The Impact of Disease-Specific Healthcare Reform on Mortality*

Felipe Menares
UC Berkeley

Pablo Muñoz
U. de Chile

(Job Market Paper)

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Abstract

We study the impact of a healthcare reform that guaranteed universal access to care for a specific set of diseases. Using the universe of death and inpatient records from Chile and a difference-in-differences research design, we show that deaths from the diseases covered by this reform decreased by 4.4%. The impact was larger for diseases that are amenable to health care, which decreased by 7.1%. Additionally, the reform led to a 16.3% increase in surgeries and a 6.9% decrease in in-hospital deaths. Longitudinal survey data and back-of-the-envelope calculations suggest that the reform also resulted in more medical visits and reduced out-of-pocket expenses for beneficiaries, ultimately increasing life expectancy by 0.29 years.

*Corresponding author: Felipe Menares, University of California at Berkeley, Department of Demography. Email: fmenares@berkeley.edu. We are particularly indebted to William H. Dow, Ronald D. Lee, and Joshua R. Goldstein for their advice and support at different stages of this project. We want to thank Tatiana Reyes, Daniela Reyes, Sofía Jordan, Andrea Miranda-Gonzalez, Julio Rodriguez, Joel Ferguson, Matias Morales, and Mathieu Pedemonte, and the Berkeley Demography Student Workshop for valuable comments and suggestions. We gratefully acknowledge financial support from The John L. Simpson ABD Graduate Students Research Fellowships in Global, International and Area Studies (GIAS), Graduate Division, UC Berkeley. All remaining errors are our own. Pablo Muñoz, Departamento de Ingeniería Industrial, U. de Chile. Email: pablomh@uchile.cl.

1 Introduction

Recent studies on healthcare reforms suggest non-negative mortality effects resulting from expansions in insurance coverage (Sommers, 2017; Goldin et al., 2020; Borgschulte and Vogler, 2020; Miller et al., 2021). Most of the existing research, however, has focused on the impact of expanding coverage to uninsured populations and little is known about the effects of guaranteeing timely coverage for specific *diseases*. Methodological constraints and lack of data have limited research on the impact of alternative health reforms (Levy and Meltzer, 2008; Moreno-Serra and Smith, 2012; Gruber and Sommers, 2019; Black et al., 2019). In particular, a key limitation is the absence of quasi-experimental variation in healthcare access that is *independent* of insurance type. We overcome this hurdle by studying Chile's most significant healthcare reform in the past 30 years: the Explicit Healthcare Guarantees program (known as "GES" for its name in Spanish).

Recognizing that coverage can differ depending on specific healthcare provisions, Congress approved a package of bills between 2002-2004 that established regulations and specific rules prioritizing the treatment of 56 health-related problems amenable to health care (heart attacks, ischemic stroke, hypertension, diabetes, pneumonia, and specific cancers: breast, lymphoma, prostate, and testicular, among others). The approved legislation established that eligibility only depended on patients' diagnosis and age and was independent of their type of insurance. In addition, the reform established specific and mandatory guidelines for providers defining a maximum time for detection, diagnosis, and treatment of the covered diseases (Missoni and Solimano, 2010). Since then, when a patient's medical diagnosis is confirmed, they are assigned to a specific network (either public or private) to initiate treatment in accordance with the established guidelines. Given budget constraints, the diseases included *de jure* in the GES program were covered in a staggered fashion.

To evaluate the impact of this reform on health outcomes, we use the universe of death and inpatient records and leverage the timing of the program's coverage expansions to implement a difference-in-differences research design. Our data comes from the Department of Health Statistics and Information. The primary data is the universe of individual-level death registries, which include cause of death, year of birth, sex, and place of residence. The secondary data corresponds to the universe of patient-level discharge records, which include the patient's dis-

charge diagnosis and demographics such as year of birth, sex, and place of residence. These records also contain information on surgeries performed, patient's status upon discharge (i.e., dead or alive), insurance coverage, and type of healthcare facility. Using these data, we first construct cells with the counts of our outcomes of interest by ICD-10 code, age group, and year. Then, we classify cells as *treated* based on the ICD-10 codes and age group included in each of the program's coverage expansions, according to the guidelines established by law. In addition, we use longitudinal survey data that includes detailed questions related to the health and expenditures of respondents.

Due to the absence of cell-specific denominators, we estimate a staggered difference-in-differences using a Poisson model. Our main finding is that the reform led to a 4.4% reduction in deaths. Taken at face value, this number implies that lives saved due to the reform represent 2.4% of the total number of deaths in 2003, suggesting an increase in life expectancy large enough to have taken us forward to the mortality conditions of 2005 when life expectancy was 77.78 years. Reassuringly, estimates from an event study are in line with our parallel (relative) trends assumption and indicate that the impact of the reform persisted until the end of our analysis period. We perform several validation exercises and show that our result: i) is not driven by any specific disease; ii) is similar when considering only treated (ever covered) cells for identification; and iii) is robust to recent developments that allow for treatment effect heterogeneity over time or across groups (Wooldridge, 2021). We also examine mortality effects on a subset of diseases that are considered to be "health care–amenable" (Nolte and McKee, 2011), which previous research suggests may be more responsive to better access to medical care (Sommers et al., 2014; Sommers, 2017; Miller et al., 2021). We document that mortality falls by 7.1% for diseases that are amenable to health care but only fall by 2.8% for non-amenable diseases.

Turning to inpatient outcomes, the reform decreased in-hospital mortality by 6.9%. This larger impact is consistent with the fact that individuals in these records sought and received medical attention. Additionally, the reform's guidelines for the timely treatment of diseases likely led to an increase in hospital procedures, as evidenced by a 16% increase in surgeries after the reform. Both of these effects are significant and stable within the time window of our analysis. To assess the socioeconomic impact of the reform, we study heterogeneous effects by type of healthcare facility—whether public or private—and we also explore demographic and geographic heterogeneity. We show that patients at public hospitals outside of the metropolitan

area drove the impact of the reform on in-hospital deaths and surgeries. As public hospitals in less-urban districts disproportionately serve the most disadvantaged patients, we interpret these findings as suggestive evidence that the reform narrowed socioeconomic gaps in access to health care.

Finally, we use longitudinal survey data to study the correlation between GES coverage and the number of medical visits, out-of-pocket health expenditures, and household debt. Among respondents who report ever being diagnosed with a health condition, we find that those whose health condition was covered by the GES program were 46% *more likely to* report a medical visit and 26% *less likely to* report out-of-pocket medical expenditures. Regarding the intensive margin, respondents whose health conditions were covered by the GES program reported 40% more medical visits and 49% lower health care spending. These effects remain sizable (albeit smaller) and statistically significant if we include person fixed-effects, thus leveraging within person variation in the timing of GES coverage. While data limitations prevent us from studying the impact on medical debt, we do not find significant effects of the reform on households' overall indebtedness.

Our paper makes several contributions to the existing literature. First, it adds to the research of health insurance on health outcomes. Most studies have focused on the effects of insurance expansion based on age or socioeconomic status. For instance, Arroyave et al. (2013) shows that in Latin America, mortality disparities decreased due to doubling health insurance in Colombia, and Parker et al. (2018) suggests that the "Seguro Popular" health insurance increased utilization and diagnosis in Mexico. Regarding the U.S's Affordable Care Act insurance expansion, Gruber and Sommers (2019) finds limited evidence of improved health outcomes, although Black et al. (2019) challenges its statistical power. Relatedly, Borgschulte and Vogler (2020) find a reduction in all-cause mortality for ages 20-64, and both Goldin et al. (2020) and Miller et al. (2021) report reductions in mortality for ages 55-64, and for causes of death likely to be influenced by access to healthcare. In contrast to these studies, we assess the impact of a program with a universal scope and a novel design aimed at prioritizing early and adequate diagnoses and treatment of a specific set of diseases.

Second, we contribute to the literature on addressing mortality inequalities by showing that the intervention had differential impacts across different groups. Building on previous studies that examine the relationship between hospital ownership and health performance in Chile

(Cid Pedraza et al., 2015; Basu et al., 2012; Alonso et al., 2019), our paper shows that inpatients at public hospitals—the largest medical bed providers serving the most disadvantaged population in the country—disproportionately benefited from this reform. In terms of demographics, we find no effects on sex-stratified samples and no effects on old age mortality compared to the groups below 80 years old. The latter is in line with the scope of the reform to prevent deaths from conditions amenable to high-quality and timely health care, usually concentrated among individuals below the ages of 75-79 (Mackenbach et al., 2017; Nolan et al., 2022). Moreover, our finding that the effects are larger outside of the major metropolitan area contributes to the literature on geographic disparities (Murray et al., 2006; Bilal et al., 2019; Mena et al., 2021) and suggests that disease-specific reforms may be an alternative way to narrow them down.

Third, our analysis using survey data contributes to the literature on the effects of health insurance on household finance and reinforces previous findings. For instance, our results align with research showing that health coverage can improve health outcomes (see Finkelstein et al. (2018) and references therein) as we find an increase in medical visits. They are also consistent with models and evidence that health insurance reforms improve household resilience to health and income shocks (Starr-McCluer, 1996; Gruber and Yelowitz, 1999), as we find a decrease in out-of-pocket medical expenses. By studying the reform's effect on overall indebtedness, our work is also related to the literature on the relationship between health coverage and debt (Gross and Notowidigdo, 2011; Barcellos and Jacobson, 2015; Mazumder and Miller, 2016), with the caveat that data limitations prevent us from studying *medical* debt.

Finally, our paper also complements previous studies of this program. Closer to our work, Nazzal et al. (2013) conducted a survey between 2008-2009 in six public hospitals, and—focusing on acute myocardial infarction—showed the policy's early success. Likewise, Frenz et al. (2014) used survey data to show that the reform improved access to health care and health status, especially among lower-income Chileans. More recently, Alonso et al. (2019) documented a higher increase in early and long-term survival, for acute myocardial infarction, in public than in private hospitals. In contrast to these papers, we use the *universe* of death and inpatient records and provide *causal* evidence using a quasi-experimental research design.

The remainder of the paper proceeds in the following way: Section 2 describes the institutional background and the GES program. In section 3, we detail the data sources and sample construction. Section 4 describes our empirical strategy and presents the main results. In Sec-

tion 5, we switch to survey data to explore the impact of the reform on other outcomes. Finally, we conclude with a discussion in Section 6.

2 The Explicit Health Guarantees (GES) Insurance Reform

2.1 The Chilean Health Care System

Chile has experienced rapid economic growth since the mid-1980s, with a GDP per capita of nearly $28,500 in 2022, the highest in Latin America. The sustained economic growth has positively correlated with health outcomes over the past decades: life expectancy, avoidable mortality, chronic disease morbidity, and self-rated health is near the OECD average and above the Latin American average (OECD, 2021). However, economic growth benefits have not been accrued to everyone equally. Chile's Gini index of 0.49 in 2017 was the second highest among OECD countries.

In the mid-80s, under dictatorship rule, a two-tier system was introduced: it stipulated a mandatory 7% contribution for workers in the formal economy who would pay into the public system but who could choose to opt-out and use the 7% for private health insurance instead. The *Fondo Nacional de Salud* (FONASA)'s public system is funded by taxes and mandatory contributions. It offers care mainly in public hospitals to everyone that requires it, with three levels of copay (0, 10, or 20%) based on the patient's income and their number of dependents.[1] Private insurance providers, *Instituciones de Salud Previsional* (ISAPREs), offer health plans for different prices and compete in a highly regulated market to attract those who have chosen to use their mandatory contributions in private insurance over the public system. Nearly 78% of the population contributes to and uses the public system while ISAPRES only covers around 17-18% of the population. The remaining 3-4% are covered under an Armed Forces insurance scheme. Moreover, FONASA serves lower-income people—a population with a higher risk of disease and health-related issues—while ISAPRES covers the wealthier, healthier, and younger population (Pardo, 2019).

[1] It is worth mentioning that *within* FONASA, there is an option to facilitate access to care known as the Free Choice Modality (*Modalidad de Libre Elección - MLE*). This option allows users in the high-income segment to use private providers while incurring an increased copayment percentage.

2.2 The Explicit Health Guarantees (GES) Reform

The GES reform was conceived in 2001 as part of major reform for the Chilean Health System toward achieving "effective" Universal Health Coverage. It was a novel effort to expand access and financial coverage, improve quality, and provide timely care administration for specific health-related problems with high mortality, morbidity, and financial impact (Vargas and Poblete, 2008). Although these health conditions were previously covered in public and private systems under the government's universal health care policies, the GES reform ensured and guaranteed timely access to high-quality care for top priority conditions (Erazo, 2011), including heart attacks, ischemic stroke, hypertension, diabetes, pneumonia, and specific cancers: breast, lymphoma, prostate, and testicular, among other.

The guidelines establish a maximum timeline for the diagnosis, treatment, and follow-up to achieve timely care administration. For instance, in the case of time-dependent diseases such as Acute Myocardial Infarction (AMI), there were no standardized procedures before the GES program. Therefore, once the intervention started, depending on the specific case and health care facility, the program covered the following: i) for diagnosis, it covers electrocardiograms and specific blood tests to estimate cell death; ii) for treatment, depending on the health care facility, it mandates an angioplasty in less than 90-120 minutes at high-complexity facilities or a thrombolysis within the first 30 minutes at low-complex facilities. Despite timely diagnoses and treatment being essential for the prognosis and mortality rate related to this pathology before the reform, procedures largely differed between public and private hospitals, particularly between metropolitan and non-metropolitan areas without highly complex facilities. In addition, the GES program certifies quality through registered and certified health providers and ensures financial security through limits to contributions, payments, and co-payments contingent on users' income. In most cases, once the diagnosis is verified by a public or private health provider, patients' are assigned for treatment in a specific network. People cannot choose where to get care; otherwise, they lose the benefit. Depending on the health-related problem, people may have access to free prescriptions.

When initially conceived, the reform was intended to cover 56 health-related problems all at once. However, it was implemented gradually to test its performance and provide the system with resources for the new national standards established in the clinical guidelines (Paraje and

Infante, 2015). It started with a small pilot in August 2002, covering terminal chronic kidney diseases, all childhood cancers, and congenital heart disease. Then, in 2003, cervicouterine and terminal cancers (palliative care) were added. Finally, in 2004, the reform started as a formal pilot for publicly insured seeking care in public hospitals, representing 73% of the population (MINSAL, 2004). This is considered the initial expansion, covering 17 new priority conditions, including high-prevalence diagnoses amenable to mortality-averting healthcare treatment, such as heart attacks, hypertension, and diabetes. Subsequent developments in 2005, 2006, 2007, 2010, 2013, and 2019 brought the total to 85 covered conditions of varying prevalence and amenability to care.

It is essential to note that the reform also targeted specific age groups for some diseases. For instance, childhood cancers cover all types of cancer for people younger than 15. On the other hand, later expansions increased age-group coverage only. For instance, bronchial asthma was covered by the 2006 expansions for people below 15, but in 2010 coverage expanded for those above 15. Finally, there are diseases expanding only for a specific age group; cholecystectomy, a standard treatment of symptomatic gallstones and other gallbladder conditions, is covered only for people between 15-39. Detailed tables with each covered health-related problem and age group can be found in Appendix Tables A.1 through A.4.

