The Impact of Standardized Disease-Specific Healthcare Coverage*

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Abstract

We study the impact of a healthcare reform that standardized procedures and timely coverage of a set of diseases. Using Chile's universe of death records and a difference-in-differences research design, we show that mortality from the diseases covered by this reform decreased by 4.4% on average. Disease-specific shocks or a resource shift from non-covered to covered diseases do not explain this effect. Evidence from poly-traumatized inpatients suggests that the reform equalized utilization rates as it reduced the dispersion of risk-adjusted surgery rates and spending across hospitals.

Keywords: Mortality, Health Policy

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1 Introduction

As the international community seeks to achieve universal health coverage through cost-effective policy interventions (The Lancet, 2019), the need for rigorous evidence on the impact of health reforms has increased.[1] Recent studies examining the expansion of insurance coverage (Finkelstein et al., 2018; Goldin et al., 2020; Miller et al., 2021) find beneficial effects on mortality. However, the reality of systems with near-universal coverage, like the NHS in the U.K., reveals significant disparities in access to care (e.g., Laudicella et al., 2012). In this paper, we study a program layered over an existing "universal coverage" system to assess how guaranteeing timely access to a high and standardized level of care for specific diseases impacts mortality.

We study Chile's most significant health reform in the past 30 years: the Explicit Healthcare Guarantees program (known as "GES" for its Spanish acronym). This reform guaranteed the treatment of 56 health-related problems amenable to health care—independent of patients' income or health insurance plan—and introduced mandatory guidelines for providers, establishing specific procedures and the maximum times for diagnosis and treatment of covered diseases (Missoni and Solimano, 2010).[2] Given budget constraints, diseases covered by the reform were included in a staggered fashion.

Leveraging rich administrative data and the timing of the GES program's coverage expansions, we evaluate the impact of this intervention on health outcomes. Our data comes from Chile's Department of Health Statistics and Information and includes the causes of death for each death registry and inpatient discharge record. Combining these datasets, we construct disease-age group cells and classify them as treated (or not) based on diagnoses and age groups covered by the reform in each expansion (e.g., coverage of ischemic strokes for patients older than 15 years old started in 2006). Since we do not observe the prevalence of each disease in the population, our outcomes are the counts of total deaths, inpatient deaths, and inpatient surgeries, and our leading research design is a difference-in-difference model using a Poisson regression (Chen and Roth, 2024; Mullahy and Norton, 2024).

[1] In 2015, United Nations member states agreed to work toward universal health coverage by 2030.

[2] Close to the Chilean reform is the landmark experience of the State of Oregon that developed a priority-setting method to operationalize a medical solution to resource allocation based on a cost-utility formula (Dixon and Welch, 1991). Both the UK and The Netherlands also explored plans to combine a focus on cost-effectiveness with the use of clinical guidelines developed by expert boards to reach quality at an affordable cost (Casparie, 1991).

We find that the reform led to an average 4.4% decrease in deaths from covered diseases. Taken at face value, this number implies that lives saved due to reform coverage represent 3.1% of the total number of deaths in 2003 (one year before the reform), suggesting an increase in life expectancy large enough to have taken Chileans in 2003 forward to the mortality conditions of 2005, when life expectancy was 77.78 years.[3] 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 medical care (Sommers et al., 2014; Sommers, 2017; Miller et al., 2021). We document that mortality falls by around 11% for the more amenable diseases, but it only falls by 2.2% for less amenable diseases. We also show that the reform decreased in-hospital mortality associated with covered diseases by 6.9%, on average; this larger impact is consistent with the fact that individuals in the discharge records sought and received medical attention.

A sensitivity analysis reveals that two diseases were key to accounting for the decrease in mortality: polytraumatisms and ischemic strokes. Removing the former disease from the treated group leads to a 14% decrease in our main effect (from -4.4% to -3.8%) while removing the latter leads to a 30% decrease in our main effect (from -4.4% to -3.1%). Regarding heterogeneous treatment effects, we find that hospitals with low utilization rates experienced a decrease in mortality three times larger than that experienced at hospitals with high utilization before the reform,[4] and we see a larger reduction in the mortality of those below 80 years old, without sizable differences between males and females. We also document that the decrease in inpatient deaths was three times larger at public hospitals than at private hospitals. Since public hospitals disproportionately serve the country's most disadvantaged population, we interpret these findings as suggestive evidence that the reform helped to narrow socioeconomic gaps in access to health care.

Several validation exercises strengthen the causal interpretation of our result. First, we present evidence from event studies that show parallel relative trends and indicate that the reform's impact persisted until the end of our analysis period. Second, we find similar ef-

[3]These back-of-the-envelope calculations use the number of deaths from covered diseases (38,129) and the total number of deaths (53,427) in 2003, the year before coverage started. Based on the estimated 4.4% average decrease in deaths, we conjecture that 1,678 deaths would have been averted in 2003 had the reform been implemented that year. All numbers are available in Table 1 and Appendix Table A.9. We compute the impact on life expectancy using the Chilean life tables; Appendix B offers details on this.

[4]We classify hospitals as having low or high utilization rates based on the average surgery rate they had before the reform.

fects when we remove never-covered cells and leverage variation in the timing of adoption among covered diseases for identification. Third, in an attempt to address competing risk concerns (Honoré and Lleras-Muney, 2006), we show that our result is robust to alternative models that allow for flexible age-group and disease-specific time trends.[5] Moreover, leveraging the World Health Organization (WHO) mortality database, we show that a) mortality from covered diseases does not decrease in comparable countries under a placebo treatment that uses the timing of coverage expansions in Chile, and b) we cannot reject the null of a zero impact of the reform on mortality from non-covered diseases (when using the mortality trends of non-covered diseases in other countries as controls). These results are consistent with the fact that the reform did not take funding away from non-covered diseases; instead, it created new sources of revenue by increasing the value-added tax by one percentage point, which brought in an additional 1.7% of the GDP in tax revenues per year.

Finally, to assess mechanisms, we focus on the only procedure available in our data during this period: inpatient surgeries. We find that inpatient surgeries for covered diseases increased by 15% on average due to the reform. Albeit sizable, this number implies around 2,800 extra surgeries per year, representing only 4% of the yearly average number of inpatient surgeries in our data.[6] We also document that public hospitals were the most responsive to the reform regarding inpatient surgeries and that the coverage of polytraumatized diseases was the main force behind the surge in surgeries, i.e., the overall impact of the reform on surgeries decreases from 15% to 5.6% when removing this category. Upon closer examination of polytraumatized patients, we find that the dispersion of hospital-level risk-adjusted surgery rates and logged spending decreased by around 30% after the reform. We interpret this reduction—consistent with the reform's emphasis on procedural standardization—as suggestive evidence of equalization in utilization rates.

Our paper makes three contributions to the existing literature. First, it complements extensive research on the effects of health reforms on health outcomes, most of which focus on expanding insurance based on age or socioeconomic status to previously uninsured populations. In the U.S., studies using experimental and quasi-experimental variations in

[5]Using inpatient records, we also show that following the intervention, patients who survive a covered disease have a lower probability of dying from non-covered diseases than from covered diseases.

[6]This back-of-the-envelope calculation uses the 15% average increase in surgeries and the number of surgeries associated with covered diseases (18,718) in 2003, the year before coverage started. It also leverages the average number of surgeries over 10 year period (761,472/10). All numbers used are available in Table 3.

Medicaid and Medicare coverage show that health insurance increases healthcare utilization and improves health (Finkelstein et al., 2012; Card et al., 2008, 2009). Relatedly, evidence from the 1970s RAND Health Insurance Experiment (Newhouse et al., 1993) suggests null effects of varying insurance generosity on deaths (below 65 years old) in a context with low baseline mortality. In more recent studies assessing the U.S.'s Affordable Care Act insurance expansion (ACA), Gruber and Sommers (2019) find limited evidence of improvements in health outcomes, but Black et al. (2019) challenges its statistical power; Borgschulte and Vogler (2020) find a reduction in all-cause mortality for ages 20-64, and Goldin et al. (2020) and Miller et al. (2021) report reductions in mortality for ages 55-64. Likewise, Wyse and Meyer (2023) finds that Medicaid expansions under the ACA reduced the mortality hazard. In Latin America, Arroyave et al. (2013) show that mortality disparities decreased due to doubling health insurance coverage in Colombia, and Parker et al. (2018) suggests that the "Seguro Popular" health insurance increased utilization and diagnosis in Mexico. In contrast to these papers, we use data on the universe of deaths in the country of interest and leverage quasi-experimental variation to assess the impact of a program that—independently of patients' insurance and layering on a "universal coverage" system—guarantees the timely and standardized treatment of a set of diseases.

