p_i^L falls below p_i^H. Therefore, LL's outpatient coverage will be above type HL's for p_i^L below, but sufficiently close to p_i^H. In other words, although type HL is (seemingly) riskier than type LL, the former enjoys less coverage. We formalize these results in the next proposition and corollary.

Proposition 3: A sufficient condition for $\left[\frac{\partial c_o^{LL}}{\partial p_i^L}\right]_{p_i^L = p_i^H} < 0$ is $\frac{c_o^{LL}}{\ell_o} > \frac{1-p_o^H}{1-p_o^L}$. This proposition leads to the most important result:

Corollary: conditional on $\left[\frac{\partial c_o^{LL}}{\partial p_i^L}\right]_{p_i^L = p_i^H} < 0$, there exists an open interval for the probability p_i^L of needing inpatient services for the low-risk type, namely $\left[p_i^H - \varepsilon, p_i^H\right]$, with $\varepsilon > 0$ such that the coverage in the outpatient dimension is larger for type LL than for type HL.[23]

We refer to this important result as "coverage reversion". Notice that any attempts to aggregate

[23] We provide a numerical example illustrating this proposition in Section 5.5

the two risks in a single dimension will have to conclude that type HL is more risky than type LL. Yet, type HL is less insured, although this is so in the dimension where the two types are identical. Therefore, this reversion does not contradict the positive correlation test, as this test is silent when one compares individuals with the same risk type. Our result warns the empiricist that aggregating risk into a single variable will lead to an underestimation of the positive relation between risk and coverage. Another question is whether, by aggregating coverage into a single dimension, one does obtain that LL has more coverage than HL, a phenomenon that we refer to as "aggregate coverage reversion". However, notice that $c_i^{LL} < \ell_i = c_i^{HL}$, which makes such aggregate reversion quite unlikely. Unfortunately, such comparison of aggregate coverage involves four different coverage levels: c_i^{LL}, c_o^{LL}, c_i^{HL}, and c_o^{HL}; that are tied through an intricate and non-linear set of incentive compatibility constraints. We have not been able to derive further analytical results in this regard. Numerical simulations suggest that aggregate coverage reversion can never occur. We further discuss the issue of coverage and loss aggregation in the next subsection.

5.3 Single loss and single coverage measures

In the received literature on insurance markets, researchers use a single measure of loss (typically related to claims) and a single measure of coverage. It is also often the case that one can only observe whether the individual has purchased insurance or not, which is obviously an unidimensional—and dichotomous—measure of coverage. Even when multi-loss and multi-coverage data is available, as in the Chilean case, one could be tempted to aggregate loss and coverage into a single dimension. As for the loss, we have already mentioned that type HL must lead to a higher measure of risk than type LL. However, when one has coverage reversion, type HL has less coverage than LL in the outpatient dimension but enjoys full coverage in the inpatient dimension. Hence the aggregation procedure matters, and is not obvious which is the "correct" procedure.

The simplest measure of aggregate coverage is expected coverage, defined as $\bar{c}^{ij} = p_i^i c_i^{ij} + p_o^j c_o^{ij}$, for any type $ij \in LL, LH, HL, HH$. Inspired by our numerical results, we establish the following:

Conjecture: Suppose that $c^{HL} < c^{LL}$. Then the incentive compatibility constraint$HL \to LL$ is violated.

The intuition goes as follows. Because the premia are fair by construction, a risk neutral in-

dividual of type HL will always prefer contract $C^{LL} = [P^{LL}, c_i^{LL}, c_o^{LL}]$ to contract contract $C^{HL} = [P^{HL}, c_i^{HL}, c_o^{HL}]$. Indeed, he obtains $w - p_i^L \ell_i - p_o^L \ell_o$ in the first case and $w - p_i^H \ell_i - p_o^L \ell_o$ in the second case, where $p_i^H > p_i^L$. Therefore type HL prefers to lie no matter the size of the coverages, as long as the coverages are fairly priced subject to truthtelling. Since the inequality is slack, for a given level of risk aversion there must exist sufficient incremental risk when lying in order to preserve incentive compatibility. If by contradiction, we have that $c^{HL} < c^{LL}$, which implies $c_o^{LL} > c_o^{HL} + \frac{(p_i^H \ell_i - p_i^L c_i^{LL})}{p_o^L}$, the incremental risk suffered when lying is small.

We have also experimented with another measure of aggregate coverage that is more favorable to obtaining aggregate reversion, namely, expected coverage over expected loss, or

$$\Psi^{ij} = \frac{\bar{c}^{ij}}{\bar{l}^{ij}}$$

where $\ell^{ij} = p_i^j \ell_i + p_o^j \ell_o$. We say that this alternative measure has ℓ^{ij} in the denominator, and $\ell^{LL} < \ell^{HL}$, which implies that $\Psi^{LL} > \Psi^{HL}$ even if $c^{LL} = c^{HL}$. However, we have not been able to find any numerical example for this possibility.[24]

5.4 General predictions

Our theoretical model predicts the configuration of coverages that is depicted in Figure 4, the theoretical analog of the curtain figure (Figure 1). The distance between coverages in that figure will be determined by the four probabilities $\left\{p_s^k\right\}_{\substack{s=i,o \\ k=L,H}}$, as well as the losses $\{\ell_s\}_{s=i.o}$.

However, according to the previous corollary, for certain values of the probabilities and losses, it it possible that the theoretical prediction shows an apparent reversal whereby the types HL and LL although having the same risk in the outpatient component have different coverages with the former having lower coverage as pointed in Figure 5.

It is obvious that if information in one of the dimensions was symmetric, then our model would be the same as in Rothschild and Stiglitz (1976), as confirmed by Proposition 2. For instance, if

[24] A third measure of aggregate risk could be considered, namely the expected fraction of coverage over loss, or $E_{ij}(c/\ell)$. However, we would run into the problem of defining what is the such fraction in the event $\emptyset$: both coverage and loss are zero and the fraction becomes indeterminate.