3 Data and Sample Construction

3.1 Data Sources

The primary mortality dataset is an individual-level deaths registry coming from the death certificates. This dataset provides us with each individual's cause of death, year of birth, sex, and place of residence. It comprises every death in the country between 1997-2017, almost 2 million records. The secondary data contains patient-level records of discharges from the entire health system between 2001 and 2017. This corresponds to almost 28 million records of patients who stay at least one night in a healthcare facility. It includes the patient's discharge diagnosis and demographics such as year of birth, sex, and place of residence. It also contains information on surgeries performed, whether the patient was dead or alive when discharged, the type of insurance coverage, and the type of health care facility (e.g., whether public or private) where they

received treatment and/or passed away.

Both datasets result from a joint effort between the National Statistics Office, the Vital Records Office, and the Statistics Department of the Ministry of Health. The primary goal of these agencies is to classify each cause of death and patient discharge diagnosis according to the 10th revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-10), the WHO's medical classification list containing codes for diseases, signs, symptoms, abnormal findings, complaints, social circumstances, and external causes of injury or diseases. Key to our empirical strategy is that the detailed clinical guidelines established during the reform are defined on a comprehensive list that includes the ICD-10 codes and the age coverage. The list is constantly updated and is publicly available from the Statistics Department of the Ministry of Health.

3.2 Sample Construction and Descriptive Statistics

To construct our analysis sample, we first identify all diseases that result in deaths. For that purpose, we keep ICD-10 codes that match all death records and in-hospital deaths by patient id, which represent 95% of deaths between 2001 and 2010. We then combined individual deaths and discharge records and constructed counts of deaths, in-hospital deaths, and surgeries by the ICD-10 diseases and 22 age groups (defined as 19 five-year age groups and three ad-hoc groups for newborns, ages 1 to 4, and open-ended interval for deaths above 100). We then classify each resulting cell of ICD-10 and age group into covered and non-covered using the comprehensive list of ICD-10 and ages covered by the GES expansions between 2004 and 2007. For heterogeneity analysis, we also classify the cells from conditions that are amenable to health care following Nolte and McKee (2011) and Sommers et al. (2014); see Appendix Table A.5 for a detailed list of the ICD-10 codes included.

We removed diseases included in the pilot program that happened between 2002-2003 from our sample because it was not clear how these conditions were chosen, and we only have data starting in 2001.[2] We also decided not to consider diseases included in the second wave of

[2]This group represents 15.8% of deaths in the study period, and 69% of these were terminal cancers. Childhood cancers and congenital heart diseases were only covered for people below 15 years, while terminal cancer coverage focused on pain treatment not intended to avoid death. Additional specific cancers started to be covered later, such as colon, ovarian, and bladder in 2013, and lung, thyroid, kidney, and myelomas in 2019.

expansions (2010, 2013, and 2019) as controls in our study. The main reason for this decision was that the 2010 and 2013 groups of diseases covered were piloted before the program formally expanded, which can introduce bias to our estimates. Data limitations also prevent us from studying diseases included in the 2019 expansion because we only have data until 2017.[3] Among diagnoses covered within our time frame, 16 did not have deaths during the study period.[4] As we mentioned in the last paragraph in subsection 2.2, for some diseases, later expansions increased age-group coverage only. Therefore, we have an unbalanced panel because later expansions are not part of the study. For example, in our sample, we only study age groups below 15 for bronchial asthma because coverage was expanded to include those above 15 in the 2010 expansion, which is not part of the expansions studied. Likewise, there are diseases (ICD-10 codes) in both covered and uncovered groups because their coverage was only for a specific age group. For a detailed number of diseases covered and cells, see Appendix Table A.6.

All in all, we end up with a panel of cells with counts by age group and ICD-10 codes for 35 health-related problems covered by the reform during the 2004-2007 expansions. Almost 60% of deaths in our sample are concentrated among diseases of the circulatory, respiratory, and digestive systems, while neoplasms and injuries account for an additional 20% (for details, see Table A.7). Table A.8 reinforces the targeted nature of the reform by showing that all expansions combined targeted almost 50% of deaths in the period, in an evenly distributed fashion (between 10-15% for each expansion). Finally, Table A.9 presents descriptive statistics regarding the age structure of our sample. We see that almost 75% of deaths occurred between the ages of 50 and 89. We also see the usual pattern of increasing deaths with age, peaking in the 80-84 age group and then decreasing. We also see that the reform covers around 50% of the deaths within each age group. For the 2007 distribution, there is an interesting pattern. The number of deaths decreased with age, which aligns with the fact that most of these deaths are related to polytraumatized health problems.

[3]Diseases included in 2019 incorporate four cancers that were covered during the 2002-2003 pilot before its specific coverage started (these were treated under cancer's palliative care). Additionally, Alzheimer's coverage started in 2019, but disease classification before 2012 is noisy and is associated only with three causes of death compared to the 15 listed in 2019. Together, these groups of deaths represented 15%.

[4]These diseases correspond to scoliosis, cataracts, refractive impairment, strabismus, oral health for children, diabetic retinopathy, detached retina, depression, orthotics for older adults (canes, wheelchairs, others), dental emergencies, tooth loss in older adults, traumatic brain injury, eye trauma, delivery care with analgesia, major burns, hypoacusis.

4 Reform Impact on Health Outcomes

4.1 Empirical Strategy

The reform's design- aimed at guaranteeing the early and adequate diagnoses and treatment of high-cost and high-mortality diseases- allows us to implement a staggered difference-in-differences research design. In particular, we leverage the timing of coverage among different disease-age cells to study changes in cell-level outcomes (e.g., deaths) before and after reform coverage. Because we only observe deaths and not how many individuals suffered from each disease, we do not have denominators for constructing disease-specific death rates. In the absence of denominators, the outcomes of interest will be yearly counts within disease-age cells, e.g., the number of deaths or surgeries associated with ischemic strokes among people between 35 and 39 years old in a given year. Thus, we will fit Poisson models for counts using a log link. The general specification that we estimate is given by:

$$y_{dt} = \exp(\alpha_d + \gamma_t + \beta GES_{dt} + \epsilon_{dt}), \tag{1}$$

where y_{dt} is the count of our outcome of interest for a cell d (disease-age) in period t. GES_{dt} is an indicator that equals one from the first time a disease-age cell is covered by GES and onward, i.e., the treatment is an absorbing state. α_d represents cells' fixed effects that control for unobservables specific to the disease-age cell and γ_t are time-fixed effects accounting for unobservable time shocks. Finally, ϵ_{dt} is an error term clustered at the level of treatment (disease-age cell). In this model, identification of the *causal* effect of the GES reform is predicated upon the assumption that—conditional on time-invariant disease-age cell indicators and year aggregate shocks—there are no unobserved factors that correlated with both the timing of coverage and other determinants of health outcomes.

Our parameter of interest is the rate ratio (RR) identified through the Poisson model. For two time period, the RR is defined as:

$$\exp(\beta) = RR = \frac{\frac{E[Y_{d2}|GES=1]}{E[Y_{d1}|GES=1]}}{\frac{E[Y_{d2}|GES=0]}{E[Y_{d1}|GES=0]}}, \tag{2}$$

where Y_{dt} is the count of deaths for diseases-age cell d in period t, and GES equals one when a cell is covered by GES. A rate ratio sometimes called an incidence density ratio or incidence rate ratio is the relative difference measure used to compare the incidence rates of events occurring at any given point in time (Dicker et al., 2006). Therefore, the interpretation of the value of a rate ratio is similar to that of the risk ratio. That is, a rate ratio of 1 indicates equal rates in the two groups, a rate ratio greater than 1 indicates an increased risk for the treated group ($GES = 1$), while a rate ratio less than 1 indicates a decreased risk for the treated group ($GES = 1$). To ease the exposition, we present our results as percent changes by subtracting 1 from the RR, i.e, $exp(\beta) - 1$. Thus, if the GES reform led to a relative decrease in the number of deaths among the covered diseases, we would expect our coefficient to be negative.

In our Poisson setting, the identification assumption, commonly known as "parallel trends", requires that the death ratios between the group of diseases (covered and not covered) would have been constant over time in the absence of the reform. For this reason, this assumption is also referred to as "parallel relative trends". In other words, the implicit identifying assumptions are: (1) fixed characteristics within diseases (no change over time) and (2) time trends for growth in deaths are the same for covered and not covered diseases. To assess the plausibility of this parallel (relative) trends assumption, we examine the dynamic effects of GES using event studies around the time a new disease is covered. The time periods and coverage expansions in our analysis allow us to have a 3-year moving window around each expansion. We will use a leads-and-lags model in event time, with the first expansion year set to zero. Specifically, we estimate the following equation:

$$y_{dt} = exp\left(\alpha_d + \gamma_t + \sum_{k=\underline{C}}^{-2} \beta_k D_{dt}^k + \sum_{k=0}^{\overline{C}} \beta_k D_{dt}^k + \epsilon_{dt}\right), \tag{3}$$

where $D_{dt}^k = 1[t = GES_d + k]$, and GES_d is the timing of inclusion of disease-age group d. D_{dt}^k is a dummy variable indicating that disease-age cell d was included in GES k periods ago (or will be included k periods ahead for negative values of k). We normalize the coefficients such that $\beta_{k=-1} = 0$—that is, treatment is re-coded in event time relative to the year in which each disease-age group was included in the GES expansions. Therefore, the β_k coefficients can be

interpreted as the effect of GES on y_{dt} for each k period, relative to the date before the inclusion of d in GES.

4.2 Main Results

We begin by exploring the mortality impact of the reform using raw data. In Figure 1 we plot the growth of the number of deaths in covered diseases against the growth in the number of deaths in never-covered diseases for each expansion. Panel (a) shows that growth in deaths covered by the 2004 expansion decreased compared to the never-covered group. Panel (b) shows that deaths of diseases covered in 2005 also decreased proportionally more than deaths of non-covered diseases a year after the expansion, although the difference between covered and non-covered is smaller in this case. Panel (c) shows the evolution of deaths for diseases whose coverage was included in 2006. In this case, there is also a decline compared with the never covered. Finally, panel (d) shows the differential trends between diseases included in the 2007 expansion and those never covered. Again, all deaths increased, but those covered by the 2007 expansion increased far less. Importantly, the overall increase in deaths shown in Figure 1 is mainly driven by an aging population.[5]

Even though previous evidence is purely descriptive, it suggests that the reform had an effect on mortality. To formally study this hypothesis—and to quantify the impact of the reform—we now present the results obtained from our difference-in-differences research design. Table 1 presents the results obtained from estimating equation (1). Our main result is presented in Column (1) and considers the count of all deaths as the dependent variable.[6] Consistent with the preliminary evidence, we find a statistically significant impact of the reform on mortality: the average risk of dying from diseases going from uncovered to covered decreases by 4.4% after the reform began. This effect is a weighted average across all disease-age cells and expansions, which allows us to compute the number of deaths averted due to the reform. In our estimation sample, the covered group had 29,331 deaths in the pre-expansion period. Therefore, there

[5] Appendix Figure A.1 shows standardized cause-specific death rates accounting for population growth and population aging by weighting yearly death rates with the age distribution in 2001. It shows that adjusted death rates are actually *decreasing* throughout the analysis window. For the interested reader, Appendix Figure A.2 presents population pyramids showing how the age distribution has changed in Chile during the last 3 decades.

[6] Appendix Table A.10 shows that we obtain similar results if we estimate a negative binomial regression or a linear regression using the log of deaths+1 and the inverse hyperbolic sine as ad-hoc transformations to deal with the zero count cells.

would have been 1,290 deaths saved once they went from uncovered to covered. Considering 53,950 deaths a year before the coverage starts lives saved *due* to the reform would represent 2.4% of the deaths in the sample.

To study the dynamics of the impact on mortality, Figure 2 presents the event-study estimates obtained from equation (3) using the count of all deaths as the dependent variable.[7] The horizontal axis shows the years relative to the expansion, with event time denoting the first year of the expansion. We omit event time -1 so that all estimates are relative to the year before the expansion. Point estimates of leads and lags are plotted along with their 95% confidence intervals. This figure shows that pre-period estimates are not statistically different from zero, a result in line with our parallel (relative) trends assumption.[8] Moreover, the figure shows a decrease in deaths in the treated diseases immediately following the expansions. Indeed, the magnitude of the point estimates grows over the post-expansion periods so that, four years post-treatment, deaths have declined by 7% ($p \leq 0.001$) relative to the year before the expansion.

Recent literature on two-way fixed effects estimators have shown that estimates from this model can differ from the group's ATT in the presence of treatment effect heterogeneity (De Chaisemartin and d'Haultfoeuille, 2020; Callaway and Sant'Anna, 2021; Sun and Abraham, 2021). To address this concern, we follow recent work by Wooldridge (2021) and Wooldridge (2022), whose method is robust even if the treatment effects are heterogeneous over time or across groups, and which can be adapted to non-linear settings such as ours. In particular, we run a regression with cell and year-fixed effects as before, but now we saturate it with the interaction of all treatment cohorts (GES expansions) and event time dummies. Intuitively, this approach assesses the impact of the GES expansions jointly but allows each expansion to have its own dynamic, using never treated cells as controls. To present our results, we recover estimates and confidence intervals from the pooled Poisson regression and plot them separately for each expansion. Figure A.4 in the appendix presents these results. Reassuringly, we find evidence consistent with our main findings across all expansions.

We also check the robustness of our results to estimating equation (1) in a sample of *ever covered* cells. In this case, identification of the impact of the reform only leverages variation

[7] Figure A.3 finds similar effects between Poisson and Negative Binomial

[8] Table A.11 complements this validation exercise by showing pre-treatment characteristics coming from the death records of covered and non-covered cells. Overall, we observe balance along an array of cell characteristics including type of insurance, education, gender, marital status, and geographical location.

in the timing of adoption among covered diseases. We find that among "ever covered" cells, expansions led to a 4% decrease in mortality (see Appendix Table A.12, column 1). This is very similar to the main estimate from our staggered difference-in-differences.[9] Finally, to assess whether our results are driven by a particular set of diseases, we estimate our main difference-in-differences model, given by equation (1), but removing one treatment cell from the sample each time. Figure 3 shows the results obtained from this exercise. Reassuringly, in all cases we find negative and statistically significant impacts of the reform on mortality. Moreover, most point estimates are around the average effect of a 4.4% mortality decrease, with the exception of the estimates obtained after the removal of Ischemic stroke from the set of treated cells, which leads to a smaller impact of the reform on mortality, suggesting that this disease accounts for a non-trivial part of the overall treatment effect.

Having stated the impact of the reform on overall mortality, we now turn to other outcomes. In light of recent research suggesting that some diseases may be more responsive to access to medical care (Sommers et al., 2014; Borgschulte and Vogler, 2020; Miller et al., 2021), we begin by studying the impact of the reform on two subsets of diseases: those that are considered to be "health care–amenable" and those that are not. For this analysis, we use the classification described in section 3.2.[10] Columns (2) and (3) of Table 1 shows the estimates obtained from estimating equation (1) on Amenable and Non-Amenable death counts. For both outcomes, the effect is negative. Nonetheless, the magnitudes of the effects are substantially different, with the effect on amenable causes of death more than doubling the effect on the rest of the causes. According to our estimates, deaths amenable to health care decreased by 7.1% as a consequence of the reform. This is a large effect on a relatively smaller set of deaths, a fact that leads us to conclude that a large part of the effect on mortality is driven by the targeting of causes of death that are amenable to health care.