Second, our work contributes to research on medical care utilization. Healthcare spending and utilization vary substantially across hospitals, even after controlling for differences in patients' risk (Skinner, 2011). In the U.S., 40–50% of the geographic variation in utilization is attributable to demand-side factors, with the remainder due to place-specific supply factors (Finkelstein et al., 2016). As stated by Chandra and Staiger (2020), "*the conventional interpretation in the medical literature is that there is a correct amount of use, so that variation across providers in risk-adjusted treatment rates is evidence of allocative inefficiency*", an interpretation that has led to an emphasis on medical guidelines. However, as pointed out by these authors, the extent to which the medical guidelines and standardization of procedures can reduce allocative inefficiencies hinges on whether the variation in utilization reflects true comparative advantage. While medical guidelines and standardization can equalize utilization rates (e.g., Frakes, 2013),[7] their impact on health outcomes is unclear and depends, among other things, on the skill level of the health practitioners (Einav et al., 2020) and on patients' compliance behavior (Chan et al., 2022). We contribute to this literature by showing the beneficial effect of a reform that standardized

[7] Frakes (2013) finds that standardization in malpractice law leads to greater practice standardization and reports evidence consistent with a "flat-of-the-curve" story.

procedures on mortality and by providing prima facie evidence of convergence in hospitals' utilization rates. Relatedly, by providing evidence that a reform enhancing health resources and reducing payment frictions had positive impacts, our work also contributes to the literature on the critical role of supply-side incentives and system preparedness in ensuring that increased coverage leads to better access and more utilization (Clemens and Gottlieb, 2014; Alexander and Schnell, 2024; Garthwaite, 2012; Dunn et al., 2024).

Third, we contribute to the literature studying 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. Regarding demographics, we find no differential effects on sex-stratified samples and document larger mortality reductions for 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). Finally, our paper also complements previous studies of this reform. Focusing on 3,500 patients with acute myocardial infarction across 10 hospitals, Nazzal et al. (2013) showed the policy's early success. Likewise, Frenz et al. (2014) used survey data to show that the reform improved access to healthcare 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 diseases covered in the first four waves of expansion and provide causal evidence using a quasi-experimental research design.

The remainder of the paper proceeds as follows: Section 2 describes the institutional background and the GES program. Section 3 provides details on data sources and the sample construction. Section 4 presents our empirical strategy and the main results. Section 5 shows the impact of the reform on procedures, and in section 6, we conclude.

2 Institutional Background and the GES Reform

2.1 The Chilean Health Care System

Chile has experienced rapid economic growth since the mid-1980s, with a GDP per capita (PPP, constant international US$ 2021) of nearly $29,500 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, a two-tier health insurance system was introduced: it stipulated a mandatory 7% contribution for workers in the formal economy, who could use these contributions to obtain public or private health insurance. The *Fondo Nacional de Salud* (FONASA)'s public system is funded by taxes and mandatory contributions. It offers *universal coverage* mainly in public hospitals to everyone that requires it, with three levels of co-payment (0%, 10%, or 20%) based on the patient's income and the number of family members that depend on her.[8] Private insurance providers, *Instituciones de Salud Previsional* (ISAPREs), offer health plans for different prices and compete in a regulated market to attract those who have chosen to use their mandatory contributions in the private insurance system. Nearly 78% of the population contributes to the public system, while ISAPREs only cover around 17-18% of the population. An Armed Forces insurance scheme covers the remaining 4-5% of the population.

While the Chilean public healthcare system has extensive coverage in primary care for individuals with limited resources, this coverage can vary across different healthcare provisions, partly because primary healthcare is provided through local governments. The ISAPREs, on the other hand, provide outpatient and inpatient services through their own clinics and hospitals or by contracting with other public or private facilities. Moreover, FONASA serves more people from disadvantaged backgrounds—a population with a higher risk of disease and health-related issues—while ISAPREs cover the wealthier, healthier,

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

and younger population (Pardo, 2019).

2.2 The GES Reform

"As part of this bill, we identify the leading causes of death: cardiovascular, cancers, and traumatism. The first group aims to decrease mortality through specific interventions for ischemic and cerebrovascular disease. Likewise, cancer mortality will be targeted through the intervention in cervix uteri, breast, vessel, and prostatic and increase palliative care coverage. Regarding traumatism, it urges stopping the increased mortality due to traffic accidents..."

Ministry of Health, Osvaldo Artaza addressing Chilean congress in 2004.

In 2001, the Chilean government conceived the GES program as a major reform to the Chilean health system to achieve *effective* "universal health coverage" (The Lancet, 2019). Recognizing that treatments can differ depending on patients' insurance and healthcare providers, the Chilean Congress approved a package of bills in 2004. The country made a novel effort to guarantee access, provide timely care administration, improve quality, and secure financial coverage for specific health-related problems with high mortality, morbidity, and financial impact (Vargas and Poblete, 2008). These conditions encompassed heart attacks, ischemic strokes, hypertension, diabetes, pneumonia, specific cancers, and traumatism, among others. Although these health conditions were previously covered under the government's universal health care policies, timely access, standardized procedures, and financial protection were limited (Paraje and Infante, 2015). Indeed, the presence of waiting lists in the public sector presented a notable obstacle to obtaining timely care, especially in the case of highly specialized treatments and complex surgeries (Erazo, 2011), and high medical expenditures were identified as the second most common cause of income shocks experienced by households (Neilson et al., 2008).

The GES reform ensured, for the first time, a standardized benefit plan that granted equal entitlement to beneficiaries of public and private insurers, guaranteeing timely access to high-quality care for top-priority conditions with financial protection (Erazo, 2011). It ensures financial security through limits to contributions, payments, and co-payments. Depending on the health-related problem, people may also have access to free prescriptions. To explore this financial dimension of the reform, we use longitudinal survey data to study

the correlation between GES coverage and the number of medical visits and out-of-pocket health expenditures. Results from this exercise are available in Appendix C. We find that—among respondents who report ever being diagnosed with a health condition—those whose health condition was covered by the GES program were 46% more likely to report a medical visit (and when reporting, reported 40% more visits) and 26% less likely to report out-of-pocket medical expenditures (and when reporting, declared 49% lower healthcare spending).[9]

The implementation of disease-specific clinical guidelines was a key aspect of the program.[10] The guidelines defined both the standardized procedures and the timeline for the diagnosis, treatment, and follow-ups. To achieve timely care administration maximum waiting times were established, after which it is possible to seek care through private providers (Bitran, 2013). In most cases, once a public or private health provider verifies the diagnosis, patients are assigned to treatment in a specific network and cannot choose where to get care; otherwise, they lose the benefit. To illustrate the changes introduced by the reform, we can consider the case of a time-dependent disease such as Acute Myocardial Infarction (AMI), for which there were no standardized timelines and procedures before the GES program. After the reform started, the GES program covers and mandates i) for diagnosis, an electrocardiogram and a specific blood test to estimate cell death; ii) for treatment, an angioplasty in less than 90-120 minutes at high-complexity facilities or a thrombolysis within the first 30 minutes at low-complex facilities. Although timely diagnoses and treatment are essential for this pathology's prognosis and mortality rate, anecdotal evidence suggests that procedures varied across providers before the reform.

As part of the GES Reform's legislative package, Bill 19,937 established the Superintendence of Health as the authority responsible for overseeing and monitoring the effective operation of the GES program. This regulatory body is mandated to ensure that every patient has the right to hold insurers and providers accountable if access to care, timely treatment, quality, or financial coverage does not meet the explicit healthcare guarantees outlined in the program. Additionally, to promote and enhance healthcare quality, the Superintendence certifies and grants legal authorization to providers, thereby safeguarding individuals' rights and protecting against discrimination.

[9]These effects remain statistically significant (albeit smaller in effect size) if we include person-fixed effects, thus leveraging within-person variation in the timing of GES coverage (see Appendix C for details).

[10]For the interested reader, this link provides access to all clinical guidelines; they may also be accessed directly at: https://diprece.minsal.cl/le-informamos/auge/acceso-guias-clinicas/guias-clinicas-auge/.

To fund the implementation of the GES program, the government passed a tax reform (Bill No. 19,888, enacted in August 2003). This reform increased the value-added tax by one percentage point, resulting in additional tax revenues amounting to 1.7% of GDP one year after its implementation. Thus, the GES program led to a significant increase in healthcare resources. Indeed, the average annual growth rate in health spending increased from 7% between 1990 and 2004 to 27% between 2005 and 2012. Among the most significant health expenditures were investments to ensure the treatment of covered diseases with adequate equipment and drugs (Benavides et al., 2013; Nazzal et al., 2013); an increase in the working hours of public-sector health workers to comply with the timely administration of care (Guillou et al., 2011); and the development and implementation of new software to manage payments and expedite the flow of resources associated with this program (Carrasco and Medina, 2019).