Figure 4: Possible configuration of plans

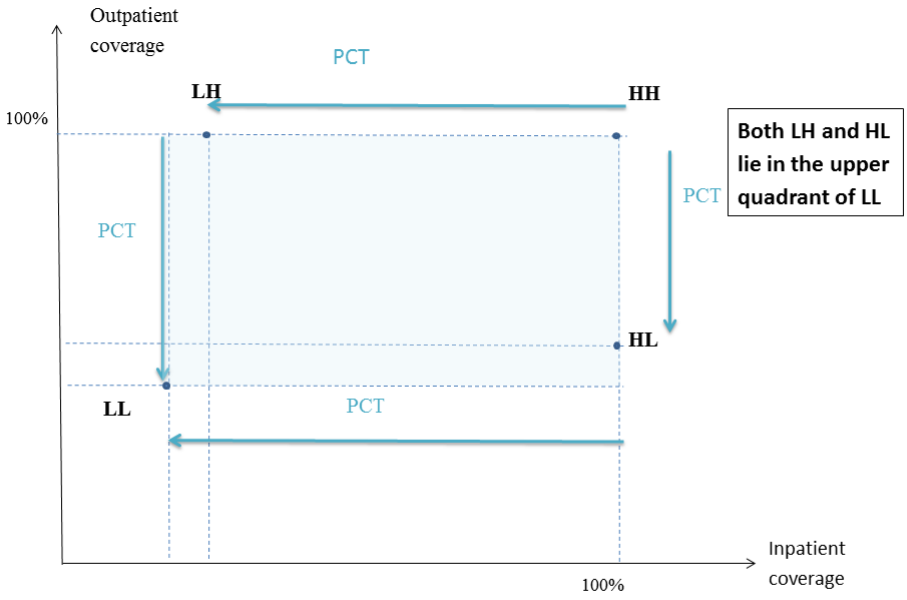


Figure 5: Possible configuration of plans

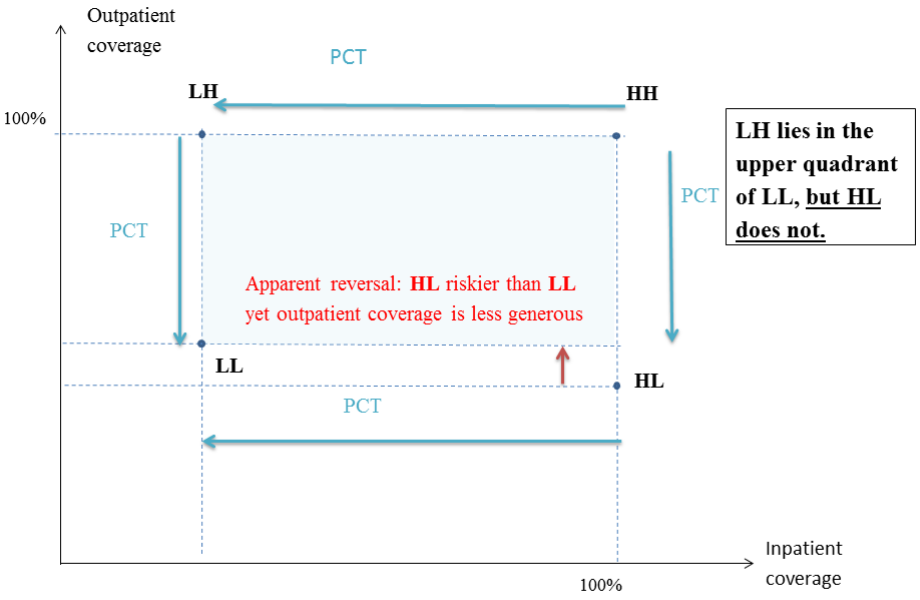
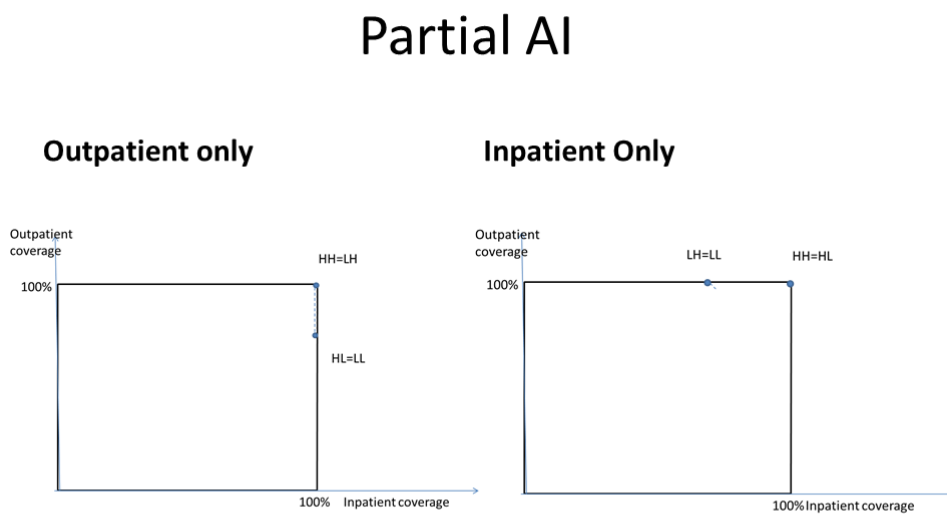


Figure 6: Partial Asymmetric Information

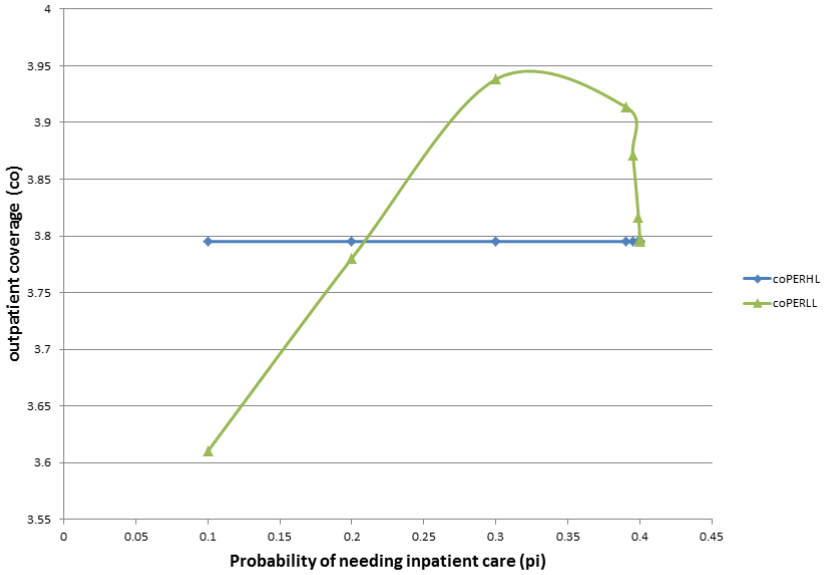


observables used by insurers completely and uniquely determine the probability of needing outpatient (inpatient) services, then the curtain figure predicted would have outpatient (inpatient) always with full coverage while for the other dimension there would be a low and a high coverage level.

5.5 Numerical Examples

In order for the real world case at hand (the Chilean private insurance market) to guide the numerical examples, we take a specific observable cell and analyze the parameters that can be elicited within this cell, namely males 35 - 39 years old. In this cell, the average yearly income is 16,800 dollars; the cost of an inpatient stay amounts to around 25% of this monthly income, whereas the cost of 10 outpatient visits is around 3.4%. We re-index income to be equal to 2.5 and this leads to a yearly impatient loss equal to 0.625 and a yearly outpatient loss equal to 0.086. The Bernouilly utility function is given by $u(z) = log(z)$. In Table 4 we report the coverage rates $(c_k^{ij}/\ell_k, k = i, o; ij \in T)$ in percentage for several configuration of the probability of needing services in each dimension. All the probability configurations have the following in common: $w = 2.5$, $\ell_i = 0.625$, $\ell_o = 0.086$; as well as $p_i^H = 0.4$, $p_o^H = 0.3$, $p_o^L = 0.2$. We therefore vary p_i^L only, in order to illustrate the message in

Figure 7: Realistic examples where reversion HL-LL occurs



Notes: Males 35 - 39 years old; w = 2.5 impatient loss = 0.625; outpatient loss = 0.086. $u(z)$ = log z; pHi =0:4, pHo = 0:3, pLo = 0.2.

proposition 2 and the Corollary. In Figure 7, we plot the coverage rates in the outpatient dimension for types LL and HL. The figure shows that for values of p_i^L above 0.3 there is coverage reversion.

Table 4: Numerical examples of coverage reversion between ypes HL and LL

Types HL and LL outpatient coverages rates		
	as p_i^L **approaches** p_i^H	
	c_o^{HL}/ℓ_o	c_o^{LL}/ℓ_o
p_i^L	%	%
0.1	3.795	3.61024
0.2	3.795	3.78031
0.3	3.795	3.93807
0.39	3.795	3.91389
0.395	3.795	3.87155
0.399	3.795	3.81628
0.3999	3.795	3.79754
0.39999	3.795	3.79527
0.399999	3.795	3.79503

6 Empirical Methodology

In this Section we show several approaches to look for evidence on adverse and advantageous selection in the data. We start by showing results from uni-dimensional approaches (Section 6.1) where we study each dimension, i.e. inpatient and outpatient, separately to move to two-dimensional approaches (Section 6.2). In Section 6.1 we show parsymonious regressions of coverage levels on risk variables that either take the form of continuous variables or the form of dummy variables associated to individuals' types. This simple approach is also useful to look for evidence on coverage reversion. The Section ends with the results from applying the PCT (Chiappori and Salanié, 2000) for each dimension, i.e. inpatient and outpatient, separately. Finally, in Section 6.2 we use a non-parametrical approach to study the two dimensions together.

6.1 One-dimensional Approaches or Partial AI tests

Simple tests of adverse/advantageous selection and coverage reversion

We start this Section by showing the results of the simplest regressions to look for evidence of adverse or advantageous selection, and coverage reversion by gender-age cell. We regress the contracted coverages of individual n of gender-age cell k for inpatient and outpatient, respectively, cov_{nki}, and cov_{nko}, against the individual's number of hospitalizations in 2007 ($hosp$) and the number of visits in 2007 ($visits$):

$$\begin{cases} cov_{nki} = c_{1ki} + \alpha_{1ki} hosp_n + \varepsilon_{nki} \\ cov_{nko} = c_{2ko} + \alpha_{2ko} visits_n + \varepsilon_{nko} \end{cases} \tag{18}$$

Adverse selection in each dimension implies $\hat{\alpha}_1, \hat{\alpha}_2 > 0$. The occurence of moral hazard, particularly in the outpatient equation, should reinforce the adverse selection effect by biasing upward $\hat{\alpha}_2$. Table 5 shows the estimates of $\hat{\alpha}_1, \hat{\alpha}_2$. There is substantial evidence of adverse selection in inpatient for all age groups and in outpatient for older groups. We also find substantial evidence of advantageous selection for relative young females.