To complement our previous result, in Figure 4 we present event study evidence on the set of amenable and non-amenable diseases. Panels (a) and (b) display the plots for the set of diseases amenable and non-amenable to health care, respectively. For amenable diseases, we see that the figure is very similar to the one considering all deaths but is larger in magnitude. For the

[9] In the appendix, we also present estimates on the impact of the reform that consider different samples of diseases included in different expansions and that only use never-treated cells as controls. Columns 2-5 of Appendix Table A.12 and Figure A.5 present these results.

[10] Our classification encompasses both the work by Nolte and McKee (2011) and by Sommers et al. (2014). See Appendix Table A.5 for details.

non-amenable or the "less amenable" set of deaths, there is also a negative but much smaller effect. Reassuringly, none of these event studies suggest evidence of the existence of pre-trends. All in all, this analysis shows that although the reform targeted deaths amenable to health care, it also had an impact, albeit smaller, on the deaths "less amenable" to care. In Appendix Table A.13 we perform a robustness check and repeat this analysis under alternative classifications of deaths, including Tobias and Yeh (2009), Nolte and McKee (2003), and the one used by the European Union. We find similar results in all these cases.

Finally, we leverage inpatient records to complement previous results. In columns (4) and (5) of Table 1, we present the estimates obtained from estimating equation 1 using in-hospital deaths and surgeries as dependent variables. We find that in-hospital deaths decreased by 6.9% as a consequence of the reform. This effect, larger than the mortality impact on the population as a whole, is consistent with the fact that in-hospital deaths come from a sample of patients for whom we know medical care was provided and who spent at least one night, i.e., they show up in a hospital's discharge records. Panel (a) of Figure 5 shows the dynamic impact of the reform on in-hospital deaths. We observe that differences between treatment and control groups were almost nonexistent before the treatment. However, exactly after the coverage expansion, the number of in-hospital deaths for covered diseases decreased significantly.

Regarding surgeries, our estimates reveal that the reform increased them by 16% (column 5 of Table 1). This is a significant increase consistent with the reform's goal of prioritizing the treatment of covered diseases. Turning to the dynamic effects, Panel (b) of Figure 5 confirms that surgeries increased in the wake of the expansions. The estimates indicate that surgeries had increased by 4% immediately after the expansions and grew over the post-expansion period. Four years after treatment, surgeries had increased by almost 30%, although our estimate for the last period is significantly noisier than the previous ones.

4.3 Socioeconomic, Demographic and Geographic Heterogeneity

To study the heterogeneous impacts of the reform along the socioeconomic, demographic, and geographic dimensions, in this subsection we replicate our main analysis in different sub-samples.

We begin by exploring socioeconomic disparities. Public hospitals are the largest medical

bed providers and serve the most disadvantaged populations.[11] Moreover, public providers tend to be more crowded and have longer wait times. Indeed, as of 2016, only 24% of the 348 hospitals in the country were private, but 55% of doctors worked in the private sector (Clinicas de Chile, 2016; Gonzalez et al., 2022). Additionally, previous studies found that patients at public hospitals show a higher risk of in-hospital death (Cid Pedraza et al., 2015). In this context: do patients seeking care at public hospitals benefit more from this reform? To answer this question, we estimate our main regression but distinguish by type of healthcare provider. Table 2 shows our results. Estimates show a statistically significant effect of the reform on mortality in public hospitals, which decreased by 7.3%. The corresponding estimate for private hospitals is only 2.5% and is not statistically significant.[12] We find a consistent pattern when focusing on surgeries. In public hospitals, surgeries increased by 23%, but they only increased by 0.8% at private hospitals. All in all, this analysis shows that most of the impact of the reform is concentrated in public hospitals, a finding that we interpret as evidence of the reform reducing socioeconomic disparities.

We now present stratified results between different sexes and age groups. This analysis is motivated by the fact that diseases expanded only for specific sex and age groups. While the disease-age group cells are very similar between the sexes, this is not the case for the age groups.[13] Table 3 presents our results. Even though the reform targeted sex-specific diseases, we find no significant differences for sex-stratified results in terms of deaths and surgeries. However, we find important differences between age groups. Notably, and despite the fact that the increase in surgeries is similar for those between 0 and 49 and those above 80, the decrease in deaths between ages 0 and 49 was almost four times larger than the decrease in deaths among those above 80. The absence of an effect on old age mortality may be associated with the scope of the reform on deaths amenable to high-quality and timely health care, which are usually found in patients below the age of 75-79 (Mackenbach et al., 2017; Nolan et al., 2022).[14] It can also be related to the fact that co-morbidity increases with older age; hence, assigning a single underlying cause of death becomes more uncertain at older ages, making the

[11] Based on discharge records, 96% of patients at public hospitals have public insurance.

[12] Appendix Table A.15 shows that this result is robust to the removal of diseases included in the pilot expansion of 2004, which exclusively targeted patients with public insurance seeking care at public hospitals.

[13] In fact, we see that observations decrease with age because, by definition, we are grouping fewer cells for older ages.

[14] In our sample, 23% of deaths amenable to healthcare are below 50 years old, 77% for those between 50 and 79 years old. None of the deaths after 80 years are classified as deaths amenable to health care

classification noisier for these groups of deaths (Weber and Clerc, 2017).

Finally, we study the heterogeneous effects of the reform by geographic location. Specifically, we estimate the impact of the GES coverage expansions in each macro zone of Chile. These macro-zones aim to represent an evenly distributed population across the country.[15] In Table 4, we present the results obtained after estimating our main equation (1) in different macro-zones. Panel A, which considers deaths as the dependent variable, shows non-significant effects for the extreme zones, such as the North and Austral zones. More interestingly, we find that in the Metro area, where the capital city—Santiago—is located, the reform did not have a significant effect on deaths. In contrast, deaths decreased in the range of 6.8 to 7.7 percent in the Center, Center-South, and South macro-zones. Looking at the impact on surgeries (Panel B), our results show large increases, ranging from 12% to 21%, in all but the South and Austral zones. This result could be explained by fewer resources (physicians, equipment) in the most extreme and rural south regions, in contrast to the capital city where it was easier to access evidence-based treatment and procedures before the reform standardized them nationwide. This could also be interpreted as evidence that with the implementation of the clinical guidelines and prioritization of specific procedures, the reform narrowed geographical disparities.

In sum, our heterogeneity analysis has shown that: i) the reform had significantly larger effects on public hospitals, suggesting it helped to reduce socioeconomic disparities; ii) had similar effects for men and women, but most of the decrease in mortality was concentrated on people ages 0-49; and iii) there was substantial variation in the impact of this reform across macro-zones, with the larger decreases in mortality outside of the metropolitan area.

5 Reform Impact on Medical Visits and Household Finance

The existing literature has identified several financial outcomes that can be affected by health reforms (see Finkelstein et al., 2018 for a review). For instance, health reforms can decrease out-of-pocket medical expenses, thus increasing household resilience to health and income shocks. Likewise, public insurance programs may decrease households' medical debt (Gross and Notowidigdo, 2011; Barcellos and Jacobson, 2015; Mazumder and Miller, 2016) and reliance on

[15]Most of the population is between the center and south macro zones, heavily concentrated in Santiago, the central Metropolitan area (Metro) in the country with almost 40% of the population, totaling 8 million people.

precautionary savings (Starr-McCluer, 1996; Gruber and Yelowitz, 1999). In this section, we study these dimensions of household finance, although not exhaustively.

For this purpose, we leverage the main longitudinal survey in the country, known as the Social Protection Survey, or EPS by its acronym in Spanish. The survey is organized into modules, including demographics and health. While the EPS reports general questions for all household members, detailed questions related to health are only asked to the head of the household. Moreover, although the first survey was done in 2002, questions related to the GES reform are only available since 2009. Consequently, we construct a panel data set at the household-head level for our analysis using the years 2009, 2012, and 2015. For each household head, we observe their age, gender, educational attainment, type of health insurance, self-perception of health status, number of medical visits, total medical expenditures, and overall indebtedness. Importantly, we also know whether the respondent has been diagnosed with a specific health condition from a set of eleven (broad) diseases and whether she has benefited from GES coverage for said condition.[16] Figure A.6 shows the share of respondents, by type of insurance, who report that GES covered their health condition. This figure highlights two facts consistent with the nature of the reform. First, coverage has been increasing over time. Second, there is no large difference between private and public insurance respondents.

We aim to study the impact of the reform on health care access, out-of-pocket health expenditures, and overall indebtedness. To do so, we focus on respondents who report ever being diagnosed with a health condition and for them, we estimate the following model:

$$Y_{it} = \alpha + \rho_t + \beta \text{GES}_{it} + \gamma X_{it} + \varepsilon_{it}, \tag{4}$$

where Y_{it} is an outcome of respondent i at time t, ρ_t are survey year fixed effects, GES_{it} is an indicator that equals one if the respondent declares to have benefited from GES coverage for her health condition, and X_{it} is a vector of controls including age, age squared, self-perceived health status, type of insurance, gender, and education indicators (any college and any high school dummies). Finally, ε_{it} is an error term clustered at the survey's respondent level.

The parameter of interest is β, which under a (strong) conditional independence assumption,

[16] The set of health conditions includes: asthma, depression, diabetes, hypertension, heart-related problems, cancer, arthritis or arthrosis, renal disease, stroke, mental illness, and HIV/AIDS.

can be interpreted as the effect of GES coverage on the outcome of interest. Taking advantage of the panel structure of our data, we also estimate a more stringent specification, including person-fixed effects. In this case, β is identified under a strict exogeneity assumption by leveraging variation in GES coverage across time. Naturally, given the short panel (3 survey years), controlling for time-invariant respondents' unobserved characteristics may come at the cost of exacerbating measurement error problems.

We study health and household finance outcomes. First, we focus on the number of medical visits during the past two years. Medical visits include general, specialty, and urgent consultations, as well as exam visits (e.g., X-rays, lab exams), surgeries, and hospitalizations. Second, we study out-of-pocket medical expenditures related to the aforementioned medical visits. Third, we look at indebtedness. Unfortunately, questions referring specifically to medical debt are not available in the survey. Thus, we construct a measure of indebtedness that considers loans from banks or financial institutions and loans from friends and informal institutions. We exclude auto loan and education loan debt to better approximate health-related debt.

Table 5 presents our estimates of equation (4). Panel A focuses on the extensive margin, i.e., we construct the outcome variables as binary indicators that equal one if the respondent had *any* medical visit, had *any* medical expenditure, or held *any* debt. Columns 1 to 3 show a positive and statistically significant impact of GES coverage on the likelihood of reporting medical visits. Likewise, columns 4 to 6 show a negative and statistically significant effect of GES coverage on the likelihood of reporting any out-of-pocket medical expenditure. When looking at indebtedness, however, we do not find robust evidence of a significant impact on the likelihood of holding debt. Indeed, the negative impact of column 7 becomes positive and non-significant after the inclusion of controls and person-fixed effects. Focusing on our preferred specifications in columns 2 and 5, and relative to the mean among non-covered, the magnitude of these impacts is economically significant; respondents whose health condition was covered by GES are 46% more likely to report a medical visit and 26% less likely to report out-of-pocket medical expenditures.

In Panel B of Table 5, we turn to the intensive margin, i.e., we construct the outcome variables as the log of the number of medical visits, the log of out-of-pocket medical expenditures (in 2022 USD), and the log of the amount of debt (in 2022 USD). As shown by the estimates, GES coverage had significant effects on the number of medical visits and the amount of out-of-

pocket health spending. In this case, again, we cannot reject the null of a zero impact of GES coverage on people's indebtedness. In terms of magnitudes, respondents whose health condition was covered by GES have 40% more medical visits and spend 49% less on health care. All in all, these results suggest that the reform led to more medical care and less out-of-pocket spending among its beneficiaries.

6 Discussion

As the international community prioritizes cost-effective policy interventions to achieve universal health coverage (UHC),[17] the need for rigorous evidence on the impact of health reforms has increased. In this article, we studied the impact of a large health reform that guaranteed medical treatment for sick patients based solely on their diagnoses (ICD-10) and age, i.e., independent of patients' insurance and income.

Leveraging rich administrative data and the staggered coverage of disease-age groups, we showed that this reform led to a 4.4% decrease in deaths. Importantly, this result translates into a substantial number of lives saved. We calculate that before the policy, about 29,000 individuals died of diseases eligible for coverage. Therefore, approximately 1,300 deaths per year were averted thanks to the reform. In terms of monetary benefits, using Chile's median estimates of the value of a statistical life (Mardones and Riquelme, 2018; Parada-Contzen, 2019), the reform created benefits valued at USD $5.2 billion. Regarding costs, the VAT increment to fund this program (Missoni and Solimano, 2010) increased revenues by about USD $1 billion in one year, which is approximately a fifth of the benefits valued because of the lives saved. Furthermore, a simple back-of-the-envelope calculation suggests that this reform increased life expectancy by 0.29 years (as of 2003, before implementation), a significant effect that would have taken us forward close to the mortality conditions of 2005, when life expectancy was 77.78 years. See Appendix 6 for details.

[17] In 2015, United Nations member states agreed to work towards UHC by 2030, following the World Health Organization and others' argument that UHC progress leads to improvements in overall population health. UHC means that all individuals and communities receive the health services they need without suffering financial hardship; this requires implementing specific policies that emphasize care for women, adolescents, and other vulnerable populations (The Lancet, 2019).

On a final note about the external validity of our results, we are aware that countries may follow different paths to achieve universal health coverage, depending on their economic and historical contexts (Reich et al., 2016). Moreover, the type of diseases targeted by reforms in other countries may be different, potentially leading to different mortality effects. Nonetheless, we hope that the targeted healthcare reform studied here can inform policymakers worldwide. Assessing how these types of disease-targeted reforms fare in different contexts is an interesting task for future work.

References

Alonso, F., C. Nazzal, F. Cerecera, and J. I. Ojeda (2019). Reducing health inequalities: comparison of survival after acute myocardial infarction according to health provider in chile. *International Journal of Health Services 49*(1), 127–141.

Arroyave, I., D. Cardona, A. Burdorf, and M. Avendano (2013, mar). The impact of increasing health insurance coverage on disparities in mortality: Health care reform in Colombia, 1998-2007. *American Journal of Public Health 103*(3).

Barcellos, S. H. and M. Jacobson (2015). The effects of medicare on medical expenditure risk and financial strain. *American Economic Journal: Economic Policy 7*(4), 41–70.

Basu, S., J. Andrews, S. Kishore, R. Panjabi, and D. Stuckler (2012). Comparative performance of private and public healthcare systems in low-and middle-income countries: a systematic review. *PLoS medicine 9*(6), e1001244.