When initially conceived, the reform intended to cover 56 health-related problems simultaneously. However, coverage was gradually rolled out to pilot performance and to provide the system with enough resources. Identifying requirements for human resources, equipment, technology, and infrastructure that considered each health condition's specific needs was critical (Paraje and Infante, 2015). The intervention 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 patients seeking care in public hospitals, who represented 73% of the population (MINSAL, 2004). This is considered the initial expansion, covering 17 priority conditions, including high-prevalence diseases such as heart attacks, hypertension, and diabetes. Subsequent developments in 2005, 2006, 2007, 2010, 2013, and 2019 brought the total to eighty-five covered conditions of varying prevalence and amenability to care. Relevant to our empirical approach is the fact that coverage also targeted specific age groups for some diseases. For instance, bronchial asthma was covered by the 2006 expansion for people below 15 years old, but in 2010, coverage expanded for those above 15 years old. Another example is cholecystectomy, a standard treatment of symptomatic gallstones and other gallbladder conditions, which is covered only for people between 15-39 years old. Detailed tables with each covered health-related problem and age group can be found in the Appendix (Tables A.1 through A.4).

Although novel, the measures implemented by the GES reform resemble practices em-

bedded in other countries' health systems. The Norwegian system, for instance, establishes "patient rights" to access quality healthcare and stipulates minimum waiting times for complex treatments, such as those requiring surgeries. Likewise, in Sweden, if the guaranteed time for treatment is exceeded, treatment is covered in private institutions or abroad. Additionally, financial protection establishes a maximum annual amount for outpatient co-payments. The U.K. and The Netherlands also explored plans to combine a focus on cost-effectiveness with the use of clinical guidelines developed by expert boards to reach quality at an affordable cost (Casparie, 1991). Outside of Europe, in the '90s, the State of Oregon in the U.S. expanded insurance coverage based on a prioritized list of health services and treatments, ranked according to their clinical cost-effectiveness (Dixon and Welch, 1991). Medicare coverage of end-stage renal disease in the U.S. also resembles the Chilean reform as it ensures access to care (including regular dialysis or kidney transplants), regardless of age.

3 Data and Sample Construction

3.1 Data Sources

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

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). Key to our empirical strategy is the fact that the reform defined coverage and clinical guidelines based exclusively on the patient's diagnosis (ICD-10 code) and age group. The list of covered diseases by ICD-10 and age group is publicly available on the Ministry of Health webpage. Regarding data quality, Chile's vital statistics rank among the best in the world (Mikkelsen et al., 2015). As shown in Appendix Figure A.1, the country has an established protocol to record deaths (Government of Chile, 2016), and neither patients nor health providers have incentives to influence the diagnoses for billing purposes. On the one hand, all patients in the public system must follow a strict referral system and cannot choose their hospitals or physicians. On the other hand, the diagnoses are recorded directly by the lead physician, who must follow the nationwide mandatory program that aims to characterize the morbidity profile of patients for policy purposes (Government of Chile, 2010). Moreover, to access the coverage provided by the GES program, the medical team must perform specific exams and provide objective evidence that backs up their diagnoses.

Finally, to address concerns related to disease-specific shocks or shifts in health resources from non-covered to covered diseases, we use the World Health Organization (WHO) mortality dataset. Specifically, we use the death counts by ICD-10 and age categories from countries in the Central and South American region that are classified as countries with high data usability, according to the WHO (WHO, 2020).[11]

3.2 Sample Construction and Descriptive Statistics

To construct our analysis sample, we first identify all diseases that result in deaths. We then combine individual deaths and discharge records to construct cells with the counts of deaths, in-hospital deaths, and surgeries by the ICD-10 disease codes and 22 age groups defined as 19 five-year age groups and three ad-hoc groups (newborns, ages 1 to 4, and open-ended intervals for deaths above 100). We classify each resulting cell as covered or non-covered using the comprehensive list of ICD-10 codes and ages covered by each of the GES expansions between 2004 and 2007.

We also identify cells from conditions that are *more* amenable to health care following Nolte and McKee (2011) and Sommers et al. (2014) (see Appendix Table A.5 for a detailed

[11] These countries are Belize, Mexico, Venezuela, Paraguay, Brazil, Costa Rica, Nicaragua, Panama, and Colombia. Data is publicly available in the WHO webpage.

list of the ICD-10 codes included).[12] Appendix Table A.6 reports the number of deaths associated with diseases covered by each expansion of the GES program and the percentage of those deaths categorized as amenable. As shown by this table, the mapping between covered condition and the amenable classification is not straightforward. Indeed, only 23% of the deaths of diseases covered by the GES program are classified as more amenable. The reason is that the more amenable classification is not only at the level of ICD-10 codes but also establishes age restrictions, which sometimes are similar to the program coverage but do not always overlap. For instance, the health-related problem "Myocardial Infarction (Heart Attack)", covered in 2004, includes all the ICD-10 codes between I21 and I23; but only 50% of the deaths associated with this health problem are below 75 years old, and thus classified as amenable to healthcare.[13]

Our sample excludes diseases considered in the pilot program that happened between 2002 and 2003 because i) it is not clear how these conditions were chosen, and ii) we only have discharge records starting in 2001.[14] We also decided not to include diseases considered in the second wave of expansions (in the years 2010, 2013, and 2019) in our study. The main reason for this decision is that the 2010 and 2013 groups of covered diseases were piloted before the program formally expanded, which could introduce bias to our estimates. Likewise, we do not consider the 2019 expansion because it included four cancers that were already covered during the 2002-2003 pilot under "cancer palliative care".[15] As mentioned in subsection 2.2, program expansions only increased age-group coverage for some diseases. This implies, for example, that 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 set of expansions that we study. For the same reason, there are diseases in both covered and uncovered groups (i.e., because their coverage was only for a specific age group and we define coverage at the disease-age level. For the exact number of diseases and disease-age cells covered, see the Appendix Table

[12] Diseases not included in the "health-care amenable" category are referred to as *less* amenable; these diseases are not unaffected by timely medical care but they are less likely to be responsive to health care coverage compared to the more amenable causes (Nolte and McKee, 2003, 2008, 2011; Sommers et al., 2014).

[13] Nolte and McKee (2003) set an age limit at 75 years, as the avoidability of death through timely responsive care becomes increasingly difficult at older ages.

[14] This group represents 15.8% of all deaths in the period studied and 69% of them were terminal cancers.

[15] The 2019 expansion also covered Alzheimer's and other forms of dementia. We decided not to include Alzheimer's and other dementia types in our analysis because the classification of deaths as a consequence of Alzheimer's has been unstable over time (e.g., some deaths previously recorded as epilepsy are now recorded as Alzheimer's). Nonetheless, our results are robust to including this category in the control group.

A.7. Moreover, among diagnoses covered within our time frame, 16 did not have deaths during the study period.[16] Thus, we end up with a yearly panel with counts by age group and ICD-10 codes for 35 health-related problems covered by the reform during the 2004, 2005, 2006, and 2007 expansions.

Almost 60% of deaths in our sample are concentrated among diseases of the circulatory, respiratory, and digestive systems. Neoplasms and injuries account for an additional 20% (for details, see Table A.8). Appendix Table A.9 underscores the targeted nature of the reform by showing that combined, all expansions targeted almost 50% of deaths in the period of our study in an evenly distributed fashion (between 10-15% in each expansion). Finally, Appendix Table A.10 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. This table also shows that the reform covered around 50% of 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.

Regarding the WHO Mortality dataset, we construct a panel of cells using the same procedure described above and classify them as covered and non-covered using the ICD-10, age categories, and the timing of the Chilean GES reform. A difference between our primary dataset and the WHO database is that the latter has an open-ended age interval of 95 years and above, while ours has an open-ended interval of 100 years and above. Moreover, the WHO database classifies deaths under chapter XIX (that ranges from S00 to T98), titled "Injury, poisoning and certain other consequences of external causes" as the *underlying* cause of death. In contrast, we considered them as the leading cause of death. As shown in the next section, we find quantitative and qualitatively similar results for Chile using the WHO data.

[16]These diseases, excluded from our analysis, corresponds 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, and hypoacusis.

4 The Impact of the GES Reform

We begin this section by presenting our empirical strategy, the main estimates of the reform's effect on mortality, and several robustness checks. Then, we show the reform's heterogeneous effects and its impact on procedures. We end this section with an assessment of potential confounders.