Because Isapres are so heterogenous, we rerun the regressions (18) also by isapre-gender-age

Table 5: First Evidence of Adverse and Advantageous Selection by Gender-Age Cell

	By Gender-Age Cell: Evidence of Adverse and Advantageous Selection			
	Inpatient ($\widehat{\alpha}_1$)		Outpatient ($\widehat{\alpha}_2$)	
	Female	Male	Female	Male
Age group	(1)	(2)	(3)	(4)
1	1.530^{***}	0.051	-0.038^{***}	-0.006
	(0.195)	(0.202)	(0.011)	(0.019)
2	0.651^{***}	1.037^{***}	-0.073^{***}	0.067^{***}
	(0.153)	(0.166)	(0.007)	(0.014)
3	0.323^{*}	1.195^{***}	-0.048^{***}	0.024^{**}
	(0.166)	(0.159)	(0.008)	(0.012)
4	0.711^{***}	1.262^{***}	-0.011	0.052^{***}
	(0.196)	(0.157)	(0.009)	(0.012)
5	0.999^{***}	1.062^{***}	0.033^{***}	0.039^{***}
	(0.218)	(0.177)	(0.010)	(0.012)
6	1.556^{***}	1.054^{***}	0.034^{***}	0.050^{***}
	(0.217)	(0.196)	(0.011)	(0.013)
7	1.474^{***}	0.731^{***}	0.037^{***}	0.029^{**}
	(0.260)	(0.233)	(0.011)	(0.013)

Note: Table shows the estimated coefficients $\widehat{\alpha}_1$ and ($\widehat{\alpha}_2$) and their robust standard errors in parenthesis from regressions (18) for inpatient (columns (1) and (2)) and outpatient (columns (3) and (4)) by gender-age cell.

group. We complement the analysis by running an extension to regression (18) that adds as control the risk in the other dimension: $cov_{nki} = c_{1ki} + \alpha_{1ki} hosp_n + \beta_{1ki} visits_n + \varepsilon_{nki}$ and $cov_{nko} = c_{2ko} + \alpha_{2ko} visits_n + \beta_{2ko} hosp_n + \varepsilon_{nko}$. Tables 6 and 7 show the percent of gender-age cells with statistically significant $\hat{\alpha}_1 s$ and $\hat{\alpha}_2 s$ by isapre for the inpatient and outpatient dimension, respectively. Overall, we find some evidence of adverse selection or moral hazard, particularly in outpatient, and close to no evidence of advantageous selection. There are 5 isapres with a large number of significant positive α_2's, evidence of adverse selection or moral hazerd in outpatient, while only 2 isapres (5 and 8) show some evidence of advantageous selection in outpatient. There is also substantial heterogeneity across isapres. For example, Isapre 6 shows strong evidence of adverse selection (or moral hazard) in both dimensions while Isapre 2, as expected, shows none.

We now modifiy the previous empirical model to adapt it further to the theoretical model developed in Section 5. Since the latter is binary, i.e individual types are either H or L in both risk dimensions, we use our dichotomize risk proxies (as described in Section 4.2) to assign individuals to types depending on their usage. Hence, an individual is classified as type HH if he/she has at least one hospitalization in 2007 and has visited the doctor at least as often as the average individual in his/her gender-age cell. We run the following regressions (19) and (22), for inpatient and outpatient, respectively:

$$cov_{nki} = \alpha_{1kHH} HH_n + \alpha_{1kHL} HL_n + \alpha_{1kLH} LH_n + \alpha_{1kLL} LL_n + \varepsilon_{nki} \tag{19}$$

We consider that there is evidence of adverse selection in inpatient in isapre j and cell k if (and advantageous selection if the inequality has the opposite sign):

$$\begin{cases} \hat{\alpha}_{1kHH} > \hat{\alpha}_{1kLH} \; and/or & \hat{\alpha}_{1kHH} > \hat{\alpha}_{1kLL} \\ \hat{\alpha}_{1kHL} > \hat{\alpha}_{1kLH} \; and/or & \hat{\alpha}_{1kHL} > \hat{\alpha}_{1kLL} \end{cases} \tag{20}$$

and there is evidence of coverage reversion of types LH-LL when:

$$\hat{\alpha}_{1kLH} < \hat{\alpha}_{1kLL}. \tag{21}$$

Similarly for outpatient:

Table 6: First Evidence of Adverse and Advantageous Selection for the Inpatient Dimension

Inpatient regression - % of significant coefficients				
	Adverse Selection		Advantageous Selection	
	Simple model $\hat{\alpha}_1 > 0$	Augmented model $\hat{\alpha}_1 > 0$	Simple model $\hat{\alpha}_1 < 0$	Augmented model $\hat{\alpha}_1 < 0$
Isapre	(1) (%)	(2) (%)	(3) (%)	(4) (%)
1	0.00	7.14	7.14	7.14
2	0.00	0.00	0.00	0.00
3	14.3	14.3	7.14	14.3
4	28.6	14.3	0.00	0.00
5	0.00	64.3	28.57	0.00
6	100.0	57.1	0.00	0.00
7	28.6	7.14	0.00	0.00
8	14.3	7.14	14.29	7.14

Note: In Column (1)-(2) we show the percent of times the OLS estimates of $\hat{\alpha}_1$ of equation (18) are statistically significantly positive in the simple and augmented models, respectively. The augmented model adds the outpatient risk in the form of number of visits, as a regressor to the inpatient equation. Columns (3)-(4) show the percentage of times that the same $\hat{\alpha}_1$s *are statistically significantly negative in the model of equation (18) and in the augmented model, respectively. The total number of tests performed by isapre in each pair of columns (1)-(3) and /2)-(4) is 14.*

Table 7: First Evidence of Adverse and Advantageous Selection for the Outpatient Dimension

Outpatient regression - % of significant coefficients				
	Adverse Selection		Advantageous Selection	
Isapre	Simple model $\hat{\alpha}_2 > 0$ (2) (%)	Augmented model $\hat{\alpha}_2 > 0$ (4) (%)	Simple model $\hat{\alpha}_2 < 0$ (6) (%)	Augmented model $\hat{\alpha}_2 < 0$ (8) (%)
1	3.57	7.14	0.00	0.00
2	0.00	0.00	0.00	0.00
3	46.4	78.46	0.00	0.00
4	57.1	92.9	0.00	0.00
5	0.00	0.00	92.86	100.0
6	89.3	78.6	7.14	7.14
7	50.0	71.4	0.00	0.00
8	21.4	28.6	35.71	28.6

Note: In Column (1)-(2) we show the percent of times the OLS estimates of $\hat{\alpha}_2$ of equation (18) are statistically significantly positive in the simple and augmented models, respectively. The augmented model adds the the inpatient risk, in the form of number of hospitalizations, as a regressor to the outpatient equation. Columns (3)-(4) show the percentage of times that the same $\hat{\alpha}_2$s *are statistically significantly negative in the model of equation (18) and in the augmented model, respectively. The total number of tests performed by isapre in each pair of columns (1)-(3) and /2)-(4) is 14.*

$$cov_{nko} = \alpha_{2kHH} HH_n + \alpha_{2kHL} HL_n + \alpha_{2kLH} LH_n + \alpha_{2kLL} LL_n + \varepsilon_{nko} \tag{22}$$

adverse selection is observed in outpatient in isapre j and cell k if (and advantageous selection if the inequality has the opposite sign):

$$\begin{cases} \hat{\alpha}_{2kHH} > \hat{\alpha}_{2kHL} \; and/or & \hat{\alpha}_{2kHH} > \hat{\alpha}_{2kLL} \\ \hat{\alpha}_{2kLH} > \hat{\alpha}_{2kHL} \; and/or & \hat{\alpha}_{2kLH} > \hat{\alpha}_{2kLL} \end{cases} \tag{23}$$

and coverage reversion of types HL-LL is observed if:

$$\hat{\alpha}_{2kHL} < \hat{\alpha}_{2kLL} \tag{24}$$

Both regressions may lead to biased coefficients if our assignment of individuals to risk types is systematically wrong. Moreover, results may be biased due to the possibility of moral hazard, particularly for outpatient. Hence these results tend to be suggestive rather than proof of the presence of adverse or advantageous selection.

Table (8) summarizes the estimations of (19) and (22) for inpatient and outpatient, respectively. There are differences across the two dimensions with inpatient showing strong evidence in favor of adverse selection and none in favor of advantageous selection, while outpatient shows roughly the same amount of evidence in favor of adverse and advantageous selection. The latter is particularly surprising due to the possibility of moral hazard. Moral hazard would lead us to classify more individuals as high risk in outpatient. Hence, if moral hazard is higher for low coverages than for higher coverages it is conceivable that we may actually bias the coefficients down lpossibly offsetting adverse selection.