Bilal, U., M. Alazraqui, W. T. Caiaffa, N. Lopez-Olmedo, K. Martinez-Folgar, J. J. Miranda, D. A. Rodriguez, A. Vives, and A. V. Diez-Roux (2019, dec). Inequalities in life expectancy in six large Latin American cities from the SALURBAL study: an ecological analysis. *The Lancet Planetary Health 3*(12), e503–e510.

Black, B. S., A. Hollingsworth, L. Nunes, and K. Simon (2019). *The effect of health insurance on mortality: power analysis and what we can learn from the affordable care act coverage expansions*. National Bureau of Economic Research Cambridge (MA).

Borgschulte, M. and J. Vogler (2020, jul). Did the ACA Medicaid expansion save lives? *Journal of Health Economics 72*.

Callaway, B. and P. H. Sant'Anna (2021). Difference-in-differences with multiple time periods. *Journal of Econometrics 225*(2), 200–230.

Cid Pedraza, C., C. A. Herrera, L. Prieto Toledo, and F. Oyarzún (2015). Mortality outcomes in hospitals with public, private not-for-profit and private for-profit ownership in chile 2001–2010. *Health policy and planning 30*(suppl_1), i75–i81.

Clinicas de Chile (2016). Dimensionamiento del Sector de Salud Privado de Chile: Actualización a Cifras 2016. Technical report.

De Chaisemartin, C. and X. d'Haultfoeuille (2020). Two-way fixed effects estimators with heterogeneous treatment effects. *American Economic Review 110*(9), 2964–96.

Dicker, R. C., F. Coronado, D. Koo, and R. G. Parrish (2006). Principles of epidemiology in public health practice; an introduction to applied epidemiology and biostatistics.

Erazo, Á. (2011). La protección social en chile el plan auge: Avances y desafíos.

Finkelstein, A., N. Mahoney, and M. J. Notowidigdo (2018). What does (formal) health insurance do, and for whom? *Annual Review of Economics 10*, 261–286.

Frenz, P., I. Delgado, J. S. Kaufman, and S. Harper (2014, sep). Achieving effective universal health coverage with equity: evidence from Chile. *Health policy and planning 29*(6), 717–731.

Goldin, J., I. Z. Lurie, and J. McCubbin (2020, dec). Health Insurance and Mortality: Experimental Evidence from Taxpayer Outreach. *The Quarterly Journal of Economics 136*(1), 1–49.

Gonzalez, F., L. R. Martinez, P. Muñoz, and M. Prem (2022). Does higher education reduce mortality? evidence from a natural experiment.

Gross, T. and M. J. Notowidigdo (2011). Health insurance and the consumer bankruptcy decision: Evidence from expansions of medicaid. *Journal of public Economics 95*(7-8), 767–778.

Gruber, J. and B. D. Sommers (2019, sep). The Affordable Care Act's Effects on Patients, Providers and the Economy: What We've Learned So Far. *Journal of Policy Analysis and Management 38*(4), 1028–1052.

Gruber, J. and A. Yelowitz (1999). Public health insurance and private savings. *Journal of Political Economy 107*(6), 1249–1274.

Levy, H. and D. Meltzer (2008, apr). The Impact of Health Insurance on Health. *Annual Review of Public Health 29*(1), 399–409.

Mackenbach, J. P., Y. Hu, B. Artnik, M. Bopp, G. Costa, R. Kalediene, P. Martikainen, G. Menvielle, B. H. Strand, B. Wojtyniak, et al. (2017). Trends in inequalities in mortality amenable to health care in 17 european countries. *Health Affairs 36*(6), 1110–1118.

Mardones, C. and M. Riquelme (2018). Estimation of the value of statistical life in chile and extrapolation to other latin american countries. *Latin American Research Review 53*(4), 815–830.

Mazumder, B. and S. Miller (2016). The effects of the massachusetts health reform on household financial distress. *American Economic Journal: Economic Policy 8*(3), 284–313.

Mena, G. E., P. P. Martinez, A. S. Mahmud, P. A. Marquet, C. O. Buckee, and M. Santillana (2021). Socioeconomic status determines covid-19 incidence and related mortality in santiago, chile. *Science 372*(6545), eabg5298.

Miller, S., N. Johnson, and L. R. Wherry (2021). Medicaid and Mortality: New Evidence from Linked Survey and Administrative Data*. *The Quarterly Journal of Economics*.

MINSAL (2004). Documento para la aplicación del sistema auge en las redes de atención del sistema nacional de servicios de salud.

Missoni, E. and G. Solimano (2010). Towards universal health coverage: the chilean experience. *World health report*.

Moreno-Serra, R. and P. C. Smith (2012). Does progress towards universal health coverage improve population health? *The Lancet 380*(9845), 917–923.

Murray, C. J., S. C. Kulkarni, C. Michaud, N. Tomijima, M. T. Bulzacchelli, T. J. Iandiorio, M. Ezzati, M. Rahbari, S. Rahlfs, E. Jortzik, I. Bogeski, and K. Becker (2006, mar). Eight Americas: Investigating mortality disparities across races, counties, and race-counties in the United States. *PLoS Medicine 12*(4).

Nazzal, C., F. Lanas, M. L. Garmendia, C. Bugueño, E. Mercadal, E. Garcés, P. Yovaniniz, and P. Sanhueza (2013, aug). [Universal health coverage and accomplishment of secondary prevention goals among patients with acute myocardial infarction]. *Revista medica de Chile 141*(8), 977–986.

Nolan, A., P. May, S. Matthews, C. Normand, R. A. Kenny, and M. Ward (2022). Public health insurance and mortality in the older population: Evidence from the irish longitudinal study on ageing. *Health Policy 126*(3), 190–196.

Nolte, E. and M. McKee (2003, sep). Measuring The Health Of Nations: Analysis Of Mortality Amenable To Health Care. *BMJ: British Medical Journal 327*(7424), 1129–1132.

Nolte, E. and M. McKee (2011). Variations in amenable mortality—Trends in 16 high-income nations. *Health Policy 103*(1), 47–52.

Parada-Contzen, M. V. (2019). The value of a statistical life for risk-averse and risk-seeking individuals. *Risk Analysis 39*(11), 2369–2390.

Paraje, G. and A. Infante (2015). La reforma auge 10 años después. *Documento de Trabajo*.

Pardo, C. (2019). Health care reform, adverse selection and health insurance choice. *Journal of health economics 67*, 102221.

Parker, S. W., J. Saenz, and R. Wong (2018, feb). Health Insurance and the Aging: Evidence From the Seguro Popular Program in Mexico. *Demography 55*(1), 361–386.

Reich, M. R., J. Harris, N. Ikegami, A. Maeda, C. Cashin, E. C. Araujo, K. Takemi, and T. G. Evans (2016). Moving towards universal health coverage: lessons from 11 country studies. *The Lancet 387*(10020), 811–816.

Sommers, B. D. (2017, jul). State medicaid expansions and mortality, revisited: A cost-benefit analysis. *American Journal of Health Economics 3*(3), 392–421.

Sommers, B. D., S. K. Long, and K. Baicker (2014). Changes in mortality after Massachusetts health care reform : A quasi-experimental study. *Annals of Internal Medicine*.

Starr-McCluer, M. (1996). Health insurance and precautionary savings. *The American Economic Review 86*(1), 285–295.

Sun, L. and S. Abraham (2021). Estimating dynamic treatment effects in event studies with heterogeneous treatment effects. *Journal of Econometrics 225*(2), 175–199.

The Lancet (2019). Ensuring and measuring universality in UHC. *The Lancet 393*(10166), 1.

Tobias, M. and L.-C. Yeh (2009). How much does health care contribute to health gain and to health inequality? Trends in amenable mortality in New Zealand 1981–2004. *Australian and New Zealand Journal of Public Health 33*(1), 70–78.

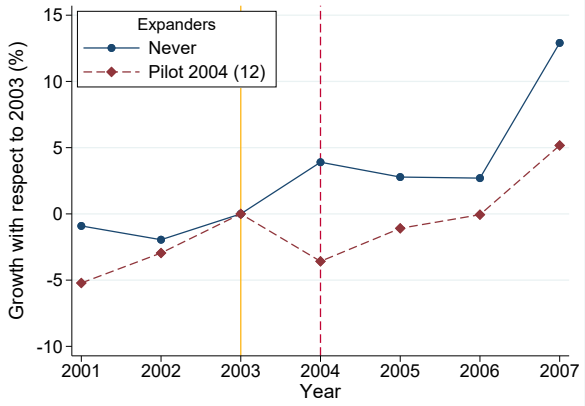
Vargas, V. and S. Poblete (2008). Health prioritization: the case of chile. *Health affairs 27*(3), 782–792.

Weber, A. and M. Clerc (2017). Deaths amenable to health care: Converging trends in the eu? *Health Policy 121*(6), 644–652.

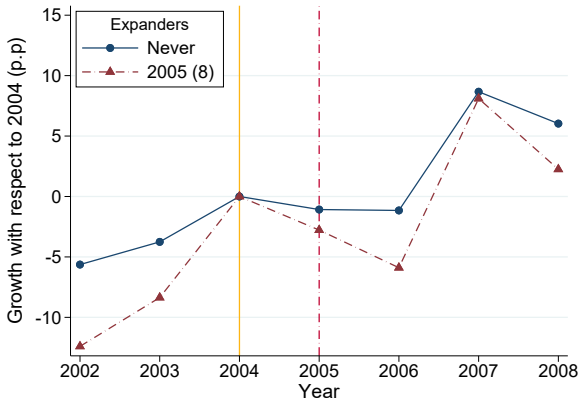
Wooldridge, J. M. (2021). Two-way fixed effects, the two-way mundlak regression, and difference-in-differences estimators. *Available at SSRN 3906345*.

Wooldridge, J. M. (2022). Simple approaches to nonlinear difference-in-differences with panel data. *Available at SSRN 4183726*.

Figure 1: Growth in deaths for each GES expansion



(a) 2004 Expansion



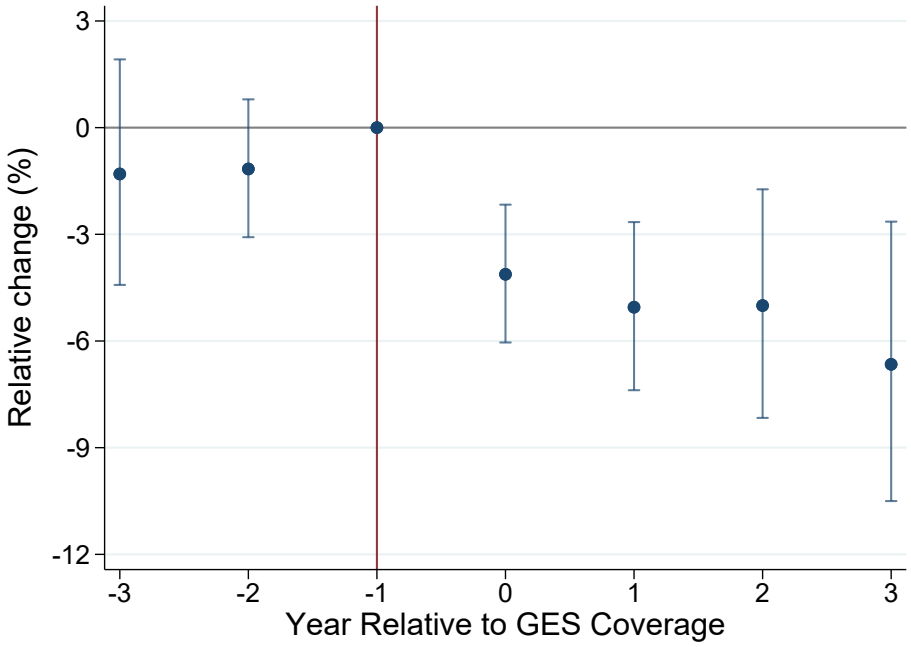
(b) 2005 Expansion

(c) 2006 Expansion

(d) 2007 Expansion

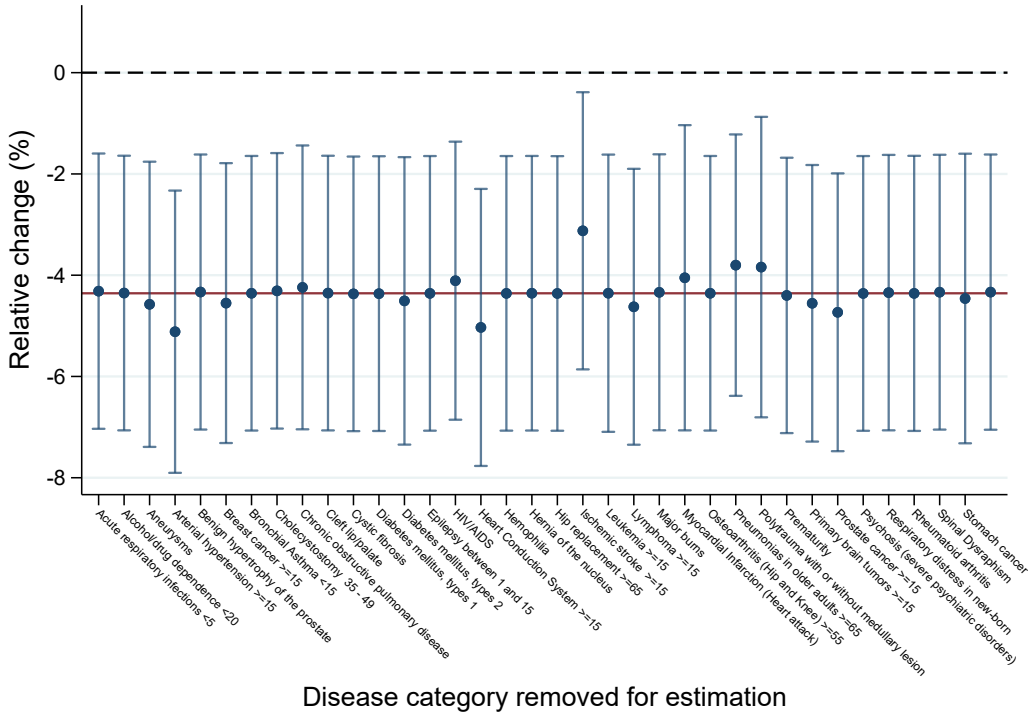
Notes: This figure shows the growth in deaths for both the diseases covered by each GES expansion and the diseases never covered by the GES reform. All growths in deaths are reported in percentages and calculated with respect to the year before each expansion. The vertical solid yellow line represents one year before the expansion.