4.1 Empirical Strategy

The phased rolled-out of the reform coverage allows us to implement a staggered difference-in-differences research design. In particular, we can use the timing of coverage among different disease-age cells to study changes in cell-level outcomes before and after coverage. We cannot construct disease-specific death rates because we only observe deaths, not how many individuals suffer from each disease. Thus, the outcomes of interest will be yearly counts within a disease-age cell (e.g., deaths or inpatient surgeries associated with polytrauma among people between 35 and 39 years old in a given year), and we fit Poisson models for counts using a log link, with a general specification 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 (a disease-age combination) in period t. GES_{dt} is an indicator that equals one from the first time a disease-age cell is covered and onward, i.e., the treatment is an absorbing state. α_d represents cells' fixed effects that control for time-invariant unobservables specific to the disease-age group, and γ_t are time-fixed effects that account for year-specific shocks common across diseases. 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.

Following Chen and Roth (2024), our parameter of interest is the average proportional

treatment effect on the treated:

$$\theta_{ATT\%} = \frac{E[Y_{dt}(1) - Y_{dt}(0) \mid GES_{dt} = 1, Post_t = 1]}{E[Y_{dt}(0) \mid GES_{dt} = 1, Post_t = 1]} \tag{2}$$

This is the percentage change in the average outcome for the covered group in the post-treatment period. Conveniently, the $\hat{\theta}_{ATT\%}$ can be recovered from $\exp(\hat{\beta}) - 1$, where $\hat{\beta}$ is the estimator obtained from the Poisson model given by equation (1).[17] Thus, we would expect the $\hat{\theta}_{ATT\%}$ to be negative if the GES reform led to a relative decrease in deaths among covered diseases and positive otherwise.

In this case, identification of the causal effect of the GES reform $\hat{\theta}_{ATT\%}$ is predicated upon the assumption that in the absence of treatment, the *percentage changes* in the mean would have been the same for the covered and non-covered groups of diseases.[18] As in Wooldridge (2023), this can be formalized using a "ratio" version of the parallel trends assumption, sometimes referred to as the "parallel relative trends" assumption:

$$\frac{E[Y_{dt}(0) \mid GES_{dt} = 1, Post_t = 1]}{E[Y_{dt}(0) \mid GES_{dt} = 1, Post_t = 0]} = \frac{E[Y_{dt}(0) \mid GES_{dt} = 0, Post_t = 1]}{E[Y_{dt}(0) \mid GES_{dt} = 0, Post_t = 0]} \tag{3}$$

Intuitively, equation (3) states that if the treatment had not occurred, the average percentage change in the mean outcome for the covered group would have been the same as the average percentage change in the mean outcome for the non-covered group. Under assumption (3), we can estimate the counterfactual percentage change in the mean outcome for the covered group using the observed percentage change for the non-covered group.

To assess the plausibility of the assumption of parallel relative trends, we examine the dynamic effects of GES using event studies around the time when new diseases became covered. Given that deaths and inpatient records have different time frames, we use a time window between 2001 and 2010, which overlaps for both datasets. This allows us to define a 3-year moving window, with a consistent number of leads and lags around each program expansion (i.e., 2004, 2005, 2006, and 2007). Nonetheless, we also present the

[17] The Poisson quasi-maximum likelihood (QMLE) consistently estimates the population coefficient β, which satisfies $\exp(\beta) - 1 = \frac{E[Y(1)]}{E[Y(0)]} - 1 = \theta_{ATT\%}$. The parameter identified through the Poisson model is known as a rate ratio (RR). 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 time (Dicker et al., 2006). Therefore, the interpretation of the value of a rate ratio is similar to that of the risk ratio.

[18] Since the treated and control groups might have different pre-treatment means, assuming parallel trends *in levels* could be a strong assumption, i.e., it may be unreasonable to expect that time-varying factors have equal level effects on the outcome between the group of covered and non-covered diseases.

event study for our main outcome (all deaths) using a 7-year moving window.[19] To estimate this dynamic version of our difference-in-differences specification, we use a leads-and-lags model in event time, with the first expansion year set to zero. Specifically, we estimate the following equation:

$$y_{dt} = \exp\left(\alpha_d + \gamma_t + \sum_{k=\underline{C}}^{-2} \beta_k D_{dt}^k + \sum_{k=0}^{\overline{C}} \beta_k D_{dt}^k\right) \epsilon_{dt}, \tag{4}$$

where $D_{dt}^k = 1[t = GES_d + k]$, and GES_d is the timing of inclusion of disease-age group d. In other words, D_{dt}^k is a dummy variable indicating that disease-age cell d was included in the GES program k periods ago (or will be included k periods ahead, for negative values of k). We normalize the coefficients such that $\beta_{k=-1} = 0$, i.e, treatment is re-coded in event time relative to the year before each disease-age group was included in a GES expansion. Therefore, the β_k coefficients can be interpreted as the effect of GES on the outcome y_{dt} for each k period relative to the year before the inclusion of d in the GES program.

Finally, it is worth noticing that identification of the ATT rests on an additional identification assumption, that is, that treatment effects are homogeneous.[20] To assess the plausibility of this assumption we implement a recent method that recovers the group's ATT in non-linear settings like ours while allowing treatment effects to be heterogeneous over time or across groups (Wooldridge, 2021, 2023; Rios-Avila, 2022).

4.2 Did the Reform Reduce Mortality?

We begin by exploring the impact of reform on mortality using raw data. In Figure 1, we plot the change in the number of deaths in covered diseases against non-covered diseases for each expansion. Panel (a) shows that the change in deaths covered by the 2004 expansion decreased compared to the non-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

[19] A seven-year window is the largest window that allows us to work with a balanced panel of events, i.e., $t = -7$ in 1997 for the first wave of coverage in 2004, and $t = 6$ in 2014 for the last wave of coverage in 2007.

[20] As shown by recent research (e.g., Callaway and Sant'Anna, 2021), two-way fixed effects estimators can deliver estimates that differ from the group's average treatment on the treated (ATT) in the presence of treatment effect heterogeneity.

smaller than in panel (a). 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 non-covered group of diseases. Finally, panel (d) shows the differential trends between diseases included in the 2007 expansion and those non-covered. Again, all deaths increased, but those covered by the 2007 expansion increased far less. It is worth mentioning that the overall increase in deaths shown in Appendix Figure 1 is mainly driven by an aging population.[21]

Although previous evidence is purely descriptive, it suggests that reform coverage decreased mortality, especially for diseases included in waves 2006 and 2007. To formally study this hypothesis—and quantify the impact of the reform—we present the results of our staggered difference-in-differences research design. Table 1 presents the estimates obtained from model (1). Our main result is presented in Column (1), which considers the count of all deaths as the dependent variable. Consistent with the preliminary evidence, we find a statistically significant impact of the reform on mortality, with the average risk of dying from diseases that went from uncovered to covered decreasing by 4.4% after the reform.[22] As shown by equation 2, this effect corresponds to the average proportional treatment effect on the treated, which allows us to compute the number of deaths averted due to the reform. In our estimation sample, the covered group had 38,129 deaths in the pre-expansion period. Therefore, 1,678 deaths were averted once they went from uncovered to covered. This number implies that lives saved due to reform coverage represent 3.1% of the total number of deaths in 2003 (the year before the reform), suggesting an increase in life expectancy large enough to have taken Chileans in 2003 forward to the mortality conditions of 2005 when life expectancy was 77.78 years.[23]

To assess the dynamics of the impact on mortality, Figure 2, panel (a), presents the

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

[22] We obtain similar results if we estimate a negative binomial regression that allows for overdispersion or if we use linear regressions either with the log of deaths+1 or the inverse hyperbolic sine of deaths as ad-hoc transformations to deal with the zero count cells. Table A.11 presents these results in the Appendix.

[23] We use the age-group coefficients in Appendix Table A.15 to calculate the lives that would have been saved for each age group. We then use the proportion of lives saved to re-weight the age-specific mortality rate in the Chilean life table in 2003, one year before the program. With this new age-specific mortality rate, we recompute the rest of the life table column and compute the life expectancy that would have been in the presence of the GES Program in 2003. For the interested reader, Appendix B offers details on this back-of-the-envelope calculation.

event study estimates obtained from model (4) using the count of deaths as the dependent variable.[24] The horizontal axis shows the years relative to the coverage expansion, with event time zero denoting the first year of 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. The figure shows that pre-period estimates are not statistically different from zero, a result in line with our parallel relative trends assumption. Moreover, the figure shows that the number of deaths in treated disease-age cells decreased after their coverage and remained stable at around -4% over time.[25]

As mentioned in section 3, death records using the ICD-10 classification have been available since 1997. Thus, we can add more pre-periods to assess better the assumption of parallel relative trends for our main outcome: the number of deaths. Figure 2, panel (b), presents the event study estimates obtained from model (4) when considering data from all years $\in \{1997, 2014\}$ and imposing the endpoint restrictions $\beta_k = \overline{\beta}$ if $k \geq 7$ and $\beta_k = \underline{\beta}$ if $k \leq -7$, which state that any dynamics wear off after seven years.[26] Reassuringly, the dynamics presented in panel (b) resemble those of panel (a), with pre-period estimates not statistically different from zero and a stable decrease in deaths after reform coverage. We complement previous evidence on the validity of our research design by showing pre-treatment characteristics (coming from the death records) for covered and non-covered cells. As shown in the Appendix Table A.12, there is balance along an array of cell characteristics, including the type of insurance, highest educational level attained, gender, marital status, and geographical location.