Tables 9 and 10 show the tests of equations (20) and (23) at the level of isapre-gender-age cell for the inpatient and outpatient dimensions, respectively. Results are very similar to the continuous versions shown above i.e. overall there is more evidence of adverse selection than of advantageous selection: 5 isapres show evidence of adverse selection in inpatient and 5 in outpatient while only two isapres show evidence of advantageous selection in outpatient. As expected Isapres 1 and 2 shows no evidence of adverse nor advantageous selection due to the lack of diversity of contracts as seen

Table 8: Second Evidence of Adverse and Advantageous Selection by Gender-Age Cell

By Gender-Age Cell: Number of times we cannot reject Adverse and Advantageous Selection

	Adverse Selection Tests						Advantageous Selection					
	Inpatient			Outpatient			Inpatient			Outpatient		
	Female	Male	Total	Female	Male	Total	Female	Male	Total	Female	Male	Total
Age group	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	(N)	(N)	(%)	(N)	(N)	(%)	(N)	(N)	(%)	(N)	(N)	(%)
1	4	2	75.0%	1	0	12.5%	0	0	0.0%	1	1	25.0%
2	4	3	87.5%	0	2	25.0%	0	0	0.0%	2	1	37.5%
3	2	4	75.0%	0	2	25.0%	0	0	0.0%	3	1	50.0%
4	4	4	100.0%	0	1	12.5%	0	0	0.0%	2	2	50.0%
5	4	4	100.0%	2	2	50.0%	0	0	0.0%	0	1	12.5%
6	4	3	87.5%	1	2	37.5%	0	0	0.0%	0	1	12.5%
7	4	3	87.5%	2	0	25.0%	0	0	0.0%	1	0	12.5%

Note: Results depend on the particular classification of individuals into types. In this table a high inpatient risk is an individual with at least one hospitalization and a high outpatient risk is an individual with at least as many visits to the doctor as the mean of the gender-age cell to which he/she belongs. In Column (1) we show the count of no-rejections of the inequalities $\hat{\alpha}_{1jkHH} > \hat{\alpha}_{1jkLH}$, $\hat{\alpha}_{1jkHH} > \hat{\alpha}_{1jkLL}$, $\hat{\alpha}_{1jkHL} > \hat{\alpha}_{2jkLH}$ *and* $\hat{\alpha}_{1jkHL} > \hat{\alpha}_{1jkLL}$ *when regression (20) is restricted to females for the different age -groups. In column (2) we show the same as in column (1) when restricting the regressions to males. Column (3) is the sum of (1)+(2). Columns (4)-(6) are the same for the outpatient dimension i.e. they count the number of no-rejections of inequalities* $\hat{\alpha}_{2jkHH} > \hat{\alpha}_{2jkHL}$, $\hat{\alpha}_{2jkHH} > \hat{\alpha}_{2jkLL}$,$\hat{\alpha}_{2jkLH} > \hat{\alpha}_{2jkHL}$ *and* $\hat{\alpha}_{2jkLH} > \hat{\alpha}_{2jkLL}$ *from estimates of equation (20). We perform a total of 8 tests by gender-age cell for inpatient and service dimension. In columns (7)-(12) we show the number of times the opposite inequalities to (1)-(6) could not be rejected.*

in Figure 2. And, as in Table 6, Isapre 6 is the one that presents the strongest evidence in favor of adverse selection in both dimensions. Most Isapres, with the exception of Isapre 5 and to a less extent 8, show no evidence in favor of advantageous Selection. For each Isapre, we run a total of 56 tests per dimension: 2 genders, 7 age groups, 4 parameter tests per gender-age-dimension.

Table 11 shows the number of times we cannot reject the two different types of coverage reversions by isapre and gender (equations 21 and 24). There is hardly any evidence of coverage reversions. The only exception is the case ofIsapre 8 where we cannot reject that there is coverage reversion of the type HL-LL in up to 4 (6) age groups for females (males). Unfortunately, this is the type of reversions where we are less confident in our test due to potential bias induced by moral hazard bias. We cannot find any evidence of reversions of the type LH-LL.

Extension of the PCT to each dimension

In this Section, we show a straightforward application of the PCT applied separately to each dimension. Chiappori and Salanié (2000) propose the following reduced form model. Consider a latent variable reflecting insurance coverage (cov^*_{nj}) of insurance holder n in firm j and a latent variable denoting insurance holder's n risk (r^*_{nj}).[25]:

$$\begin{cases} cov^*_{nj} = X_{1nj}\theta_{1j} + \varepsilon_{1nj} \\ r^*_{nj} = X_{1nj}\theta_{2j} + \varepsilon_{2nj} \end{cases} \tag{25}$$

where $\begin{pmatrix} \varepsilon_{1nj} \\ \varepsilon_{2nj} \end{pmatrix} \sim N(0, \Sigma)$ with correlation coefficient ρ, where X_{1nj} denotes all variables observed by the insurer j that are used for pricing purposes. Note that cov^* and r^* reflect coverage and risk for either the inpatient or the outpatient dimension. A positive ρ is indicative of the existence of adverse selection as unobservables that increase the probability to buy higher coverage also lead to higher risk. A negative ρ is indicative of advantageous selection. Equation (25) differs from our previous regressions because there are no endogeneous regressors. Nonetheless, were moral hazard to exist, it would still bias ρ upward. This shortcoming is less likely in inpatient than in outpatient. To account for multidimensional asymmetric information, we extend the model to include a set of *unused variables* in underwriting X_2 as potential sources of adverse or advantageous selection (Finkelstein

[25]Some papers correctly point out that expected risk $E(r^*|Z)$ rather than realized risk is the relevant variable to enter the insurance status equation.

Table 9: Second Evidence of Adverse and Advantageous Selection for the Inpatient Dimension

Inpatient Regression - Number of times we cannot reject Adverse and Advantageous Selection

	Adverse Selection Tests				Advantageous Selection			
	(HH vs LH/LL)	(HL vs LH/LL)	Total	% AdveS	(HH vs LH/LL)	(HL vs LH/LL)	Total	% AdvaS
	(N)	(N)	(N)	(%)	(N)	(N)	(N)	(%)
Isapre	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
1	0	0	0	0.0%	2	0	2	3.6%
2	0	0	0	0.0%	0	0	0	0.0%
3	3	4	7	12.5%	2	2	4	7.1%
4	7	2	9	16.1%	0	0	0	0.0%
5	5	10	15	26.8%	5	0	5	8.9%
6	21	7	28	50.0%	0	1	1	1.8%
7	7	0	7	12.5%	0	0	0	0.0%
8	1	0	1	1.8%	2	3	5	8.9%

Note: Results depend on the particular classification of individuals into types. In this table a high inpatient risk is an individual with at least one hospitalization and a high outpatient risk is an individual with at least as many visits to the doctor as the mean of the gender-age cell to which he/she belongs. In Column (1) we show the number of times the inequalities $\hat{\alpha}_{1jkHH} > \hat{\alpha}_{1jkLH}$ *and* $\hat{\alpha}_{1jkHH} > \hat{\alpha}_{1jkLL}$ *from estimates of equation (20) cannot be rejected. In column (2) we show the number of times the inequalities* $\hat{\alpha}_{1jkHL} > \hat{\alpha}_{1jkLH}$ *and* $\hat{\alpha}_{1jkHL} > \hat{\alpha}_{1jkLL}$ *from estimates of equation (20) cannot be rejected.. Column (3) gives the sum of (1)+(2) and column (4) gives the percent the total in column (3) represent in the total of 56 tests. In columns (5)-(6) we show the number of times the opposite inequalities to (1)-(2) could not be rejected. Column (7) is the sum of (5) and (6) and column 8 shows the total of column (7) as a percent of the 56 tests.*

Table 10: Second Evidence of Adverse and Advantageous Selection for the Outpatient Dimension

Outpatient Regression - Number of times we cannot reject Adverse and Advantageous Selection

	Adverse Selection Tests				Advantageous Selection			
	(HH vs HL/LL)	(LH vs HL/LL)	Total	% AdveS	(HH vs HL/LL)	(LH vs HL/LL)	Total	% AdvaS
	(N)	(N)	(N)	(%)	(N)	(N)	(N)	(%)
Isapre	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
1	2	1	3	5.4%	0	0	0	0.0%
2	0	0	0	0.0%	0	0	0	0.0%
3	5	14	19	33.9%	2	2	4	7.1%
4	4	13	17	30.4%	3	1	4	7.1%
5	0	0	0	0.0%	16	22	38	67.9%
6	9	10	19	33.9%	2	2	4	7.1%
7	7	7	14	25.0%	1	0	1	1.8%
8	10	11	21	37.5%	3	6	9	16.1%

Note: Results depend on the particular classification of individuals into types. In this table a high inpatient risk is an individual with at least one hospitalization and a high outpatient risk is an individual with at least as many visits to the doctor as the mean of the gender-age cell to which he/she belongs. In Column (1) we show the number of times the inequalities $\hat{\alpha}_{2jkHH} > \hat{\alpha}_{2jkHL}$ *and* $\hat{\alpha}_{2jkHH} > \hat{\alpha}_{2jkLL}$ *from estimates of equation (20) cannot be rejected. In column (2) we show the number of times the inequalities* $\hat{\alpha}_{2jkLH} > \hat{\alpha}_{2jkHL}$ *and* $\hat{\alpha}_{2jkLH} > \hat{\alpha}_{2jkLL}$ *from estimates of equation (20) cannot be rejected.. Column (3) gives the sum of (1)+(2) and column (4) gives the percent the total in column (3) represent in the total of 56 tests. In columns (5)-(6) we show the number of times the opposite inequalities to (1)-(2) could not be rejected. Column (7) is the sum of (5) and (6) and column 8 shows the total of column (7) as a percent of the 56 tests.*