Figure 2: Event study: GES impact on deaths



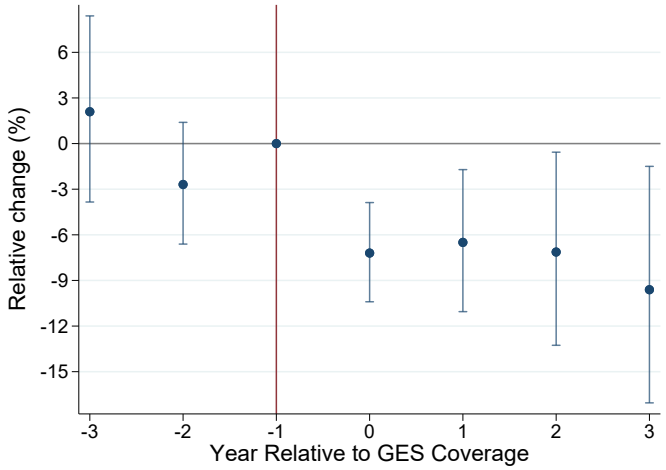
Notes: This figure shows the results obtained from estimating the dynamic difference-in-differences presented in equation (3) using the count of deaths as the dependent variable in a Poisson regression. All regressions control for disease-age cell and year-fixed effects. Standard errors are clustered at the level of treatment: disease-age cell. The relative change reported in the y-axis corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta_k) - 1$. The interpretation of the value of a rate ratio of 1 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and, if less than 1, indicates a decreased risk for the covered group. Each RR is capturing the effect of each period relative to one year before each group of diseases started to be covered. 95% confidence intervals for RR are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression.

Figure 3: Sensitivity of the treatment effect to targeted diseases

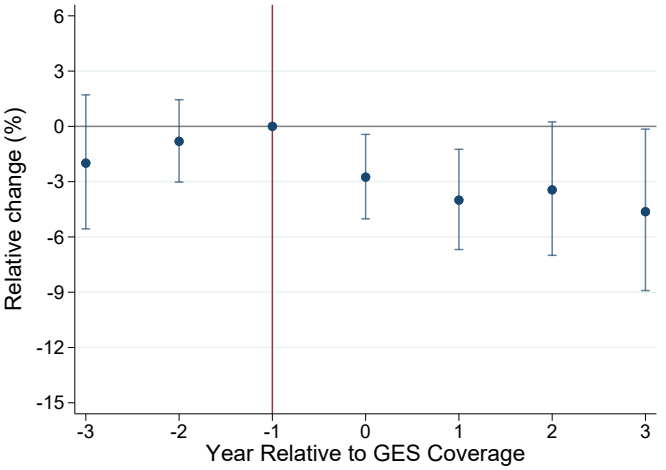


Notes: This figure shows the results obtained from estimating (several times) the dynamic difference-in-differences presented in equation (3) using the count of deaths as the dependent variable in a Poisson regression. Each point estimate and confidence interval comes from a regression in which we remove one treatment cell at a time, as indicated per the x-axis. All regressions control for disease-age cell and year-fixed effects. Standard errors are clustered at the level of treatment: disease-age cell. The relative change reported in the y-axis corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta_k) - 1$. The interpretation of the value of a rate ratio of 1 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and, if less than 1, indicates a decreased risk for the covered group. Each RR is capturing the effect of each period relative to one year before each group of diseases started to be covered. 95% confidence intervals for RR are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression.

Figure 4: Event study: GES impact on amenable and non-amenable deaths



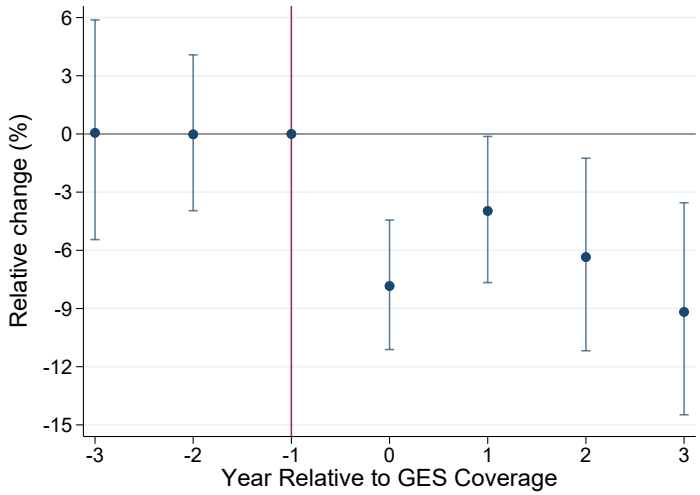
(a) Amenable



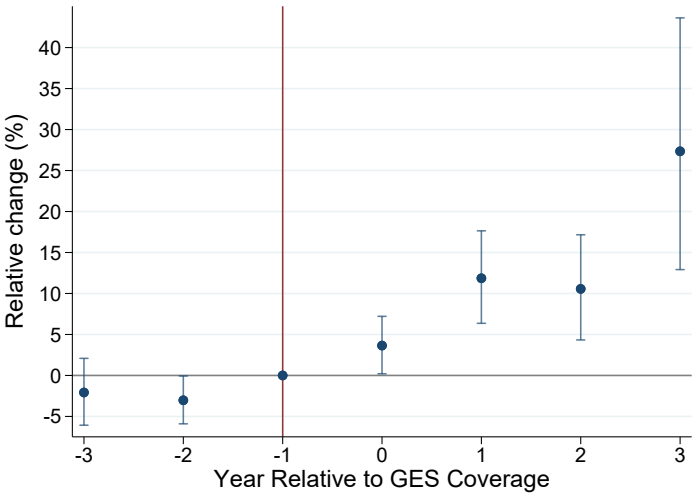
(b) Non-Amenable

Notes: This figure shows the results obtained from estimating the dynamic difference-in-differences presented in equation (3) using the count of deaths as the dependent variable in a Poisson regression. All regressions control for disease-age cell and year-fixed effects. Standard errors are clustered at the level of treatment: disease-age cell. The relative change reported in the y-axis corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta_k) - 1$. The interpretation of the value of a rate ratio of 1 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and, if less than 1, indicates a decreased risk for the covered group. Panel (a) shows the event study for the set of deaths amenable to health care (Nolte and McKee, 2011; Sommers et al., 2014). Panel (b) shows the event study for the set of deaths non-amenable to health care. Non-amenable deaths do not mean they cannot be impacted by health care, only that these deaths are likely to be less responsive to health care coverage than other causes. Each RR is capturing the effect of each period relative to one year before each group of diseases started to be covered. 95% confidence intervals for RR are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression. For details about the Amenable and Non-Amenable classification, see Appendix Table A.5.

Figure 5: Event Study: GES impact on in-hospital outcomes



(a) Deaths



(b) Surgeries

Notes: These figures show the results obtained from estimating the dynamic difference-in-differences presented in equation (3) using the count of in-hospital deaths and in-hospital surgeries as dependent variables in Poisson regressions. All regressions control for disease-age cell and year-fixed effects. Standard errors are clustered at the level of treatment: disease-age cell. The relative change reported in the y-axis corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta_k) - 1$. The interpretation of the value of a rate ratio of 1 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and, if less than 1, indicates a decreased risk for the covered group. Panel (a) shows the event study for the count of in-hospital deaths. Panel (b) shows the event study for the count of surgeries. Each RR captures the effect of each period relative to one year before each group of diseases started to be covered. 95% confidence intervals for RR are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression.

Table 1: GES impact on health outcomes

	Deaths			In-Hospital	
	All	Amenable	Non-Amenable	Deaths	Surgeries
	(1)	(2)	(3)	(4)	(5)
After GES Expansion	-0.044***	-0.071***	-0.028*	-0.069***	0.163***
	(0.014)	(0.026)	(0.016)	(0.020)	(0.033)
# Counts of dep. var	521,300	96,966	424,334	173,263	790,512
# Counts of dep. var covered by GES (as of 2003)	29,331	7,693	21,638	7,942	14,202
Total No. disease-age cells (obs.)	99,146	18,236	80,910	81,745	107,447

Notes: This table shows the results obtained from estimating the staggered difference-in-differences presented in equation (1) using Poisson regressions. All regressions control for disease-age cell and year-fixed effects. Standard errors are clustered at the level of treatment: disease-age cell. *After GES Expansion* corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta)-1$. The interpretation of the value of a rate ratio of 1 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and, if less than 1, indicates a decreased risk for the covered group. The RRs captures the average effect for all groups of diseases after they started to be covered. Non-amenable deaths do not mean they cannot be impacted by health care, only that these deaths are likely to be less responsive to health care coverage than other causes. The Poisson estimation drops disease-age cells with all zero outcomes in the study period. Standard errors for RR are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table 2: GES impact on health outcomes by type of health care provider

	All	Type of Hospital	
	inpatients	Public	Private
	(1)	(2)	(3)
Panel A: In-hospital Deaths			
After GES Expansion	-0.069***	-0.073***	-0.025
	(0.020)	(0.021)	(0.029)
# Deaths	173,263	155,379	17,884
# Deaths Covered (as of 2003)	7,942	7,110	832
Total No. disease-age cells (obs.)	81,745	78,220	30,880
Panel B: Surgeries			
After GES Expansion	0.163***	0.230***	0.008
	(0.033)	(0.037)	(0.030)
# Surgeries	790,512	563,503	227,009
# Surgeries Covered (as of 2003)	14,202	10,482	3,720
Total No. disease-age cells (obs.)	107,447	96,354	74,559

Notes: This table shows the results obtained from estimating the staggered difference-in-differences presented in equation (1) using Poisson regressions on inpatient records. All regressions control for disease-age cell and year-fixed effects. Standard errors are clustered at the level of treatment: disease-age cell. *After GES Expansion* corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta) - 1$. The interpretation of the value of a rate ratio of 1 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and if less than 1 indicates a decreased risk for the covered group. The RRs captures the average effect for all groups of diseases after they started to be covered. The Poisson estimation drops disease-age cells with all zero outcomes in the study period. Panel (a) shows the RR for the count of in-hospital deaths. Panel (b) shows the RR for the count of surgeries. Standard errors for RR are computed using the method for univariate transformations on the coefficient estimated from the Poisson regression. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table 3: GES impact on health outcomes by demographics

	Sex		Age Group		
	Female	Male	0-49	50-79	80+
	(1)	(2)	(3)	(4)	(5)
Panel A: Deaths					
After GES Expansion	-0.052***	-0.038***	-0.082***	-0.047**	-0.022
	(0.017)	(0.014)	(0.022)	(0.018)	(0.029)
# Deaths	226,327	294,973	89,850	252,845	178,605
# Deaths Covered (as of 2003)	13,499	15,832	2,459	15,362	11,510
Total No. disease-age cells (obs.)	77,145	80,558	42,145	36,415	20,586
Panel B: Surgeries					
After GES Expansion	0.151***	0.198***	0.211***	0.078**	0.186***
	(0.030)	(0.041)	(0.046)	(0.033)	(0.071)
# Surgeries	398,254	392,258	473,906	282,943	33,663
# Surgeries Covered (as of 2003)	7,005	7,197	7,119	6,097	986
Total No. disease-age cells (obs.)	86,310	86,171	58,294	34,892	14,261

Notes: This table shows the results obtained from estimating the staggered difference-in-differences presented in equation (1) using Poisson regressions. All regressions control for disease-age cell and year-fixed effects. Standard errors are clustered at the level of treatment: disease-age cell. *After GES Expansion* corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta) - 1$. The interpretation of the value of a rate ratio of 1 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and, if less than 1, indicates a decreased risk for the covered group. The RRs captures the average effect for all groups of diseases after they started to be covered. The Poisson estimation drops disease-age cells with all zero outcomes in the study period. Panel (a) shows the RR for the count of deaths. Panel (b) shows the RR for the count of surgeries. Standard errors for RR are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table 4: GES impact on health outcomes by major geographic areas

	North	Center	Metro	Center-South	South	Austral
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Deaths						
After GES Expansion	-0.013	-0.069***	-0.003	-0.077***	-0.068***	-0.057
	(0.024)	(0.018)	(0.016)	(0.018)	(0.018)	(0.043)
# Deaths	34,038	80,661	192,498	132,338	73,371	8,394
# Deaths Covered (as of 2003)	1,681	4,663	10,891	7,542	4,113	441
Total No. disease-age cells (obs.)	38,133	52,524	73,654	61,897	50,021	18,621
Panel B: Surgeries						
After GES Expansion	0.128***	0.181***	0.155***	0.211***	0.083	0.113
	(0.043)	(0.039)	(0.047)	(0.030)	(0.060)	(0.096)
# Surgeries	56,474	137,197	332,623	187,852	62,470	11,551
# Surgeries Covered (as of 2003)	733	2445	6055	3044	1653	251
Total No. disease-age cells (obs.)	44,030	57,489	86,038	68,161	44,694	20,523

This table shows the results obtained from estimating the staggered difference-in-differences presented in equation (1) using Poisson regressions. All regressions control for disease-age cell and year-fixed effects. Standard errors are clustered at the level of treatment: disease-age cell. *After GES Expansion* corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta) - 1$. The interpretation of the value of a rate ratio of 0 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and, if less than 1, indicates a decreased risk for the covered group. The RRs capture the average effect for all groups of diseases after they started to be covered. The Poisson estimation drops disease-age cells with all zero outcomes in the study period. Panel (a) shows the RR for the count of deaths. Panel (b) shows the RR for the count of surgeries. Standard errors for RR are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression. Geographic Areas are administrative regions grouped using the Ministry of Science and Technology definition. North: Arica y Parinacota, Tarapacá, Antofagasta, and Atacama; Center: Coquimbo and Valparaíso; Metro: Metropolitan Region; Center-South: O'Higgins, Maule, Ñuble and Biobío; South: La Araucanía, Los Ríos and Los Lagos. Austral: Aysen and Magallanes. The Metro area represents almost 40% of the population and includes the capital city. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

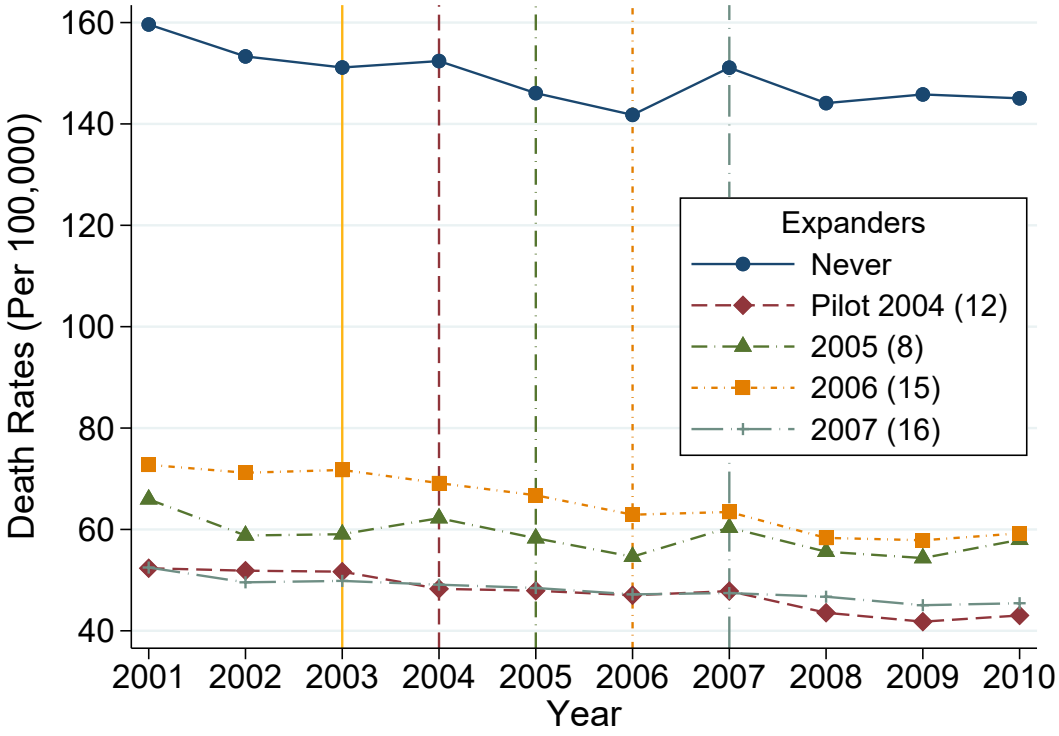
Table 5: GES impact on household finance