Recent literature on two-way fixed effects estimators has shown that estimates from linear models can differ from the group's average treatment on the treated (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 implement a recent method that recovers the group's ATT in non-linear settings like ours while

[24] Appendix Figure A.4 presents the corresponding event study for Poisson and Negative Binomial models.

[25] Appendix Table A.13 and Figure A.5 present estimates of the impact of the reform when considering different expansions of the program (i.e., different sets of diseases covered at different points in time) and only never-covered cells as controls. Albeit noisy, the results suggest that only the 2006 and 2007 waves significantly impacted mortality.

[26] For another example of such endpoint restrictions, see McCrary (2007) and Kline (2011). In light of recent work by Borusyak et al. (2024) showing that a binned specification could lead to bias, we replicate our results without binning the endpoints in Appendix Figure A.6. Results from this robustness exercise are similar to those obtained from our main specification and do not suggest that the dynamics change its pre-program trend or post-program effect.

allowing treatment effects to be heterogeneous over time or across groups (Wooldridge, 2021, 2023; Rios-Avila, 2022). Our estimated ATTs imply a decrease in deaths of 6.1% (with a standard error of 1.6%) when using *never covered* cells as controls and a reduction in deaths of 4.6% (with a standard error of 1.8%) when using *not yet covered* cells as controls. These estimates are statistically significant and align with the ones presented in Table 1. In the same vein of the previous exercise, column (2) of Table 1 presents the results obtained from estimating equation (1) in a sample of *ever covered* cells. In this case—where we only leverage variation in the timing of adoption among covered diseases for identification—we also find that expansions led to a 4% decrease in mortality.

To complement our results, we also study the impact of the reform on inpatient deaths. In column (3) of Table 1, we present the estimates obtained from model (1) using the count of in-hospital deaths as the dependent variable. We find that in-hospital mortality decreased by 6.9% as a consequence of the reform. This effect, larger than the 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 at a healthcare facility, i.e., they show up in the hospital's discharge records. Panel (c) of Figure 2 shows the event study for in-hospital deaths. Similar to the dynamics observed for other counts of deaths, differences between covered and non-covered diseases were almost nonexistent before the reform. However, after expansion coverage, the number of inpatient deaths in covered diseases decreased and remained permanently lower.

To study whether a particular disease or group of diseases drives our results, we follow recent research suggesting that some diseases may be more responsive to medical care than others (Sommers et al., 2014; Borgschulte and Vogler, 2020; Miller et al., 2021), and study the differential impact of the reform on diseases considered to be more "health care–amenable". For this analysis, we classify cells as more amenable and less amenable following the work of Nolte and McKee (2011) and Sommers et al. (2014).[27] We estimate a variant of model (1) that includes an interaction between the GES_{dt} indicator and a variable that equals one if the cell d is classified as more amenable. Columns (4) to (6) of Table 1 present our results. Column (4) shows that mortality fell by around 11% among "More amenable" cells while it only fell by 2.2% for the rest. Columns (5) and (6) show similar results when we consider deaths from ever-covered diseases and deaths from inpatient

[27] Our classification encompasses both the work by Nolte and McKee (2011) and by Sommers et al. (2014). See Appendix Table A.6 for details.

records. Appendix Figure A.7 presents event study estimates of model (4) in the sample of more amenable and less amenable diseases and confirms that the impact of the reform was larger on diseases more amenable to health care.[28]

Finally, to assess the sensitivity of our main result to particular diseases, we estimate the main difference-in-differences model, given by equation (1), but removing one covered cell (i.e., a disease-age category) from the sample at a time. Figure 3 plots the point estimates and 95% confidence intervals derived from this exercise. In all regressions, 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% decrease in mortality. A few disease-age categories stand out as triggers of changes in our main estimate. Among them, we see arterial hypertension, disorders of the heart conduction systems, pneumonia in older adults, ischemic strokes, and polytraumatisms (with and without medullary lesions). On the one hand, the removal of arterial hypertension and disorders of the heart conduction systems leads to relatively larger estimated impacts of the reform ($\sim$ 5% instead of a 4.4% decrease in mortality). On the other hand, removing pneumonia, polytraumatisms, and ischemic strokes leads to smaller estimated impacts of the reform.

The most salient change in the estimated impact of the reform on mortality happens when we remove ischemic strokes from the estimation sample. In this case, the estimated decrease in deaths shrinks from 4.4% to 3.1%. Ischemic strokes were an important contributor to mortality in Chile. Indeed, among diseases covered during the 2006 expansion, ischemic strokes are the largest category in terms of deaths, i.e., they represented 30.6% of all deaths in 2006.[29] Moreover, the reform significantly modified procedures for the diagnoses, treatment, and follow-ups associated with this disease. Before the reform, the diagnosis was made through computed tomography (CT) scans of the brain; after GES, in addition to the CT scan, an angiography of the brain and neck is recommended. Regarding treatment, for those with an intracranial large vessel occlusion, thrombolysis was the standard procedure before the reform; after it, neurologists performed thrombectomies. In the case of a stroke with a foramen ovale, it also must be closed (in addition to the antithrombotic treatment). In terms of medication, the reform's guidelines suggest using oral

[28] As a robustness check, we estimate our main model in different samples of more amenable and less amenable diseases according to alternative classifications including Tobias and Yeh (2009), Nolte and McKee (2003), and the one used by the European Union. As shown by Appendix Table A.14, we find that the impact of the reform is larger on more amenable diseases independent of the more amenable/less amenable classification.

[29] See Appendix Table A.3 for details.

anticoagulants instead of the vitamin K antagonists that were previously used. Last, regarding follow-ups, the guidelines suggest initiating motor therapy within the first 24 hours and a high volume of rehabilitation sessions. Before the GES program, there were no clear timelines nor guarantees for when to start rehabilitation.[30]

The removal of polytraumatisms from the estimation sample also leads to a sizable decrease in the estimated impact of the reform. In this case, the estimated decrease in deaths shrinks from 4.4% to 3.8%. The reform aimed at guaranteeing timely access to care to anyone who presents traumatic injuries affecting at least two systems (of which the failure of one can be life-threatening). Before the reform, procedures to treat polytraumatism were not uniform across providers; after it, clinical guidelines defined how to manage polytraumatized patients from the moment of rescue at the accident site until the completion of treatment in the intensive care unit, emphasizing damage control, care, and rehabilitation. In Section 5, we study the case of polytraumatisms to assess the role of standardization of procedures as a mechanism.

Heterogenous Impacts of the Reform: Before concluding, we replicate our analysis in different sub-samples to study the heterogeneous effects of the reform along the socioeconomic, demographic, and geographical dimensions.

We estimate the model given by equation (1) in different sex and age group samples. This analysis is motivated by the fact that some diseases expanded only for specific sex and age groups. Columns (1) to (5) of Appendix Table A.15 present our results. Even though the reform targets sex-specific diseases, we find no significant differences in mortality for males and females. In contrast, we do see important differences between age groups. Notably, the decrease in deaths between ages 0 and 49 is almost four times larger than the decrease in fatalities among those above 80. The absence of an effect on old age mortality may be associated with the focus 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).[31] 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

[30]For more details, see the corresponding clinical guideline available in this link or directly accessing: https://diprece.minsal.cl/garantias-explicitas-en-salud-auge-o-ges/guias-de-practica-clinica/ataque-cerebrovascular-isquemico-en-personas-de-15-anos-y-mas/recomendaciones-2/.

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

uncertain at older ages, making the classification noisier for these groups of deaths (Weber and Clerc, 2017).

Columns (6) to (8) of Appendix Table A.15 present the results obtained when we use discharge records to estimate the model given by equation (1) stratifying inpatients by type of healthcare provider. We find that the reform reduced mortality in public hospitals by 7.3%, a large and statistically significant effect.[32] In private hospitals, however, the reform reduced mortality only by 2.5%, a smaller and not statistically significant effect. Insofar as public hospitals are the most prominent medical bed providers and serve the most disadvantaged population,[33] we interpret this result as evidence that the reform contributed to closing socioeconomic gaps in healthcare. Furthermore, motivated by the literature on geographical disparities in health (Murray et al., 2006; Bilal et al., 2019; Mena et al., 2021), we also study heterogeneous effects of the reform by the geographic location (residence) of the deceased. Appendix Table A.17 presents the results obtained from estimating the model given by equation (1) in six different samples defined by the major geographical areas of Chile. Our estimates show that the reform decreased mortality by less in the relatively more affluent northern and metropolitan areas.