Table 11: Evidence of Coverage Reversions by Isapre

Number of coverage reversions						
	reversion LH-LL			reversion HL-LL		
	Inpatient Model			Outpatient Model		
	Females	Males	Total	Females	Males	Total
Isapre	(1)	(2)	(3)	(4)	(5)	(6)
1	0	0	0	0	0	0
2	0	0	0	0	0	0
3	0	0	0	0	0	0
4	0	0	0	**1**	0	1
5	0	0	0	0	0	0
6	0	0	0	0	0	0
7	0	0	0	0	**1**	**1**
8	0	0	0	**4**	**6**	**10**

Note: Results depend on the particular classification of individuals into types. In this table a high inpatient risk is an individual with at least one hospitalization and a high outpatient risk is an individual with at least as many visits to the doctor than the mean of the cell to which he/she belongs. Columns (1)-(3) show the results of the tests of equation (21) for female, male and the sum of the two while columns (4)-(6) show the results from tests of equation (24) again for female, male and the sum of the two.

and Poterba, 2014):

$$\begin{cases} cov^*_{nj} = X_{1nj}\gamma_{1j} + X_{2nj}\gamma_{2j} + u_{1nj} \\ r^*_{nj} = X_{1nj}\mu_{1j} + X_{2nj}\mu_{2j} + u_{2nj} \end{cases} \tag{26}$$

In order to estimate the bi-probit models proposed in (25) and (26), we must dichotomize the coverage variables as well as the risk variables for both dimensions. Regarding the latter, we use the dichotomization already used in equations (19) and (22). We discuss the former next.

Discretizing the coverage variable

We define a cutoff above (below) which the coverage is considered high (low), similarly to what we did in Section 4.3. The way the coverage cutoffs are defined, however, is not innocuous. For example, if by high outpatient coverage we define only those contracts which cover 100% of outpatient care, then even those individuals with high risk of outpatient usage would be unlikely to choose them. Hence, the choice of the cutoffs affects the assignment of types to contracts and, therefore, the conclusion of whether or not there is evidence of adverse or advantageous selection in the data.

There are no guidelines with respect to what is considered a high coverage contract. One possibility is to follow R&S literally and consider high coverage contracts only those with full coverage (i.e. 100% coverage). While this option may be reasonable for inpatient coverage it may be extreme for the outpatient coverage (see Figure 1). We, therefore, opted to use a number of different cutoffs, three for inpatient coverage (90, 95, and 100%) and four for the outpatient coverage (80, 90, 95, and 100%), and compare our results under these different cutoff definitions.

Results of the PCT

The estimates of the correlation coefficient ρ between the error terms for the inpatient and outpatient models are reported in Table 12 (Panels A and B for inpatient and outpatient dimension, respectively). Our baseline specification in column (1) considers that X_1 in equation (26) is a vector of individual characteristics over which ISAPREs are allowed, by law, to price discriminate. Since we do not observe the loading factors directly, column 1's specification includes dummies for all age group categories used by Isapres to adjust the price of the plans (7 categories);[26] a gender indicator; and

[26] Age group categories for pricing: [25-29], [30-34], [35-39], [40-44], [45-49], [50-54], [55-59].

interactions between gender and age groups. Additionally, we include as control variable Isapre fixed effects. Controlling for fixed effects is important for at least two reasons. First, because the relevant asymmetry of information is conditional on the observable characteristics of the beneficiaries within a given Isapre (e.g. Rothschild and Stiglitz, 1976). Second, in order to make sure that a positive risk-coverage correlation would not simply reflect specialization across Isapres. The latter would be the case if Isapres engage in horizontal differentiation by focusing on different risk-segments of the market whereby those Isapres with relatively higher risk clientele offer more generous coverage. In columns (2) and (3) we include other variables X_2 which are observable by Isapres but cannot be used in their pricing scheme: income and region of residence dummies. Although individual income and region are characteristics that cannot be legally used for pricing plans, they may be a source of adverse selection or, on the contrary, act as a source of advantageous selection either because the Isapres use them to select the menu of plans offered to clients or because income affects individual preference for plans.

Panel A of Table 12 shows the results of the positive correlation test for the inpatient dimension when the coverage dummy takes the value 1 if inpatient care coverage of the plan is 90% or more and the risk variable is at least one hospitalization in 2007. We find that the estimated correlation coefficient ρ is 0.084 and it is statistically different from zero (column 1). This positive correlation is consistent with the presence of information asymmetries between policyholders and their Insurer. Hospitalizations are tipically assumed to be free of (patients) moral hazard (Manning et al., 1987; Chiappori et al, 1998; Sapelli and Vial, 2003; Gardiol et al., 2005; Olivella and Vera-Hernández, 2013, among others), consequently, the finding of a positive risk-coverage correlation constitutes strong evidence in favor of adverse selection. Including income or region fixed effects (columns (2) and (3)) only slightly reduces the magnitude of the estimated correlation coefficient which remain statistically significant. If anything, income and region of residence are acting as additional sources of adverse selection since the estimated correlation coefficients are lower in magnitude once controlling for these variables. The evidence of adverse selection, vanishes completely once we increasing the coverage cutoff to 95 or 100%.

Panel B of Table 12 shows the results of the positive correlation test for the outpatient dimension when the coverage dummy takes the value 1 if outpatient care coverage of the plan is 80% or more and the risk variable is at least 5 visits to the doctor (upper panel) and the risk variable is at

least as many visits as the average of the cell (lower panel). This measure of risk is likely subject to moral hazard and, therefore, the finding of a positive risk-coverage correlation would not imply necessarily the presence ,of adverse selection while a negative or zero correlation would indicate the presence of advantageous selection. The estimated $\rho's$ for the 80% coverage cutoffs are either negative or very low and not statistically different from zero, which constitutes evidence in favor of advantageous selection, specially considering they may be biased upwards. For 90 and 95% coverage cutoffs, however, the estimated $\rho's$ increase substantially, although, with the exception of those in column (2), they remain not statistically significant.

Table 12: Correlation estimates bivariate probit model

Panel A: Inpatient Model									
	Inpatient full coverage definition								
	90%			95%			100%		
Risk definition	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
At least one hospitalization									
$\hat{\rho}$	0.084	0.077	0.082	0.002	0.014	0.000	0.003	0.015	0.001
p-val of the LR test of $\rho = 0$	0.000	0.000	0.000	0.915	0.226	0.984	0.852	0.176	0.920

Panel B: Outpatient Model												
	Outpatient full coverage definition											
	80%			90%			95%			100%		
Risk definition	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)	(1)	(2)	(3)
5 or more visits to MD												
$\hat{\rho}$	-0.014	0.008	-0.018	0.052	0.076	0.042	0.055	0.079	0.046	-0.017	0.006	-0.021
p-val of the LR test of $\rho = 0$	0.619	0.711	0.536	0.194	0.012	0.278	0.194	0.015	0.265	0.559	0.773	0.479
equal or more visits than mean number of visits to MD in cell												
$\hat{\rho}$	-0.034	-0.005	-0.037	0.03	0.061	0.019	0.033	0.064	0.024	-0.036	-0.007	-0.04
p-val of the LR test of $\rho = 0$	0.291	0.828	0.241	0.467	0.037	0.619	0.433	0.038	0.564	0.256	0.769	0.211
Age (14 categories), gender, age*gender	x	x	x	x	x	x	x	x	x	x	x	x
Isapre FE	x	x	x	x	x	x	x	x	x	x	x	x
Income (logs) + dummy income missing		x			x			x			x	
Region FE			x			x			x			x

Notes: This table gives the results on the correlation ρ between the residuals of a two equation model of risk and coverage assuming that the error terms are distributed according to a bi-variate normal. Robust standard errors clustered at isapre-region level. In all models the sample size is 588,678.

Note that previous research by Sappeli and Vial (2003) did not find evidence of adverse selection for dependent workers in private insurance in Chile in either dimension.

Aggregate Uni-dimensional Tests- To be done

6.2 Non-parametrical Approach

Given a set of cutoffs and upon the dichotomization, our data is classified into a 4×4 matrix of 4 types $\{HH, HL, LH, LL\}$ by 4 contract coverages $\{hh, hl, lh, hh\}$ for each gender-age cell. With such a matrix configuration, independence of risk and coverage would lead to a measure of 6.25% observations in each matrix-cell if the two densities were uniformly distributed.[27] A configuration where most observations are in the diagonal—which should account for 25% of the observations under uniformity and independence—as illustrated by the "As" in Figure 8, would be consistent with adverse selection in both outpatient and inpatient dimensions. A configuration where most observations are in the block diagonal, i.e the As and Bs in Figure 8 —which should account for 50% of the observations under uniformity and independence—would be consistent with adverse selection in inpatient only while no conclusion could be drawn from the behavior regarding the outpatient dimension. A configuration corresponding to the As and the Cs in Figure 9 would reveal the existence of adverse selection in outpatient only. It is straightforward to work out the scenarios with advantageous selection in only one or in both dimensions (e.g. the latter would be produced by observations mainly in the opposite diagonal i.e. the one with positive slope).