	Medical Visits			Medical Expenditures			Indebtedness		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Panel A: Extensive Margin (Any)									
Declared to have benefited from GES coverage	0.043*** (0.003)	0.039*** (0.004)	0.028*** (0.007)	-0.170*** (0.007)	-0.130*** (0.008)	-0.073*** (0.011)	-0.030*** (0.006)	0.002 (0.006)	0.007 (0.008)
# Observations	18,769	15,954	14,238	18,769	15,954	14,238	18,769	15,954	14,238
# Individuals	10165	7489	5773	9402	7000	4534	10619	7489	6418
Mean Dep. Variable	0.852	0.830	0.845	0.493	0.503	0.487	0.155	0.151	0.153
Panel B: Intensive Margin (Log of)									
Declared to have benefited from GES coverage	0.477*** (0.017)	0.397*** (0.019)	0.247*** (0.026)	-0.504*** (0.066)	-0.486*** (0.073)	-0.333** (0.157)	-0.084 (0.065)	-0.048 (0.076)	0.243 (0.165)
# Observations	16,937	14,122	11,981	4,436	3,749	1,590	3,025	2,526	929
# Individuals.	10165	7489	5773	10165	7489	5773	10165	7489	5773
Mean Dep. Variable	11.95	11.73	11.76	644.6	635.6	784	4459	4692	5092
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Controls	No	Yes	Yes	No	Yes	Yes	No	Yes	Yes
Person FE	No	No	Yes	No	No	Yes	No	No	Yes

Notes: This table uses panel data from the EPS survey for the years 2009, 2012, and 2015. We focus on respondents who declare ever being diagnosed with asthma, depression, diabetes, hypertension, heart-related problems, cancer, arthritis or arthrosis, renal disease, stroke, mental illness, or HIV/AIDS. The explanatory variable is an indicator that equals 1 if the respondent reports that GES covered her illness. *Medical Visits* corresponds to the number of medical visits during the past two years. *Medical Expenditures* corresponds to the total out-of-pocket medical expenses per visit among those who had at least one medical visit during the past two years. *Indebtedness* corresponds to loans from banks or financial institutions (excluding auto loan and education loan debt) and loans from friends and informal institutions. Both *Medical Expenditures* and *Indebtedness* are measured in 2022 USD. Controls include age, age squared, self-perceived health status, type of insurance, gender, and education indicators of any college and any high school. Standard errors are clustered at the respondent level. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

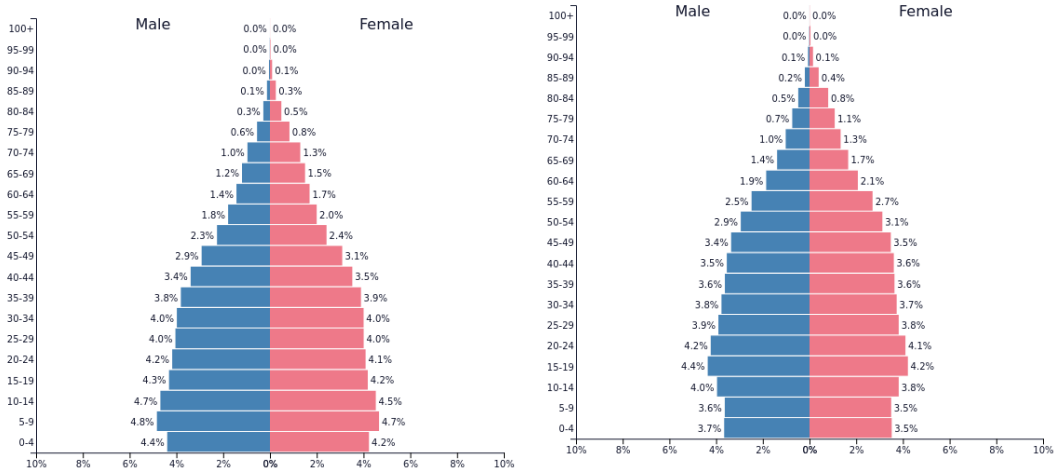
Appendix A: Additional Figures and Tables

Figure A.1: Age Standardized Cause-Specific Death Rate



Notes: This figure shows the decrease in cause-specific death rates for each group of treated diseases; in this case, all rates are standardized using the 2001 age distribution to account for the age structure of the population. To adjust death rates, we proceed in the following way: i) we calculate crude death rates for age x as the number of deaths for each group of disease-population of age x divided by the population of age x, where x stands for 5-year age groups (i.e., 0, 1-4 years, 5-9 years,..., 85-99 years, and greater than 100 years); ii) we multiply the ratio obtained in step i) by the population share in 2001; and finally, iii) we sum across all the weighted age-specific shares obtained in step ii). The number of treated diseases in each group ("Expanders") is listed in parentheses. Vertical solid yellow lines represent one year before the expansion. Vertical dashed lines represent the year of each of the expansions. All is based on data from the Death Registry, Vital Statistics, Census, and GES eligibility rules.

Figure A.2: Population pyramids



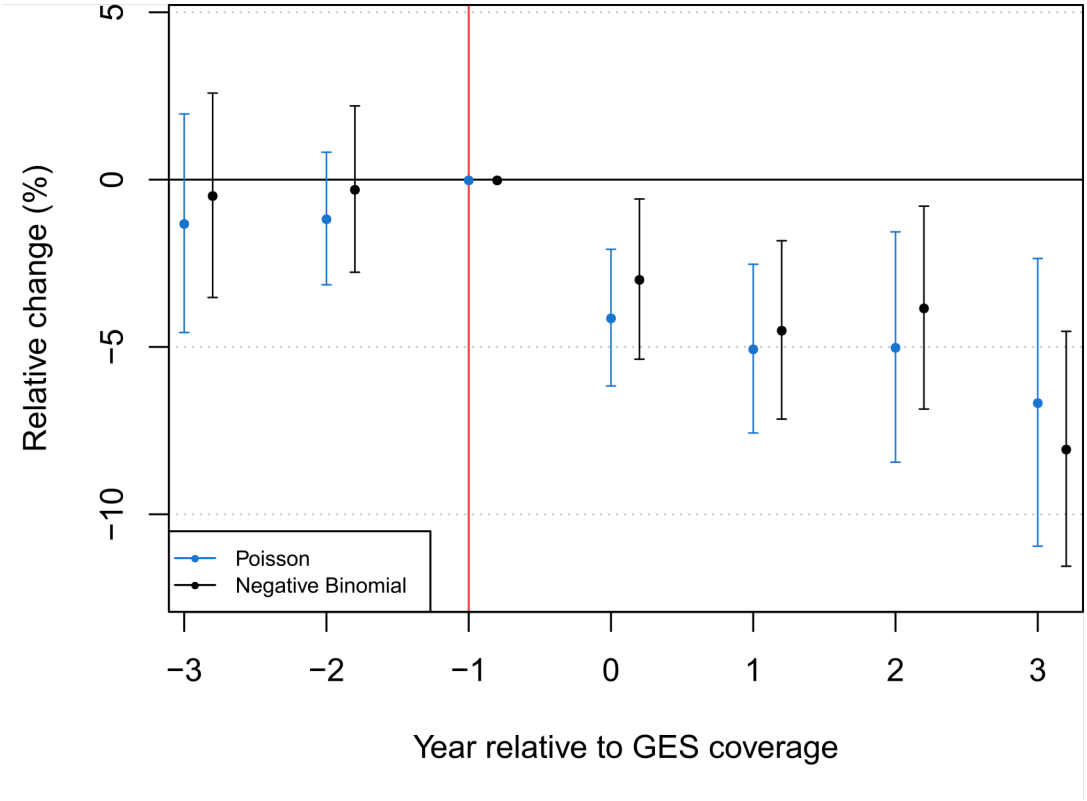
(a) 2000, Pop: 15,32,350

(b) 2010, Pop: 17,062,531

(c) 2020, Pop 19,611,208

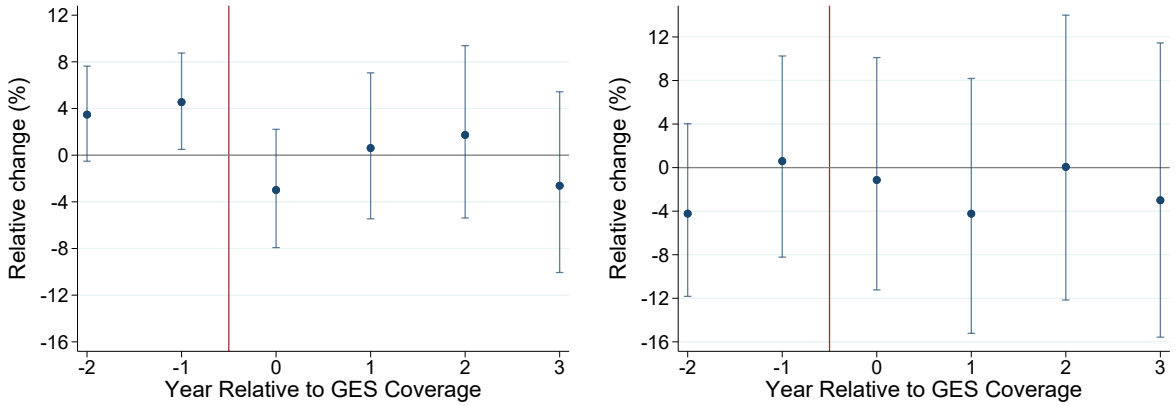
Notes: This figure shows population pyramids for Chile in the years 2000, 2010, and 2020. Source: Pyramids.net.

Figure A.3: Event study: GES impact on deaths alternative models



Notes: This figure shows the results obtained from estimating the dynamic difference-in-differences presented in equation (3) using the count of deaths as the dependent variable in a Poisson compared to a Negative Binomial regression. All regressions control for disease-age cell and year-fixed effects. Standard errors are clustered at the level of treatment: disease-age cell. The relative change reported in the y-axis corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta_k) - 1$. The interpretation of the value of a rate ratio of 1 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and, if less than 1, indicates a decreased risk for the covered group. Each RR is capturing the effect each period relative to one year before each group of diseases started to be covered. 95% confidence intervals for RR are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression. The negative Binomial model was estimated using R's fixest package

Figure A.4: Event Study: GES impact on deaths, by expansion, using alternative estimation method



(a) 2004 Expansion

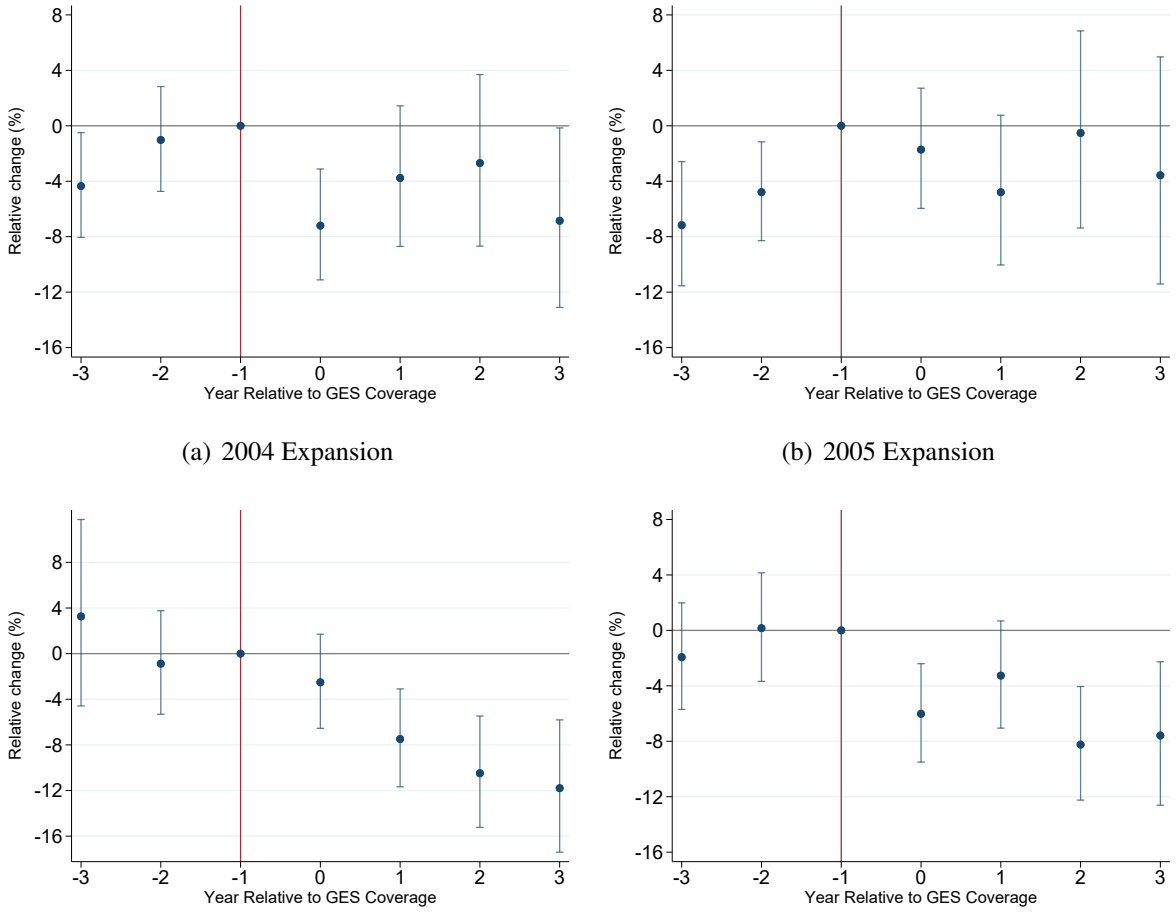
(b) 2005 Expansion

(c) 2006 Expansion

(d) 2007 Expansion

Notes: These figures display the point estimates and 95% confidence intervals obtained from a Poisson model that is robust even if the treatment effects are heterogeneous over time or across groups. Specifically, we follow Wooldridge (2021) and estimate a Poisson regression saturated with the interaction of all treatment cohorts (GES expansions) and event time dummies. The regression includes cell and year-fixed effects. The relative change reported in the y-axis corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta_k) - 1$. The interpretation of the value of a rate ratio of 1 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and, if less than 1, indicates a decreased risk for the covered group. Coefficients capture the effect of each period relative to one year before each group of diseases started to be covered. 95% confidence intervals for RR are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression.