Finally, to further explore the geographic heterogeneity, in Appendix Table, A.18, we extend our previous findings by showing that hospitals with low utilization rates before the intervention experienced a decrease in mortality three times greater than that observed in hospitals with high utilization rates before the reform. Leveraging our research design, we distinguish between hospitals with high and low capacity and construct disease-age cells similar to those used in our main analysis, but now stratified by baseline utilization.[34] We interpret these results as suggestive evidence that the health system had the capacity to accommodate additional demand, particularly in under-utilized hospitals, once resources became available. These findings strengthen our overall conclusion by demonstrating that the GES program had a more pronounced effect on hospitals that likely required additional resources. Moreover, the results remain robust in the sample of hospitals that performed at

[32] Appendix Table A.16 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.

[33] In Chile, public hospitals are more crowded and have longer wait times. 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; González et al., 2023). Additionally, previous studies found that patients at public hospitals show a higher risk of in-hospital death (Cid Pedraza et al., 2015). In our discharge records, 96% of patients at public hospitals have public insurance.

[34] For this analysis, we classify hospitals based on their average pre-reform surgery rates, focusing only on those that performed at least one inpatient surgery before the reform.

least one surgery, showing a 6.3% reduction in mortality—though smaller than the overall reduction, it is comparable to the effect observed for all in-hospital deaths.

In summary, our heterogeneity analysis shows that the reform i) had similar effects for men and women but a more considerable impact on the mortality of people below 80 years old, an age group where deaths amenable to high-quality and timely health care are concentrated; ii) had a significant effect on public but not in private hospitals, suggesting it helped to reduce socioeconomic disparities; iii) had a weaker impact on richer geographical areas, suggesting that the reform also helped to narrow geographic disparities; and iv) had a larger effect on underutilized hospitals, suggesting that the resources went increase the efficiency of hospitals that likely required additional resources.

4.3 Assessment of Potential Confounders

A natural concern in our setting is that of competing risks (Honoré and Lleras-Muney, 2006), i.e., if a person's death from covered diseases is averted thanks to the reform, she might become more likely to die of an uncovered disease later on. In our case, competing risk might increase mortality in the "control group", leading to overestimating the reform's impact. We have already shown that the reform led to a 4% decrease in mortality when we removed never-covered cells from our sample and leveraged only variation in the timing of adoption among covered diseases for identification. Nonetheless, this subsection offers additional evidence to strengthen the causal interpretation of our findings.

If competing risks are at play, the reform's positive impact on covered diseases might have changed the trends of never-covered diseases. To empirically address this concern, we estimate more flexible models that allow for age group- and disease-specific time trends. To do so, we include disease-by-year, age-group-by-year, and cell (disease-age)-by-year fixed effects. Appendix Table A.19 shows that, after the inclusion of these flexible trends, the difference-in-differences estimates of equation (1) remain statistically significant and fluctuate around our main result (-4.4%) in the range of -4.5 and -3.7 percent. We can further enhance the flexibility of our models by interacting the aforementioned set of fixed effects with an indicator that equals one for the post-reform period (i.e., year $\geq$ 2004), thus allowing for differential trends in age groups and diseases before and after the reform. Columns (5) to (7) of Appendix Table A.19 show that, in this case, the difference-in-differences estimates of equation (1) also remain statistically significant and fluctuate around our main

result (-4.4%) in the range of -4.6 and -3.9 percent.

We complement previous evidence with a non-parametric survival analysis to assess whether individuals who survived a covered disease are more likely to die from non-covered diseases later on. Leveraging hospital records and focusing on the post-reform period, we calculate non-parametric death hazard rates that proxy the risk of dying from covered and non-covered diseases (conditional on survival from a covered disease).[35] Appendix Figure A.8 shows these post-discharge death rates for the covered and non-covered groups of diseases, separately by age group. Reassuringly, we find that the probability of dying from non-covered diseases is smaller than the probability of dying from covered diseases for all age groups (0-49, 50-79, 80+). Although descriptive, this additional evidence also goes against the idea that we are overestimating the reform's impact due to competing risks.

A related concern is that resources might have shifted from non-covered to covered diseases, inadvertently causing a worsening in the provision of healthcare for non-covered diseases.[36] In light of this, it is worth noticing that—*de jure*—the reform did not remove funding from non-covered diseases. Instead, the government passed a tax reform to fund the GES program, which increased the value-added tax by one percentage point (bill No. 19,888, enacted in August of 2003) and brought in an additional 1.7% of the GDP in tax revenues one year after its implementation, leading to a significant increase in healthcare resources. Indeed, health spending—as a percentage of the GDP—increased by 13% between 1998 and 2003 (from 2.3 to 2.9), but it increased by 37% between 2004 and 2011 (Toro, 2021). Fiscal evidence notwithstanding, whether the reform impacted the mortality from diseases not covered by the reform is ultimately an empirical issue. To study this, we use the World Health Organization's mortality database, which allows us to compare the evolution of mortality in Chile vis-a-vis other countries of the Central and South American region.[37] For each country, we construct a panel of disease-age group cells resembling the ones used in our main analysis and classify them as covered or non-covered using the

[35] Simply put, we count all in-hospital and out-of-hospital deaths (from covered and non-covered diseases) and divide them by the number of patients discharged alive after admission for a covered disease.

[36] Unfortunately, no data on wait lists is available during our study period. The most recent data started in 2008, but major changes were introduced in 2012 to improve reliability. Therefore, we cannot assess the impact of the GES program on waiting times.

[37] We only consider countries with high data usability as defined by WHO (2020). These countries are Belize, Mexico, Venezuela, Paraguay, Brazil, Costa Rica, Nicaragua, Panama, Colombia, and Chile. All data is publicly available via the WHO webpage (https://www.who.int/data/data-collection-tools/who-mortality-database).

ICD-10 codes, age categories, and the timing of the Chilean GES reform.

If the reform led to worse healthcare for non-covered diseases, then deaths from non-covered diseases in Chile should have increased after the reform. Using the WHO data, Panel (a) of Appendix Figure A.9 shows that the rate of deaths from non-covered diseases in Chile was similar to the trends observed in other countries, fluctuating closely around zero over our sample period. We enhance this descriptive analysis by constructing a synthetic control for Chile.[38] Panel (b) of Appendix Figure A.9 shows that the logarithm of deaths (from non-covered diseases) in Chile matches the evolution of deaths (from non-covered diseases) in the synthetic control closely up to 2004, with no clear signs of divergence afterward, lending further support to our previous finding of no abnormal growth in mortality among non-covered diseases in Chile after the reform.

To further assess if healthcare provision for non-covered diseases got worse and also to address concerns related to disease-specific shocks that could confound our results, we estimate alternative difference-in-differences models using different samples of the WHO mortality database. We use the same disease-age level of analysis for this exercise; thus, we account for differences in the age composition of mortality in each country. In this specification, we also include the fixed effects of our main specification (disease-age and year-fixed effects) interacted with country dummies. We present these results in Table 2. As a data sanity check, we repeat the estimation of model (1) in this data and find similar results. As shown by Column (1), the impact of the reform, using the WHO data, corresponds to a 3.6% decrease in deaths.[39] In column (2), we also focus on Chile but now consider exclusively ever-covered cells (i.e., removing non-covered cells from the control group). In this case, when we leverage only the timing of coverage among covered diseases, the magnitude of the treatment effect is -3.9%, similar to the -4.0% previously reported in column (2) of Table 1.

We performed a placebo check using other countries to address concerns about disease-

[38]For our synthetic control, we use lags of the logarithm of deaths, the logarithm of cumulative deaths, and the growth of deaths before 2004, the first year of the GES reform. These variables are employed to calculate the weights given to each country following Abadie et al. (2010, 2015).