Figure 8:

	types			
contracts	HH	HL	LH	LL
hh	A	B		
hl	B	A		
lh			A	B
ll			B	A

Running an extension of the chi-2 independence test used in Chiappori and Salanié (2000) we cannot reject the null hypothesis that the two variables risk and coverage are independent in most cells.[28] The independence test, however, is mute with respect to the alternative.

[27] Note that uniformity does not occur as we know that types that are H risk in inpatient are only around 10% of the observations. Hence, the 6.25% figure is only for reference.

[28] The Chiappori and Salanié (2000) text has a typo in the formula of the test. We use the correct formula.

Figure 9:

	types			
contracts	HH	HL	LH	LL
hh	A		C	
hl		A		C
lh	C		A	
ll		C		A

We start by defining a pair of cutoffs for the inpatient and outpatient coverages, respectively. For a given pair of cutoffs we compute the percent of observations per isapre and gender that are consistent with the following situations: 1) Adverse selection in both dimensions i.e most observations are in the diagonal (as the As in Figure 8); 2) Advantageous selection in both dimensions i.e most observations are in the opposite diagonal; 3) Full information in either dimension (i.e. 100% coverage). For this first round of computations, we sum the observations in 4 out of the 16 cells. In a second round of computations, we sum the observations in 8 out of the 16 cells to compute the percent of observations per isapre and gender that are consistent with the following situations: 1) Adverse selection in inpatient only i.e. most observations are in the block diagonal (as the As and Bs in Figure 8) and in outpatient only (as the As and Cs in Figure 9); 2) Advantageous selection in one dimension either inpatient or outpatient; Full information in inpatient or outpatient.

We repeat the two exercises for several reasonable coverage cutoffs and summarize the result in Table 13. While most results for most Isapres are relatively robust to the set of cutoffs, Isapres 1, 4 and 8 show very different results depending on the cutoffs chosen. Isapres 3, 5, 6 and 7 show that adverse selection in both dimensions is the most probable result. Isapre 2, shows evidence of full information, which is due to the lack of variability in the plans offered, which can be confirmed in Figure 2. Surprisingly, we find a couple of Isapres (1 and 4) where advantageous selection in inpatient is obtained.

Table 13:

Most Likely Scenario by Isapre and coverage cutoff								
	Isapres							
	1	2	3	4	5	6	7	8
Cutoffs								
≥90% inpatient; ≥80% outpatient	FI, FI	FI, FI-i	PS-AS, FI-i	FI, FI-i	PS-AS, FI-i	AS-AS, AS-i	PS-AS, FI-i	FI, FI-i
≥95% inpatient; ≥80% outpatient	PS-AS, FI-o	FI, FI	AS-AS, AS-i	PS-AS, FI-o	AS-AS, AS-i	AS-AS, AS-i	AS-AS, AS-i	FI, AS-i
≥100% inpatient; ≥80% outpatient	PS-AS, FI-o	FI, FI	AS-AS, AS-i	PS-AS, FI-o	AS-AS, AS-i	AS-AS, AS-i	AS-AS, AS-i	FI, AS-i
≥95% inpatient; ≥90% outpatient	PS-AS, FI-o	FI, FI-i	AS-AS, AS-i	PS-AS, FI-i	AS-AS, AS-i	AS-AS, AS-i	AS-AS, AS-i	AS-AS, AS-i
≥100% inpatient; ≥90% outpatient	PS-AS, FI-i	FI, FI-i	AS-AS, AS-i	PS-AS, FI-i	AS-AS, AS-i	AS-AS, AS-i	AS-AS, AS-i	AS-AS, AS-i

Note: FI stands for Full-Information i.e. most observations are in high coverage of both inpatient and outpatient dimensions; FI-i(o) stands for Full information only in the inpatient (outpatient) dimension. AS-AS means evidence of adverse selection in both inpatient and outpatient (i.e. most observations in the diagonal); AS-PS means evidence of adverse selection in inpatient and propitious (or advantageous) selection in outpatient. PS-PS means evidence of advantageous selection in both inpatient and outpatient (i.e. most observations are in the opposite diagonal). AS-i means evidence of adverse selection only in inpatient (i.e. most observations are in the block diagonal such as in matrix 8). AS-o means evidence of adverse selection only in outpatient (i.e. most observations are as in matrix9)

7 Conclusion

To be completed.

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Appendix A: Proofs

Proof of Lemma 1:

Step 1: The RHS is increasing in $c_o^{HL} < \ell_o$:

$$\frac{\partial\left((1-p_o^H)u(w_i{}' - p_o^L c_o^{HL}) + p_o^H u(w_i{}' - \ell_o + c_o^{HL}(1-p_o^L))\right)}{\partial c_o^{HL}} =$$
$$(1-p_o^H)u{}'(w_i{}' - p_o^L c_o^{HL})(-p_o^L) + p_o^H u{}'(w_i{}' - p_o^L c_o^{HL} + c_o^{HL} - \ell_o)(1-p_o^L) >$$
$$(1-p_o^H)u{}'(w_i{}' - p_o^L c_o^{HL})(-p_o^L) + p_o^H u{}'(w_i{}' - p_o^L c_o^{HL})(1-p_o^L) =$$
$$\left[(1-p_o^H)(-p_o^L) + p_o^H(1-p_o^L)\right] u{}'(w_i{}' - p_o^L c_o^{HL}) =$$
$$\left[p_o^H - p_o^L\right] u{}'(w_i{}' - p_o^L c_o^{HL}) > 0$$

Step 2: Suppose $c_o^{HL} = \ell_o$. Then the RHS becomes:

$$(1-p_o^H)u(w_i{}' - p_o^L \ell_o) + p_o^H u(w_i{}' - \ell_o + \ell_o(1-p_o^L)) =$$
$$(1-p_o^H)u(w_i{}' - p_o^L \ell_o) + p_o^H u(w_i{}' + \ell_o(-p_o^L)) = u(w_i{}' - p_o^L \ell_o)$$

This is larger than the LHS $u(w_i{}' - p_o^H \ell_o)$, since $p_o^L < p_o^H$. Therefore the ICC is violated.

Step 3: Now, if $c_o^{HL} = 0$ then, using Jensen's inequality we can write:

$$(1-p_o^H)u(w_i{}' - p_o^L c_o^{HL}) + p_o^H u(w_i{}' - \ell_o + c_o^{HL}(1-p_o^L)) =$$
$$(1-p_o^H)u(w_i{}') + p_o^H u(w_i{}' - \ell_o) < u[(1-p_o^H)w_i{}' + p_o^H(w_i{}' - \ell_o)] =$$
$$u(w_i{}' - p_o^H \ell_o)$$

Step4: By continuity, there must be some c_o^{HL*} at which the ICC is binding.

Proof of Lemma 2: The proof is symmetric to the one given for Lemma 1. Replace c_o^{HL} by c_i^{LH}, ℓ_o by ℓ_i, p_o^L by p_i^L, and $w_i{}' = w - p_i^H \ell_i$ by $w_o{}' = w - p_o^H \ell_o$.

Proof of Proposition 1:

We show the first statement. The other follows by symmetry. By a previous result?, c_i^{LH} is fully

determined by:

$$u(w - P^{HH}) = (1 - p_i^H)u(w - P^{LH}) + p_i^H u(w - P^{LH} - \ell_i + c_i^{LH}).$$

Differentiate totally with resect to p_i^L, bearing in mind that $P^{LH} = p_i^L c_i^{LH} + p_o^H \ell_o$

$$0 = (1-p_i^H)u'(w-P^{LH})\left(-c_i^{LH} - p_i^L \frac{\partial c_i^{LH}}{\partial p_i^L}\right) + p_i^H u'(w-P^{LH}-\ell_i+c_i^{LH})\left(-c_i^{LH} - (p_i^L - 1)\frac{\partial c_i^{LH}}{\partial p_i^L}\right)$$

Let $n^{LH} = w - P^{LH}$ and $a^{LH} = w - P^{LH} - \ell_i + c_i^{LH}$. The previous expression can then be rewritten as:

$$\frac{\partial c_i^{LH}}{\partial p_i^L} = \frac{-c_i^{LH}\left((1 - p_i^H)u'(n^{LH}) + p_i^H u'(a^{LH})\right)}{p_i^L(1 - p_i^H)u'(n^{LH}) - (1 - p_i^L)p_i^H u'(a^{LH})}$$