Figure A.5: Event Study: GES impact on deaths, by expansion



(a) 2004 Expansion

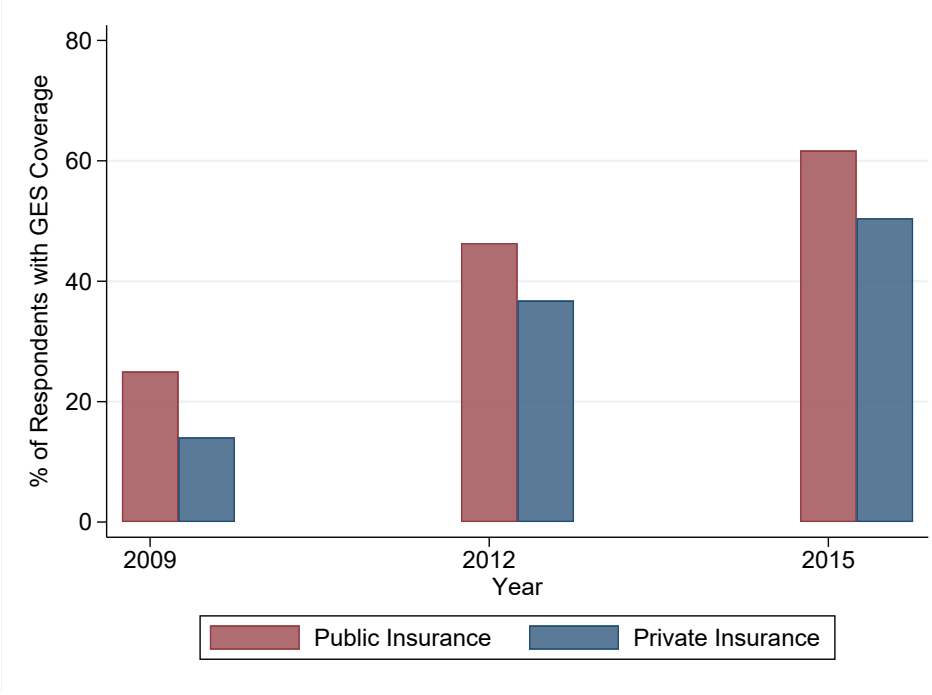
(b) 2005 Expansion

(c) 2006 Expansion

(d) 2007 Expansion

Notes: These figures show the coefficients obtained from estimating the dynamic difference-in-differences presented in equation (3). Each regression considers each expansion independently using never treated cells. All regressions control for disease-age cell and year-fixed effects. Standard errors are clustered at the level of treatment: disease-age cell. The relative change reported in the y-axis corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta_k) - 1$. The interpretation of the value of a rate ratio of 1 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and, if less than 1, indicates a decreased risk for the covered group. Coefficients capture the effect of each period relative to one year before each group of diseases started to be covered. 95% confidence intervals for RR are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression.

Figure A.6: Share of respondents with GES coverage



Notes: This figure uses panel data from the EPS survey for the years 2009, 2012, and 2015. We focus on respondents who declare ever being diagnosed with asthma, depression, diabetes, hypertension, heart-related problems, cancer, arthritis or arthrosis, renal disease, stroke, mental illness, or HIV AIDS. Among them, we display the share (in percentual points) that reports that their illness was covered by GES, by type of insurance.

Table A.1: Health related problems: Pilot 2004

Health Related Problem	Deaths	%
Myocardial Infarction (Heart attack)	58,469	71.41
Breast cancer (15+ years old)	11,634	14.21
Lymphoma (15+ years old)	5,708	6.97
HIV/AIDS	4,160	5.08
Testicular cancer (15+ years old)	973	1.19
Diabetes mellitus, types 1	280	0.34
Psychosis (severe psychiatric disorders)	283	0.35
Spinal Dysraphism	214	0.26
Hip replacement (65+ years old)	90	0.11
Cleft lip/palate	63	0.08
Total	81,874	100.00

Notes: This table shows deaths for the health-related problems included in the 2004 pilot between 2001 and 2010. Diseases with zero deaths in the period are not included.

Table A.2: Health related problems: 2005 Expansion

Health Related Problem	Deaths	%
Pneumonias in older adults (65+ years old)	28,605	27.63
Diabetes mellitus, types 2	27,795	26.84
Arterial hypertension (15+ years old)	27,385	26.45
Heart Conduction System (15+ years old)	15,689	15.15
Prematurity	2,853	2.76
Acute respiratory infections (5- years old)	1,090	1.05
Epilepsy (between 1 and 15 years old)	122	0.12
Total	103,539	100.00

Notes: This table shows deaths for the health-related problems included in the 2005 Expansion between 2001 and 2010. Diseases with zero deaths in the period are not included.

Table A.3: Health related problems: 2006 Expansion

Health Related Problem	Deaths	%
Ischemic stroke (15+ years old)	35,199	30.95
Stomach cancer	31,207	27.44
Chronic obstructive pulmonary disease	27,809	24.45
Prostate cancer (15+ years old)	15,667	13.78
Respiratory distress in new-born	1,603	1.41
Cholecystostomy (between 35 to 49 years old)	1,495	1.31
Benign hypertrophy of the prostate	700	0.62
Hemophilia	32	0.03
Bronchial Asthma (15- years old)	10	0.01
Total	113,722	100.00

Notes: This table shows deaths for the health-related problems included in the 2006 Expansion between 2001 and 2010. Diseases with zero deaths in the period are not included.

Table A.4: Health related problems: 2007 Expansion

Health Related Problem	Deaths	%
Polytrauma with or without medullary lesion	42,646	52.99
Aneurysms	22,814	28.35
Primary brain tumors (15+ years old)	5,555	6.90
Leukemia (15+ years old)	5,370	6.67
Major burns	2,881	3.58
Rheumatoid arthritis	1,042	1.29
Cystic fibrosis	154	0.19
Alcohol/drug dependence (20- years old)	15	0.02
Osteoarthritis (Hip and Knee) (55+ years old)	3	0.00
Total	80,480	100.00

Notes: This table shows the health-related problems included in the 2007 Expansion between 2001 and 2010. Diseases with zero deaths in the period are not included.

Table A.5: Definitions of deaths amenable to health care

Condition(s)	**ICD-10 Codes**	***Nolte & McKee***	***Sommers***	***Ours***
Infectious & Parasitic Diseases (ALL)	A00-B99		X	
-Tuberculosis	A16-19, B90	X	X	X
-Other specific infections (diphtheria, tetanus, septicemia, poliomyelitis, whooping cough, measles)	A00-09 (age 0-14), A33, A35-36, A37 (age 0-14), A40-41, A80, B05 (age 1-14)	X	X	X
Neoplasms (ALL)	C00-D48		X	
-Malignant neoplasm of colon and rectum	C18-C21	X	X	X
-Malignant neoplasm of skin	C44	X	X	X
-Malignant neoplasm of breast	C50	X	X	X
-Malignant neoplasm of cervix or uterus	C54-55 (age 0-44)	X	X	X
-Malignant neoplasm of testis	C62	X	X	X
-Hodgkin's disease	C81	X	X	X
-Leukemia	C91-C95 ($\leq$ 45 years)	X	X	X
Disorders of thyroid gland	E00-E07	X	X	X
Diabetes Mellitus	E10-E14	X	X	X
Epilepsy	G40-G41	X	X	X
Chronic rheumatic heart diseases	I05-I09	X	X	X
Hypertensive diseases	I10-I13, I15	X	X	X
Ischemic heart diseases	I20-I25	X	X	X
Cardiomyopathy	I42		X	X
Atrial fibrillation and flutter	I48		X	X
Other cardiac arrhythmias	I49		X	X
Heart failure	I50		X	X
Cerebrovascular diseases	I60-I69	X	X	X
All respiratory diseases	J00-J98		X	
-Respiratory diseases (excl. pneumonia, influenza)	J00-09, J20-99 (age 1-14)	X		X
-Respiratory diseases	J10-18	X		X
Gastric and duodenal ulcers	K25-K27	X	X	X
Gastrojejunal ulcers	K28		X	X
Diseases of appendix	K35-K38	X	X	X
Hernia	K40-K46	X	X	X
Diseases of gallbladder and biliary tract	K80-K83	X	X	X
Acute pancreatitis	K85		X	X
Infections of the skin and subcutaneous tissue	L00-L08		X	X
Infectious arthropathies	M00-M02		X	X
Glomerular diseases	N00-N07	X	X	X
Renal tubulo-interstitial diseases	N10-N15		X	X
Renal failure	N17-N19	X	X	X
Unspecified contracted kidney, small kidney unknown cause	N26-N27	X		X
Hyperplasia of prostate	N40	X		X
Pregnancy, childbirth and the puerperium	O00-O99	X	X	X
Perinatal deaths, all causes (excl. stillbirths)	P00-P96	X		X
Congenital malformations	Q20-28	X		X
Misadventures to patients during surgical and medical care	Y60-Y69, Y83-Y84	X	X	X

Notes: This table shows the classification of conditions as amenable to health care, according to different authors. *Nolte and McKee* corresponds to the classification used in Nolte and McKee, 2011, *Sommers* corresponds to the classification used in Sommers et al., 2014, and *Ours* corresponds to the classification used in this paper; which is as a combination of Nolte and McKee, 2011 and Sommers et al., 2014.

Table A.6: Targeted diseases, targeted cells (disease-age groups), and the total number of deaths

	Deaths			In-Hospital	
	All	Amenable	Non-Amenable	Deaths	Surgeries
	(1)	(2)	(3)	(4)	(5)
Panel A: Diseases (ICD-10)					
Total	1,027	317	944	1,017	1,002
Covered	315	132	284	308	309
Uncovered	763	227	668	756	742
Panel B: Disease-Age Cells					
Total	10,982	2,057	8,925	9,037	11,768
Covered	3,558	778	2,780	2,875	3,411
Uncovered	7,424	1,279	6,145	6,162	8,357
Panel C: # Deaths					
Total	521,300	96,966	424,334	173,263	790,512
Covered	264,974	62,070	202,904	77,206	195,958
Uncovered	256,326	34,896	221,430	96,057	594,554
Total No. of disease-age cells (obs.)	99,146	18,236	80,910	81,745	107,447

Notes: This table describes the sample in terms of the number of targeted diseases (ICD-10), targeted group of disease-age (ICD-10-Age) cells, and the total number of deaths. The sample only includes diseases covered in the 2004 Pilot, in 2005, 2006, and 2007 expansions, and the never-covered diseases. Panel A shows counts for diseases. In this case, *Covered* and *Uncovered* do not add up since some diseases are in both groups because the coverage is for a specific group of ages. Panel B shows counts for disease-age cells. In this case, the number of disease-age cells is not balanced for some groups of ages. This is because Poisson estimation drops disease-age cells (obs.) with all zero outcomes in the period of study. Additionally, some groups of ages are not considered because they are covered as part of later expansions outside the window used in our study, e.g Bronchial Asthma was covered by the 2006 expansions for people below 15, but in 2010 expanded the age coverage for those above 15. Panel C shows counts for the total number of deaths in our sample. The total number of disease-age cells (obs.) is the result of the covered cells in the 7-year window and the uncovered cells in the period of study.

Table A.7: Deaths covered by ICD10 chapters

Chapters	All		Never Covered		Covered in Expansion: 2004		2005		2006		2007	
	N	%	N	%	N	%	N	%	N	%	N	%
Diseases of the circulatory system	184,292	35.35	73,196	39.72	41,358	22.44	29,084	15.78	24,402	13.24	16,252	8.82
Diseases of the respiratory system	61,987	11.89	22,187	35.79	-	-	20,205	32.60	19,595	31.61	-	-
Diseases of the digestive system	61,552	11.81	61,497	99.91	-	-	-	-	55	0.09	-	-
Neoplasms	60,535	11.61	6,415	10.60	12,231	20.20	-	-	33,945	56.07	7,944	13.12
Injury, poisoning and certain other consequences of external causes	58,608	11.24	26,512	45.24	-	-	-	-	-	0.00	32,096	54.76
Endocrine, nutritional and metabolic diseases	27,324	5.24	7,398	27.08	219	0.80	19,589	71.69	-	0.00	118	0.43
Certain infectious and parasitic diseases	15,756	3.02	12,754	80.95	2,948	18.71	54	0.34	-	0.00	-	-
Diseases of the genitourinary system	14,315	2.75	13,758	96.11	-	-	-	-	557	3.89	-	-
Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified	7,249	1.39	7,249	100.00	-	-	-	-	-	0.00	-	-
Diseases of the nervous system	7,209	1.38	7,108	98.60	13	0.18	88	1.22	-	0.00	-	-
Certain conditions originating in the perinatal period	5,391	1.03	2,612	48.45	-	0.00	1,823	33.82	956	17.73	-	-
Congenital malformations, deformations and chromosomal abnormalities	5,274	1.01	4,873	92.40	186	3.53	-	-	215	4.08	-	-
Mental, Behavioral and Neurodevelopmental disorders	3,741	0.72	3,560	95.16	176	4.70	-	-	-	0.00	5	0.13
Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	2,928	0.56	2,896	98.91	7	0.24	-	-	25	0.85	-	-
Diseases of the skin and subcutaneous tissue	2,506	0.48	2,506	100.00	-	0.00	-	-	-	0.00	-	-
Diseases of the musculoskeletal system and connective tissue	2,353	0.45	1,525	64.81	50	2.12	-	-	-	0.00	778	33.06
Pregnancy, childbirth and the puerperium	276	0.05	276	100.00	-	0.00	-	-	-	0.00	-	-
Diseases of the eye and adnexa	4	0.00	4	100.00	-	0.00	-	-	-	0.00	-	-
Total	521,300	100	256,326	49.17	57,188	10.97	70,843	13.59	79,750	15.30	57,193	10.97

Notes: This table shows the number and percentage of deaths (from the deaths records), by reform coverage and ICD-10 chapter. We list the chapter's title according to the international version of the ICD-10, grouping deaths in our sample by the code range of each chapter.

Table A.8: Deaths covered by year

	All		Never Covered		Covered in Expansion:							
					2004		2005		2006		2007	
Year	N	%	N	%	N	%	N	%	N	%		
2001	31,707	6.08	23,877	75.31	7,830	0.00	-	0.00	-	0.00	-	0.00
2002	40,757	7.82	23,626	57.97	8,017	22.36	9,114	22.36	-	0.00	-	0.00
2003	53,427	10.25	24,096	45.10	8,261	17.84	9,534	17.84	11,536	21.59	-	0.00
2004	62,829	12.05	25,036	39.85	7,965	16.56	10,404	16.56	11,505	18.31	7,919	12.60
2005	62,535	12.00	24,766	39.60	8,171	16.17	10,115	16.17	11,482	18.36	8,001	12.79
2006	61,961	11.89	24,747	39.94	8,256	15.80	9,791	15.80	11,185	18.05	7,982	12.88
2007	67,057	12.86	27,206	40.57	8,688	16.77	11,247	16.77	11,669	17.40	8,247	12.30
2008	56,484	10.84	26,546	47.00	-	18.83	10,638	18.83	11,017	19.50	8,283	14.66
2009	47,345	9.08	27,770	58.65	-	0.00	-	0.00	11,356	23.99	8,219	17.36
2010	37,198	7.14	28,656	77.04	-	0.00	-	0.00	-	0.00	8,542	22.96
Total	521,300	100	256,326	49.17	57,188	10.97	70,843	13.59	79,750	15.30	57,193	10.97

Notes: This table shows the number and percentage of deaths (from the deaths records), by reform coverage and year.