[39]The difference between this and our main estimate of -4.4% might stem from the fact that the WHO has a different age grid for the elderly and classifies deaths under chapter XIX differently than the Statistics Department of the Chilean Ministry of Health. The difference between the Chilean and WHO data is that the latter has an open-ended age interval of 95 years and above, while the former has an open-ended interval of 100 years and above. Moreover, the WHO database classifies deaths under chapter XIX (that range from S00 to T98), titled "Injury, poisoning and certain other consequences of external causes", based on the *underlying* cause of death. In contrast, we consider them as the leading cause of death.

specific shocks to mortality that could have coincided with the timing of the GES reform. Specifically, we only considered the disease-age groups that were ever covered. For them, we estimate a difference-in-differences that uses the timing of coverage of the Chilean reform. To be consistent across specifications, we interact cell and year dummies with country-fixed effects. As shown by column (3) of Table 2, we cannot reject the null of a zero impact of the timing of coverage in Chile on other countries' mortality. In column (4), we extend the previous specification, now considering all countries and adding an interaction between a binary indicator for "After GES Expansion" and a binary indicator for "Chile". Insofar as we use the evolution of *covered* diseases in other countries as a counterfactual for Chile, this specification allows us to isolate the impact of the reform from i) idiosyncratic trends in non-covered diseases and ii) shocks that are specific to covered diseases (and common across countries). Reassuringly and consistent with columns (2) and (3), we find that the negative impact of the reform on deaths is significant in Chile but not in other countries. Finally, in column (5), we compare the evolution of mortality in non-covered diseases in Chile to those of other countries. For this, we interact an indicator variable equal to one for "Chile" with an indicator variable equal to one for the period after 2004 (when the reform started).[40] In line with our previous results, we cannot reject the null of a zero impact of the reform on non-covered diseases.[41]

Taking stock of our results: Our analysis thus far reveals that the healthcare reform that standardized procedures and guaranteed the timely coverage of a set of diseases led to a 4.4% reduction in deaths from covered diseases. We also find that the impact of the reform was larger on diseases that were more amenable to health care. Importantly, a battery of empirical exercises suggests that our result is not driven by increased mortality in the never-covered set of diseases. We find similar results when we estimate alternative models allowing flexible age-group and disease-specific time trends and when we leverage only variation in the timing of adoption among covered diseases for identification. Moreover, WHO data shows no indication of an abnormal increase in mortality from non-covered diseases in Chile, relative to comparable countries, after the reform.

Based on our results, we conjecture that this reform was highly cost-effective. Our

[40] In addition to year and cell fixed effects, this specification also includes an indicator variable equal to one for Chile.

[41] The estimated impact is not negligible (2.3%), but, despite a large number of observations (> 1 million), it is very noisy. In unreported results, we estimate this regression several times to compare deaths in Chile to deaths in other countries, one at a time. For 5 (out of 9) countries, we find a relative *decrease* in deaths in non-covered diseases in Chile after 2004.

estimated impact on mortality and estimates of the median value of a statistical life in Chile suggest that the reform created benefits of around USD $5.7 billion per year.[42] Using the surge in the value-added tax that funded the reform as a proxy for its cost, a back-of-the-envelope calculation (that abstracts, for instance, from distortions caused by increased taxation) suggests that the benefits of the reform outweighed the costs by a factor of four.[43]

5 Mechanisms: Impact on Procedures

The GES reform ensured, for the first time in Chile, a standardized benefit plan that granted equal entitlement to beneficiaries of public and private insurers by introducing mandatory guidelines for providers to guarantee timely access to high-quality care for a set of diseases. We have documented the beneficial effects of this reform on mortality. In this section, we assess if the reform led to equalization of utilization rates, as expected, given its emphasis on standardization of procedures.

Unfortunately, detailed procedure data is unavailable within our study's time frame. However, inpatient records report whether a patient had surgery, which we use as a proxy for procedures.[44] In Table 3, we present the estimates obtained from our preferred Poisson model given by equation (1), now using the count of inpatient surgeries as the dependent variable. Column (1) shows that surgeries increased by 15% due to the reform. Albeit sizable (as a percentage increase among covered diseases), the surge in covered surgeries implies a 4% increase in overall surgeries, i.e., the 15% increase represents around 2,800 extra inpatient surgeries per year, and the average number of surgeries per year in the country is around 70,000. Regarding heterogeneous effects, columns (2) to (5) of Table 3 show that the impact of the reform on inpatient surgeries is larger for males than for females (20% vs. 13%) and entirely driven by public hospitals (i.e., the estimated impact

[42] The Value of Statistical Life in Chile, in U.S. dollars of 2022, ranges between $0,50 to $6,33 million depending on the method and purpose (Mardones and Riquelme, 2018; Parada-Contzen, 2019). For the interested reader, Appendix B offers details on this back-of-the-envelope calculation.

[43] We compute the number of deaths that would have been saved one year before the GES Program using the 4.4% reductions in deaths. In our estimation sample, the covered group had 38,129 deaths in the pre-expansion period. Therefore, 1,678 deaths were averted once they went from uncovered to covered. We then combined them with the median value of statistical life and computed the benefits that would have been because of the 1,678 lives saved in the first year. For the interested reader, Appendix B offers details on this simple back-of-the-envelope.

[44] Notice that, insofar as many diseases covered by the reform do not require overnight surgery, our analysis cannot provide a complete picture of the impact of the reform on procedures.

of the reform on surgeries at private hospitals is indistinguishable from zero). The latter result, coupled with the fact that mortality decreased significantly more in public hospitals (see Appendix Table A.15) lead us to conjecture that access to more healthcare options was not a key driver of the reform's impact. [45] Reassuringly, the corresponding event studies presented in Appendix Figure A.10 show no evidence of pre-trends and indicate that surgeries increased steadily only after the reform.

To study whether a particular disease or set of diseases is driving the increase in surgeries, we repeat the sensitivity analysis from the previous section, in which we estimate our difference-in-differences model but remove one covered cell from the sample at a time. Appendix Figure A.11 plots the point estimates and 95% confidence intervals obtained from this exercise. Most estimates are around the average effect of a 15% increase of inpatient surgeries, except for the one obtained after removing the category of polytraumatized. As shown by columns (6) and (7) of Table 3, the impact of the reform on surgeries goes from 15% (p-val $<$ 0.01) to 5.6% (p-val $<$ 0.05) after removing this category. This result is consistent with the aim of the reform of guaranteeing timely access to care to anyone who presents traumatic injuries affecting at least two systems (of which the failure of one can be life-threatening).[46]

In light of these results, we zoom in on polytraumatized cases. Specifically, we leverage the discharge records and focus on inpatients with polytraumatism. Before the reform, procedures to treat polytraumatism were not uniform across providers; after it, clinical guidelines address the management of polytraumatized patients from the moment of rescue at the accident site until the completion of treatment in the intensive care unit, emphasizing damage control, care, and rehabilitation.

To assess the impact of the reform on utilization, we estimate hospitals' risk-adjusted surgery rates among inpatients before and after the reform's coverage of polytraumatisms. We estimate these risk-adjusted measures in the spirit of Card et al. (2023). Specifically, we consider the following logistic regression:

$$\mathrm{P}(\mathrm{Surgery}_{idht} = 1 \mid X_{iht}) = \Lambda(\beta X_{iht} + \gamma_t + \delta_h), \tag{5}$$

[45] This was guaranteed by the possibility of turning to private providers if treatment was not received within a certain time frame at public hospitals

[46] This finding is also consistent with Ramos et al. (2021), whose study of a landmark public hospital in Chile shows that more than 50% of polytraumatized patients receive surgery as part of their treatment.

where Λ is the logistic CDF and Surgery_{iht} is an indicator that equals one if patient i received surgery at hospital h in year t, and zero otherwise; X_{iht} is a vector of controls that includes the patient's sex, 22 age-groups and their interaction with a patient's sex, the patient's specific diagnoses within the broader polytraumatism category, and her type of insurance to proxy for socioeconomic determinants. We also control for hospital size (i.e., the number of discharges at time t) and year-fixed effects γ_t, to account for time-specific shocks common to all hospitals. The parameters of interest are the hospital fixed effects ($\hat{\delta}_h$), which we interpret as hospital risk-adjusted surgery rates.

For estimation, we restrict our sample to inpatients with polytraumatized health problems who stayed at hospitals that performed at least one surgery during the analysis period (2004-2009). We set the hospital with the highest number of surgeries in the whole period as the omitted category. Since the coverage of polytraumatized cases started in 2007, we estimate the model separately in 2004-2006 (48,909 observations) and 2007-2009 (63,455 observations).

Panel (a) of Figure 4 plots the distributions of the hospital's fixed effects estimated in each period. At the top left of each plot, we report the standard deviation of risk-adjusted surgery rates (i.e., hospital fixed effects) in each period. We find that the standard deviation decreased by 33% after reform coverage. We reject the null of the two-sample Kolmogorov–Smirnov test for the equality of these distributions and interpret this finding as evidence that there was standardization in the decision to perform surgery after the reform. We complement this exercise by estimating a variant of the model (5) that does not include hospital fixed effects but adds regional indicators instead. Then, we perform a simple between- and within-hospital variance decomposition using the patient-level residual from the model above as a proxy of the risk-adjusted likelihood of surgery. As shown by Appendix Figure A.12, and consistently with our previous result, we find that the total variance decreases after coverage, mostly driven by a decrease in the between-hospital variance component.