The last expression is positive if and only if $(1 - p_i^H)p_i^L u'(n^{LH}) - (1 - p_i^L)p_i^H u'(a^{LH}) < 0 \Leftrightarrow$ $(1 - p_i^H)p_i^L u'(n^{LH}) < (1 - p_i^L)p_i^H u'(a^{LH})$ or

$$\frac{(1 - p_i^H)p_i^L}{(1 - p_i^L)p_i^H} < \frac{u'(a^{LH})}{u'(n^{LH})},$$

where the LHS is less than one since $(1 - p_i^H) < (1 - p_i^L)$ and $p_i^L < p_i^H$ and the RHS is larger than one since $a^{LH} < n^{LH}$ but u´ is decreasing. **QED**

Proof of Proposition 2

The binding ICC HH→LH, i.e.,

$$Eu(HH, HH) = Eu(HH, LH),$$

can be rewritten as:

$$u(w - P^{HH}) = (1 - p_o^H - p_i^H + p_i^H p_o^H)u(w - P^{LH}) + p_i^H(1 - p_o^H)u(w - P^{LH} - \ell_i + c_i^{LH}) +$$
$$p_o^H(1 - p_i^H)u(w - P^{LH}) + p_i^H p_o^H u(w - P^{LH} - \ell_i + c_i^{LH})$$

The RHS can be rewritten as:

$$(1-p_i^H)u(w-P^{LH})+p_i^H u(w-P^{LH}-\ell_i+c_i^{LH})$$

Now suppose that $p_i^L=p_i^H=p_i$. Then $w-P^{HH}=w-p_i\ell_i-p_o^H\ell_o$ and $w-P^{LH}=w-p_ic_i^{LH}-p_o^H\ell_o$. Hence the ICC becomes:

$$u(w-p_i\ell_i-p_o^H\ell_o)=(1-p_i)u(w-p_ic_i^{LH}-p_o^H\ell_o)+p_iu(w-p_ic_i^{LH}-p_o^H\ell_o-\ell_i+c_i^{LH})$$

Notice that $\frac{\partial RHS}{\partial c_i^{LH}}=(1-p_i)u'(w-p_ic_i^{LH}-p_o^H\ell_o)(-p_i)+p_iu'(w-p_ic_i^{LH}-p_o^H\ell_o-\ell_i+c_i^{LH})(1-p_i)>0$ if and only if $u'(w-p_ic_i^{LH}-p_o^H\ell_o-\ell_i+c_i^{LH})>u'(w-p_ic_i^{LH}-p_o^H\ell_o)$ which is true since u' is decreasing and $c_i^{LH}<\ell_i$.

Hence the RHS reaches a maximum at $c_i^{LH}=\ell_i$. Notice also that, if $c_i^{LH}=\ell_i$, the first term in the RHS becomes $(1-p_i)u(w-p_i\ell_i-p_o^H\ell_o)$, the second term becomes $p_iu(w-p_i\ell_i-p_o^H\ell_o)$, and hence the sum of the two terms coincides with the LHS. Therefore$c_i^{LH}=\ell_i$ is the unique solution for the ICC.

We need to show that the proposed conditions guarantee that the 3 remaining binding ICCs, are satisfied (binding constraint HH→LH has already been used above). Let us analyze the ICC HL→LL in general first. The LHS is given by:

$$Eu(HL,HL)=(1-p_o^L)u(w-P^{HL})+p_o^Lu(w-P^{HL}-\ell_o+c_o^{HL}).$$

The RHS is given by:

$$Eu(HL,LL)=(1-p_o^L-p_i^H+p_i^Hp_o^L)u(w-P^{LL})+p_i^H(1-p_o^L)u(w-P^{LL}-\ell_i+c_i^{LL})+$$
$$p_o^L(1-p_i^H)u(w-P^{LL}-\ell_o+c_o^{LL})+p_i^Hp_o^Lu(w-P^{LL}-\ell_i-\ell_o+c_i^{LL}+c_o^{LL})$$

Evaluate at $p_i^L=p_i^H=p_i$. Then $w-P^{HL}=w-p_i\ell_i-p_o^Lc_o^{HL}$ and $w-P^{LL}=w-p_ic_i^{LL}-p_o^Lc_o^{LL}$.

Hence the ICC HL→LL becomes:

$$(1-p_o^L)u(w-p_i\ell_i-p_o^Lc_o^{HL})+p_o^Lu(w-p_i\ell_i-p_o^Lc_o^{HL}-\ell_o+c_o^{HL})=$$
$$(1-p_o^L-p_i^H+p_i^Hp_o^L)u(w-p_ic_i^{LL}-p_o^Lc_o^{LL})+p_i^H(1-p_o^L)u(w-p_ic_i^{LL}-p_o^Lc_o^{LL}-\ell_i+c_i^{LL})+$$
$$p_o^L(1-p_i^H)u(w-p_ic_i^{LL}-p_o^Lc_o^{LL}-\ell_o+c_o^{LL})+p_i^Hp_o^Lu(w-p_ic_i^{LL}-p_o^Lc_o^{LL}-\ell_i-\ell_o+c_i^{LL}+c_o^{LL})$$

If $c_i^{LL}=\ell_i$ then the LHS stays the same whereas the RHS becomes:

$$RHS=(1-p_o^L-p_i^H+p_i^Hp_o^L)u(w-p_i\ell_i-p_o^Lc_o^{LL})+p_i^H(1-p_o^L)u(w-p_i\ell_i-p_o^Lc_o^{LL})+$$
$$p_o^L(1-p_i^H)u(w-p_i\ell_i-p_o^Lc_o^{LL}-\ell_o+c_o^{LL})+p_i^Hp_o^Lu(w-p_i\ell_i-p_o^Lc_o^{LL}-\ell_o+c_o^{LL})$$

or

$$RHS=(1-p_o^L)u(w-p_i\ell_i-p_o^Lc_o^{LL})+p_o^Lu(w-p_i\ell_i-p_o^Lc_o^{LL}-\ell_o+c_o^{LL})$$

Hence HL→LL becomes:

$$(1-p_o^L)u(w-p_i\ell_i-p_o^Lc_o^{HL})+p_o^Lu(w-p_i\ell_i-p_o^Lc_o^{HL}-\ell_o+c_o^{HL})=$$
$$(1-p_o^L)u(w-p_i\ell_i-p_o^Lc_o^{LL})+p_o^Lu(w-p_i\ell_i-p_o^Lc_o^{LL}-\ell_o+c_o^{LL})$$

which implies that $c_o^{LL}=c_o^{HL}$ is a solution.

We now show that if $p_i^L=p_i^H=p_i$ and $c_i^{LL}=\ell_i$ then the solution for c_o^{HL} in the ICC HH→HL is the same as the solution for c_o^{LL} in the ICC LH→LL.

Let us analyze the ICC LH→LLin general first. The LHS is given by:

$$Eu(LH,LH)=(1-p_i^L)u(w-P^{LH})+p_i^Lu(w-P^{LH}-\ell_i+c_i^{LH}).$$

The RHS is given by:

$$Eu(LH,LL) = (1 - p_o^H - p_i^L + p_i^L p_o^H)u(w - P^{LL}) + p_i^L(1 - p_o^H)u(w - P^{LL} - \ell_i + c_i^{LL}) +$$
$$p_o^H(1 - p_i^L)u(w - P^{LL} - \ell_o + c_o^{LL}) + p_i^L p_o^H u(w - P^{LL} - \ell_i - \ell_o + c_i^{LL} + c_o^{LL})$$

We proved that if $p_i^L = p_i^H = p_i$ then $c_i^{LH} = \ell_i$. Moreover, $w - P^{LH} = w - p_i\ell_i - p_o^H\ell_o$ and $w - P^{LL} = w - p_i c_i^{LL} - p_o^L c_o^{LL}$. Hence the ICC LH→LL becomes:

$$u(w - p_i\ell_i - p_o^H\ell_o) =$$
$$(1 - p_o^H - p_i + p_i p_o^H)u(w - p_i c_i^{LL} - p_o^L c_o^{LL}) +$$
$$+p_i(1 - p_o^H)u(w - p_i c_i^{LL} - p_o^L c_o^{LL} - \ell_i + c_i^{LL}) +$$
$$+p_o^H(1 - p_i)u(w - p_i c_i^{LL} - p_o^L c_o^{LL} - \ell_o + c_o^{LL}) +$$
$$+p_i p_o^H u(w - p_i c_i^{LL} - p_o^L c_o^{LL} - \ell_i - \ell_o + c_i^{LL} + c_o^{LL})$$