Table A.9: Deaths covered by age group

	All		Never Covered		Covered in Expansion:							
					2004		2005		2006		2007	
Agr Group	N	%	N	%	N	%	N	%	N	%	N	%
0-14	16,564	3.18	11,129	67.19	197	1.19	2615	15.79	1,265	7.64	1,358	8.20
15-49	73,286	14.06	39,961	54.53	7602	10.37	1749	2.39	3,504	4.78	20,470	27.93
50-54	22,464	4.31	12,275	54.64	3107	13.83	1192	5.31	1,921	8.55	3,969	17.67
55-59	27,969	5.37	14,986	53.58	4013	14.35	2033	7.27	2,946	10.53	3,991	14.27
60-64	35,865	6.88	18,405	51.32	5078	14.16	3198	8.92	4,808	13.41	4,376	12.20
65-69	43,777	8.40	20,802	47.52	5963	13.62	5185	11.84	7,309	16.70	4,518	10.32
70-74	55,351	10.62	25,095	45.34	7345	13.27	7841	14.17	10,658	19.26	4,412	7.97
75-79	67,419	12.93	29,973	44.46	7853	11.65	10759	15.96	14,012	20.78	4,822	7.15
80-84	68,060	13.06	31,035	45.60	6754	9.92	12192	17.91	13,906	20.43	4,173	6.13
85-89	58,242	11.17	27,259	46.80	5236	8.99	11751	20.18	11,046	18.97	2,950	5.07
90-94	36,864	7.07	17,492	47.45	2971	8.06	8572	23.25	6,256	16.97	1,573	4.27
95-99	12,967	2.49	6,573	50.69	913	7.04	3114	24.01	1,860	14.34	507	3.91
100+	2,472	0.47	1,341	54.25	156	6.31	642	25.97	259	10.48	74	2.99
Total	521,300	100.00	256,326	49.17	57,188	10.97	70843	13.59	79,750	15.30	57,193	10.97

Note: This table shows the number and percentage of deaths (from the deaths records), by reform coverage and age group. The 0-14 age group was combined because of the few deaths reported in the age groups used in the main analysis: newborns, 1-4 years, 5-9, and 10-14.

Table A.10: Robustness of GES impact on deaths to alternative models

	Non-linear		Linear	
	Poisson	Neg-Bin	Log	IHS
	(1)	(2)	(3)	(4)
Panel A: All Diseases				
After GES Expansion	-0.044***	-0.045***	-0.011*	-0.015*
	(0.014)	(0.012)	(0.006)	(0.008)
Observations	99,146	99,146	99,146	99,146
Panel B: Ever GES				
After GES Expansion	-0.040***	-0.030*	-0.033***	-0.041***
	(0.010)	(0.012)	(0.012)	(0.015)
Observations	24,906	24,906	24,906	24,906

Notes: This table shows the results obtained from variations of the staggered difference-in-differences model given by equation (1). Column (1) presents the estimates from our main model while column (2) presents the estimates from a negative binomial regression. Columns (3) and (4) show the results obtained from linear models (OLS). Log stands for a logarithmic transformation of the outcome as *Ln*(deaths+1). IHS stands for the Inverse Hyperbolic Sine transformation of the outcome. For the Poisson model (column 1), *After GES Expansion* corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta) - 1$. All regressions control for disease-age cell and year-fixed effects using the main sample. Standard errors are clustered at the level of treatment: disease-age cell. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table A.11: Pre-treatment characteristics between covered and non-covered cells

	Level (2001)		Growth (2001-2003)			
	GES	Non-GES	GES	Non-GES	$\hat{\beta}$	p-value
	(1)	(2)	(3)	(4)	(5)	(6)
% Public Insurance	0.297	0.282	0.371	0.320	0.051	.089
% Private Insurance	0.078	0.046	0.045	0.038	0.007	.680
% High School	0.449	0.268	0.002	0.011	-0.009	.757
% Female	0.328	0.469	0.031	0.006	0.024	.344
% Married	0.359	0.380	-0.051	-0.064	0.013	.653
% Rural	0.099	0.160	-0.025	-0.022	-0.004	.817
% North	0.085	0.078	-0.018	-0.008	-0.010	.547
% Centre	0.159	0.155	-0.006	0.010	-0.016	.489
% Metro	0.462	0.367	0.019	-0.000	0.019	.524
% Center-South	0.200	0.245	-0.020	0.001	-0.021	.426
% South	0.084	0.137	0.013	-0.004	0.016	.371
% Austral	0.010	0.018	0.012	0.001	0.012	.164

Notes: This table shows pre-treatment characteristics from the death records of covered and non-covered cells. Columns (1) and (2) show the average of each characteristic among covered and non-covered cells in 2001. Columns (3) and (4) show the linear growth between 2001 and 2003 of each characteristic among covered and non-covered cells. Column (5) shows the coefficient obtained from a linear projection of growth on an indicator of GES coverage. Column (6) are the p-values associated with the column (5) coefficients. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table A.12: GES impact on deaths by GES expansion and among ever GES

	Analysis Sample				
	Ever	Only Expansion:			
	GES	2004	2005	2006	2007
	(1)	(2)	(3)	(4)	(5)
After GES Expansion	-0.040***	-0.034	0.014	-0.089***	-0.058***
	(0.010)	(0.025)	(0.036)	(0.025)	(0.017)
# Deaths	264,974	313,514	327,169	336,076	313,519
# Deaths Covered (as of 2003)	29,331	8,261	10,404	11,482	7,982
Total No. disease-age cells (obs.)	24,906	78,517	79,119	76,879	87,351

Notes: This table shows the coefficients obtained from estimating the staggered difference-in-differences presented in equation (1) using Poisson regressions on the count of deaths. Column (1) only considers ever-covered diseases and leverages differences in the timing of adoption among them for identification. Columns (2)-(5) consider the impact of each expansion separately, using never covered diseases as controls. All regressions control for disease-age cell and year-fixed effects. Standard errors are clustered at the level of treatment: disease-age cell. *After GES Expansion* corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta) - 1$. The interpretation of the value of a rate ratio of 1 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and, if less than 1, indicates a decreased risk for the covered group. The Poisson estimation drops disease-age cells with all zero outcomes in the period of study. Standard errors for RR are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table A.13: GES impact on deaths using alternative amenable death classifications

	Ours		Nolte & McKee (2011)		Tobias & Yeh (2009)		European Union (2015)	
	Amenable	Non-Amenable	Amenable	Non-Amenable	Amenable	Non-Amenable	Amenable	Non-Amenable
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
After GES Expansion	-0.071***	-0.028*	-0.063**	-0.029*	-0.047**	-0.025	-0.057**	-0.026
	(0.026)	(0.016)	(0.026)	(0.016)	(0.022)	(0.018)	(0.024)	(0.017)
# Deaths	96,966	424,334	86,324	434,976	134,481	386,819	106,780	414,520
# Deaths Covered (as of 2003)	7,693	21,638	7,121	22,210	12,741	16,590	8,807	20,524
Total No. disease-age cells (obs.)	18,236	80,910	15,538	83,608	20,346	78,800	22,216	76,930

Notes: This table shows the coefficients obtained from estimating the staggered difference-in-differences presented in equation (1) using Poisson regressions for the count of amenable and non-amenable deaths, as classified by different authors. *Ours* corresponds to the classification used in our main analyses. All regressions control for disease-age cell and year-fixed effects. Standard errors are clustered at the level of treatment: disease-age cell. *After GES Expansion* corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta) - 1$. The interpretation of the value of a rate ratio of 1 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and, if less than 1, indicates a decreased risk for the covered group. The coefficients capture the average effect for all groups of diseases after they started to be covered. The Poisson estimation drops disease-age cells with all zero outcomes in the period of study. Standard errors for RR are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table A.14: GES impact on health outcomes by type of insurance

	Insurance		Type of Public Insurance				
	Private	Public	A	B	C	D	NA
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Panel A: In-Hospital Deaths							
After GES Expansion	-0.089***	-0.065***	-0.065**	-0.069***	-0.097**	-0.0531	-0.0548
	(0.026)	(0.021)	(0.025)	(0.025)	(0.040)	(0.0385)	(0.0759)
# Deaths	19,628	153,635	61,816	69,980	7,791	11,474	2,574
# Deaths Covered (as of 2003)	971	6,971	2,811	3,381	298	370	111
Total No. disease-age cells (obs.)	33,433	77,745	58,475	51,182	21,935	24,552	10,873
Panel B: Surgeries							
After GES Expansion	0.012	0.219***	0.302***	0.198***	0.230***	0.139***	-0.059
	(0.028)	(0.038)	(0.047)	(0.041)	(0.049)	(0.045)	(0.071)
# Deaths	209,559	580,953	204,198	202,431	72,784	84,651	16,889
# Deaths Covered (as of 2003)	3,760	10,442	3,582	3,874	1,206	1,262	518
Total No. disease-age cells (obs.)	74,652	96,949	73,305	69,077	43,510	49,304	23,069

Notes: This table shows the coefficients obtained from estimating the staggered difference-in-differences presented in equation (1) using Poisson regressions. All regressions control for disease-age cell and year-fixed effects. Standard errors are clustered at the level of treatment: disease-age cell. *After GES Expansion* corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta) - 1$. The interpretation of the value of a rate ratio of 1 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and, if less than 1, indicates a decreased risk for the covered group. The coefficients capture the average effect for all groups of diseases after they started to be covered. The Poisson estimation drops disease-age cells with all zero outcomes in the study period. Panel (a) shows the RR for the count of in-hospital deaths. Panel (b) shows the RR for the count of surgeries. Insurance information is only available from the inpatient records. Private and Public correspond to ISAPRE and FONASA, respectively. The type of Public Insurance corresponds to the four types of co-payment faced by the FONASA beneficiaries as a function of their income. Standard errors for RR are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Table A.15: GES impact by type of health care provider removing the 2004 (pilot) expansion

	All	Type of Hospital	
	inpatients	Public	Private
	(1)	(2)	(3)
Panel A: In-hospital Deaths			
After GES Expansion	-0.074***	-0.079***	-0.031
	(0.023)	(0.024)	(0.034)
# Deaths	161,269	145,224	16,045
# Deaths Covered (as of 2003)	6,078	5,541	537
Total No. disease-age cells (obs.)	78,343	75,042	29,291
Panel B: Surgeries			
After GES Expansion	0.178***	0.245***	0.017
	(0.037)	(0.040)	(0.035)
# Surgeries	776,790	554,386	222,404
# Surgeries Covered (as of 2003)	12,111	9,053	3,058
Total No. disease-age cells (obs.)	104,507	94,065	72,774

Notes: This table shows the results obtained from estimating the staggered difference-in-differences presented in equation (1) using Poisson regressions on inpatient records. All regressions control for disease-age cell and year-fixed effects. Standard errors are clustered at the level of treatment: disease-age cell. *After GES Expansion* corresponds to percent changes by subtracting 1 from the rate ratio (RR), i.e., $exp(\beta) - 1$. The interpretation of the value of a rate ratio of 1 indicates equal rates in the two groups (covered and non-covered), a rate ratio greater than 1 indicates an increased risk for the covered group, and if less than 1, indicates a decreased risk for the covered group. The RRs captures the average effect for all groups of diseases after they started to be covered. The Poisson estimation drops disease-age cells with all zero outcomes in the study period. Panel (a) shows the RR for the count of in-hospital deaths. Panel (b) shows the RR for the count of surgeries. Standard errors for RR are computed using the method for univariate transformations on the coefficient estimated from the Poisson regression. Significance levels: *** p<0.01, ** p<0.05, * p<0.1.

Appendix B: The Valuation of Lives Saved

In this appendix, we present a back-of-the-envelope calculation on the impact of the GES reform on life expectancy, and we also show a simple cost-benefit analysis based on estimates of the value of statistical life and the cost of the reform.

Impact on life expectancy: Life expectancy at birth was 77.33 years in 2003, the pre-reform year for which official data is reported in detail.[18] Thus, based on our estimates, we apply the relative decrease in deaths to the age-specific mortality rates from the life table and then recalculate life expectancy, finding that the reform led to an increase of 0.29 years in terms of life expectancy as of 2003.[19] Such a decline would have taken us forward close to the mortality conditions of 2005, when life expectancy was 77.78 years. Therefore, we can say that the progress in life expectancy, which would typically take two years, was achieved before it would have been without the reform.

Cost-Benefit Analysis: The value of a statistical life (VSL) might be helpful to guide policymakers in their analysis of the benefits of the reform. VSL represents risk-money trade-offs for small changes in risk. Thus estimates are usually based on the extra wages that workers receive for facing increased fatality risk at work. For instance, a worker who receives extra pay of $1,000 to face a risk of 1/10,000 has a value per unit risk (or VSL) of $1,000/(1/10,000) = $10 million, regardless of age (Viscusi, 2018). The VSL varies with countries' income levels, as do many other expenditures. For Chile, there are numbers ranging from $0,69 to $8,69 million depending on the method and purpose (Mardones and Riquelme, 2018; Parada-Contzen, 2019). Using Chile's median estimates - $USD 4,000,000-, which are similar to half of those estimated by (Viscusi, 2018) 's for the U.S, we can say that the 1,290 lives saved thanks to the GES reform (in one year) would be valued at about USD $5,200,000,000.

Evaluating the cost of measures taken to save people's lives is challenging. However, the tax reform implemented to fund the GES Program in 2003 brought about USD $1,000,000,000 in additional revenues after one year of its implementation. Therefore, we can say that the cost of the reform was approximately a quarter of the benefits that were brought because of the lives saved.

[18] Notice that period life expectancy assumes that people live their entire life, from birth to death, under the mortality conditions of 2003 (Human Mortality Database, 2022). In other words, this indicator implicitly assumes that the benefits from the GES reform are experienced each year over and over again as a person gets older.

[19] We compared our results using Table 1, column (1), and Table 3 columns (3-5) age-specific coefficients to compute the total and age-specific relative decrease in deaths, finding a 0.01 difference between them.

Appendix References

Human Mortality Database (2022): "Human Mortality Database," .

Mardones, C. and M. Riquelme (2018): "Estimation of the value of statistical life in Chile and extrapolation to other Latin American countries," *Latin American Research Review*, 53, 815–830.

Nolte, E. and M. McKee (2011): "Variations in amenable mortality—Trends in 16 high-income nations," *Health Policy*, 103, 47–52.

Parada-Contzen, M. V. (2019): "The Value of a Statistical Life for Risk-Averse and Risk-Seeking Individuals," *Risk Analysis*, 39, 2369–2390.

Sommers, B. D., S. K. Long, and K. Baicker (2014): "Changes in mortality after Massachusetts health care reform : A quasi-experimental study," *Annals of Internal Medicine*.

Viscusi, W. K. (2018): "Pricing lives: International guideposts for safety," *Economic Record*, 94, 1–10.

Wooldridge, J. M. (2021): "Two-way fixed effects, the two-way mundlak regression, and difference-in-differences estimators," *Available at SSRN 3906345*.