If $c_i^{LL} = \ell_i$ then the LHS stays the same whereas the RHS becomes:

$$RHS = (1 - p_o^H - p_i + p_i p_o^H)u(w - p_i\ell_i - p_o^L c_o^{LL}) + p_i(1 - p_o^H)u(w - p_i\ell_i - p_o^L c_o^{LL} - \ell_i + \ell_i) +$$
$$p_o^H(1 - p_i)u(w - p_i\ell_i - p_o^L c_o^{LL} - \ell_o + c_o^{LL}) + p_i p_o^H u(w - p_i\ell_i - p_o^L c_o^{LL} - \ell_i - \ell_o + \ell_i + c_o^{LL})$$

or:

$$RHS = (1 - p_o^H)u(w - p_i\ell_i - p_o^L c_o^{LL}) + p_o^H u(w - p_i\ell_i - p_o^L c_o^{LL} - \ell_o + c_o^{LL})$$

In sum, the ICC LH→LL becomes:

$$u(w - p_i\ell_i - p_o^H\ell_o) = (1 - p_o^H)u(w - p_i\ell_i - p_o^L c_o^{LL}) + p_o^H u(w - p_i\ell_i - p_o^L c_o^{LL} - \ell_o + c_o^{LL}) \qquad (27)$$

Compare this equation with the equation that determines c_o^{HL}, that is, equation (13), for the case when $p_i^H = p_i^L = p_i$, which is given by:

$$u(w - p_i\ell_i - p_o^H\ell_o) = (1 - p_o^H)u(w - p_i\ell_i - p_o^L c_o^{HL}) + p_o^H u(w - p_i\ell_i - p_o^L c_o^{HL} - \ell_o + c_o^{HL}) \qquad (28)$$

Notice that (27), which uniquely determines c_o^{LL}, and (28), which uniquely determines c_o^{HL}, are

the same and therefore yield the same solution. **QED**

Proof of Proposition 3:

Differentiating totally the ICC LH→LL with respect to p_i^L yields:

$$\begin{gathered}(-1)u(n^{LH})+(1-p_i^L)u'(n^{LH})\left(-c_i^{LH}-p_i^L\frac{\partial c_i^{LH}}{\partial p_i^L}\right)+u(a^{LH})+\\+p_i^Lu'(a^{LH})\left(-c_i^{LH}-(p_i^L-1)(\frac{\partial c_i^{LH}}{\partial p_i^L})\right)=\\(p_o^H-1)u(n^{LL})+(1-p_o^H-p_i^L+p_i^Lp_o^H)u'(n^{LL})\left(-c_i^{LL}-p_i^L\frac{\partial c_i^{LL}}{\partial p_i^L}-p_o^L\frac{\partial c_o^{LL}}{\partial p_i^L}\right)+\\+(1-p_o^H)u(a_i^{LL})+p_i^L(1-p_o^H)u'(a_i^{LL})\left(-c_i^{LL}-p_i^L\frac{\partial c_i^{LL}}{\partial p_i^L}-p_o^L\frac{\partial c_o^{LL}}{\partial p_i^L}+\frac{\partial c_i^{LL}}{\partial p_i^L}\right)+\\+p_o^H(-1)u(a_o^{LL})+p_o^H(1-p_i^L)u'(a_o^{LL})\left(-c_i^{LL}-p_i^L\frac{\partial c_i^{LL}}{\partial p_i^L}-p_o^L\frac{\partial c_o^{LL}}{\partial p_i^L}+\frac{\partial c_o^{LL}}{\partial p_i^L}\right)+\\+p_o^Hu(a_b^{LL})+p_i^Lp_o^Hu'(a_b^{LL})\left(-c_i^{LL}-p_i^L\frac{\partial c_i^{LL}}{\partial p_i^L}-p_o^L\frac{\partial c_o^{LL}}{\partial p_i^L}+\frac{\partial c_i^{LL}}{\partial p_i^L}+\frac{\partial c_o^{LL}}{\partial p_i^L}\right)\end{gathered}$$

Evaluate at $p_i^L=p_i^H=p_i$, which leads to the solution $c_i^{LH}=c_i^{LL}=\ell_i$, so $P^{LH}=p_i\ell_i+p_o^H\ell_o$ and $P^{LL}=p_i\ell_i+p_o^Lc_o^{LL}$, and let $n^{LH}=w-P^{LH}=w-p_i\ell_i-p_o^H\ell_o$, $a^{LH}=w-P^{LH}+c_i^{LH}-\ell_i=w-p_i\ell_i-p_o^H\ell_o=n^{LH}$, $n^{LL}=w-p_i\ell_i-p_o^Lc_o^{LL}$. $a_i^{LL}=w-p_i\ell_i-p_o^Lc_o^{LL}-\ell_i+c_i^{LL}=w-p_i\ell_i-p_o^Lc_o^{LL}=n^{LL}$, $a_o^{LL}=w-p_i\ell_i-p_o^Lc_o^{LL}-\ell_o+c_o^{LL}$, $a_b^{LL}=w-p_i\ell_i-p_o^Lc_o^{LL}-\ell_o+c_o^{LL}-\ell_i+c_i^{LL}=w-p_i\ell_i-p_o^Lc_o^{LL}-\ell_o+c_o^{LL}=a_o^{LL}$. Then the differentiation becomes, after quite a bit of algebra.

$$\frac{\ell_i[u'(n^{LL})-u'(n^{LH})]}{[1-p_o^L]p_o^Hu'(a_o^{LL})-[1-p_o^H]p_o^Lu'(n^{LL})}=\left[\frac{\partial c_o^{LL}}{\partial p_i^L}\right]_{p_i^L=p_i^H}$$

which is negative if and only if (since $u'(n^{LL})-u'(n^{LH})<0$)

$$[1-p_o^L]p_o^Hu'(a_o^{LL})-[1-p_o^H]p_o^Lu'(n^{LL})>0$$

if and only if

$$\begin{gathered}[1-p_o^L]p_o^Hu'(a_o^{LL})>[1-p_o^H]p_o^Lu'(n^{LL})\\\frac{[1-p_o^L]p_o^H}{[1-p_o^H]p_o^L}>\frac{u'(n^{LL})}{u'(a_o^{LL}}\end{gathered}$$

We know that RHS <1, whereas $p_o^H>p_o^L$ implies $\frac{[1-p_o^L]p_o^H}{[1-p_o^H]p_o^L}>1$. Therefore, the condition is always satisfied. To conclude, $[\frac{\partial c_o^{LL}}{\partial p_i^L}]_{p_i^L=p_i^H}<0$. **QED**

Appendix B: Figures and Tables

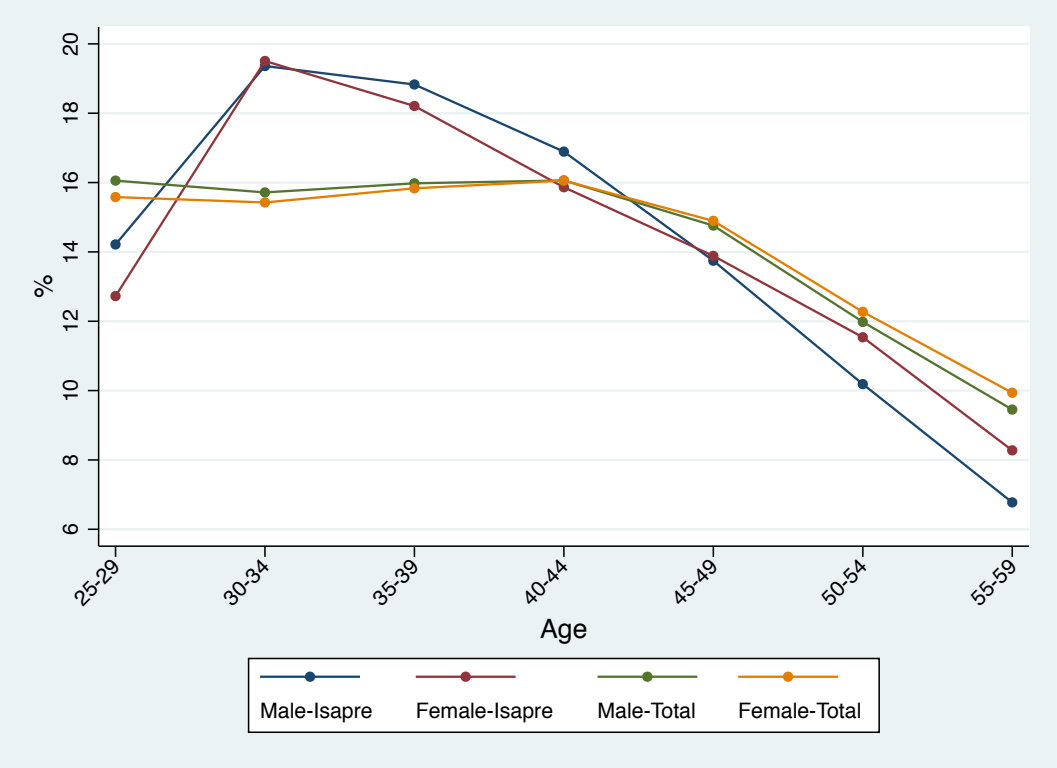

Figure 10: Distribution of beneficiaries vs distribution of Chilean population