In Panel (b) of Figure 4, we repeat this exercise but replace the dependent variable of the model given by equation (5) for the logged spending associated with surgeries related to polytraumatisms.[47] Focusing exclusively on polytraumatized patients who had surgery, we find that the standard deviation of risk-adjusted spending decreased by 31.5% after reform

[47]To recover spending, we created a crosswalk between the surgery code and the procedure price set by the public health insurance FONASA.

coverage, although we fail to reject the null of the two-sample Kolmogorov–Smirnov test in this case. Overall, we interpret the effects on risk-adjusted surgery rates and spending as prima facie evidence that the reform equalized hospital utilization rates.

Additional non-causal evidence of the improvement in procedures comes from an early study by Nazzal et al. (2013). They studied 3,500 patients with myocardial infarction, which shows that—following the introduction of GES coverage—the use of thrombolysis went from 50% to 60%, and angioplasty procedures increased from 2.3% to 7.4%. The authors mention that when thrombolysis was not administered (after the GES program began), it was not due to a lack of medication, as was the case before the intervention. Instead, this delay could be attributed to an adaptation and learning period for healthcare workers as they became proficient in managing the newly accessible drug. This is consistent with Figure 2 in our manuscript, which shows that the protective effect of GES coverage strengthened over time. Furthermore, (Monsalves et al., 2020), who studies cervical, breast, and gallbladder cancers, and Mondschein et al. (2022) who studies colorectal cancer, included in the GES program only in 2013—and thus falls outside our study period— argues that GES coverage did not create sizable reductions in mortality due to the absence of early diagnosis through screening measures. In contrast to colorectal cancer, the cancers covered during our study period benefited from national screening programs or preventive measures.[48]

Notwithstanding, another component of the GES program was financial coverage, which in turn might have led to increased utilization. We provide suggestive evidence that limiting out-of-pocket expenditures was not a key driver of the decrease in mortality. Leveraging that lower-income patients in Chile have access to free healthcare in public hospitals (i.e., they have a zero co-payment policy), we can study the impact of the reform when the out-of-pocket component is muted. Specifically, leveraging in-patient records (which report the patients' insurance), we can replicate Figure 2 in a sample of copayment-exempt in-patients. Our results are presented in Appendix Figure A.13, and show that the reform had similar impacts on these lower-income patients, who de facto did not benefit from the limited out-of-pocket expenditures dimension of the GES reform.

Finally, based on our evidence and the program's guidelines, we conjecture that timely access to care was key in the case of polytraumatism, a disease for which treatment became

[48]For instance, the GES reform covered cervical cancer screening via Pap smears for women aged 25 to 65 and breast cancer screening through mammograms for women aged 50 to 59. Additionally, gallbladder cancer prevention was supported through cholecystectomy for individuals aged 35 to 49 with gallstones.

guaranteed within 24 hours from the time of rescue, even if it requires air transportation. For ischemic strokes, on the other hand, we conjecture that the bundle of timely access (e.g., initiating motor therapy within the first 24 hours) and the standardization of procedures across hospitals (e.g., performing an angiography of the brain and neck in addition to the CT scan) drove the decrease in mortality. Overall, given the available data, it is challenging to unpack the specific features of the GES Program that led to the observed decrease in mortality. However, our findings suggest that this reduction is primarily driven by the combined effect of improved access and timely care. This, in turn, results from the standardization of procedures outlined in the national clinical guidelines, which specify how to treat patients with particular diseases, using designated procedures and medications, all within a defined timeframe.[49]

6 Conclusion

Countries may follow different paths to improve their healthcare systems, depending on their economic and historical contexts (Lagomarsino et al., 2012; Atun et al., 2015; Reich et al., 2016). In this article, we studied the impact of a large health reform—layered over an existing "universal coverage" system—that standardized procedures and guaranteed medical treatment for sick patients independent of their insurance or income and based solely on their diagnoses and age group. Our results show that this reform led to a 4.4% decrease in deaths, implying that 1,678 deaths per year were averted thanks to this policy. This result is robust to several specification checks, and it is not driven by competing risks or a shift in healthcare resources from non-covered to covered diseases.

We also document that the reform led to a larger decrease in mortality among diseases that are more amenable to care and that it increased inpatient surgeries, especially among polytraumatized cases, a health problem for which we find suggestive evidence of equalization in utilization rates. Although we show that the reform decreased mortality, it is worth noticing that data limitations prevented us from an in-depth study of the reform's impact on procedures. Our paper is silent, for instance, on whether introducing medical guidelines and standardizing procedures reduced allocative inefficiencies, an important issue that we

[49]Regarding other aspects of the GES reform, such as quality, financial coverage and the possibility to refer patients from public to private health care facilities, we also provide suggestive evidence that indicates this were not features that play a fundamental role in achieving the decrease in mortality.

hope can be addressed in future work.

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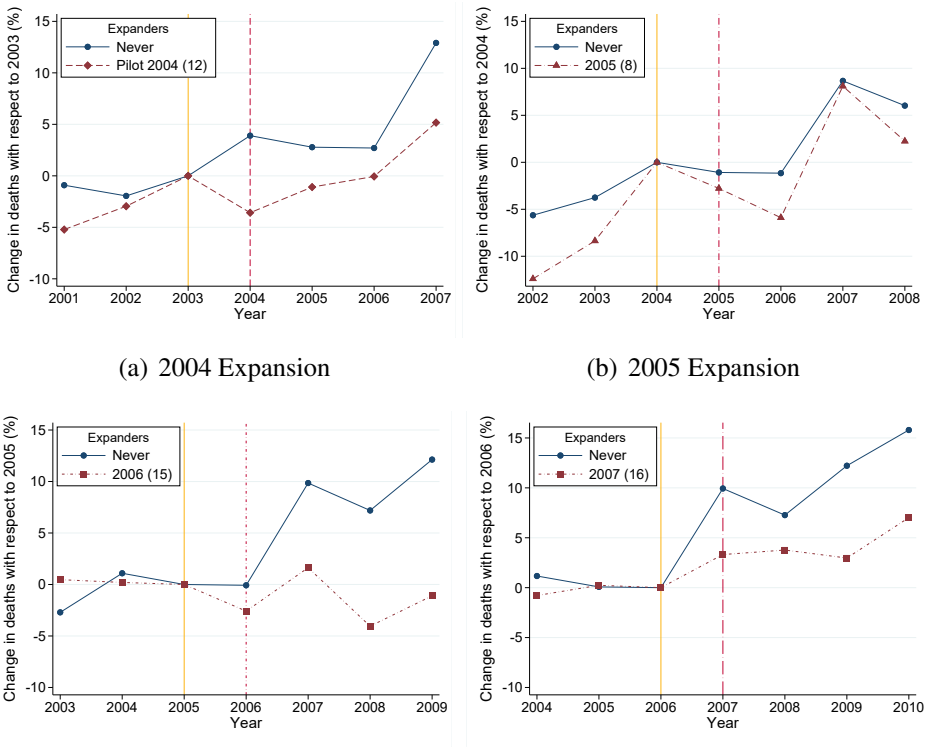
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Figure 1: Change in Deaths for Each GES Expansion



(a) 2004 Expansion

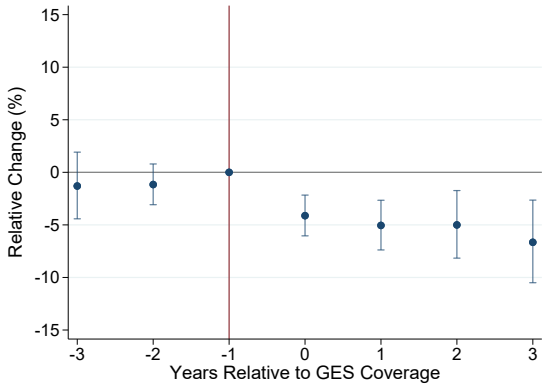
(b) 2005 Expansion

(c) 2006 Expansion

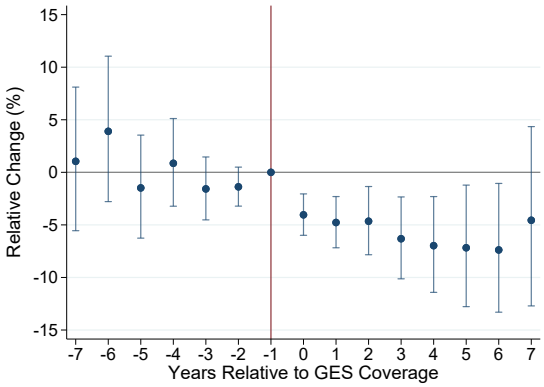
(d) 2007 Expansion

Notes: This figure uses raw data to show the change in deaths for the diseases covered by each GES expansion and the diseases never covered by the GES reform. All changes 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. The vertical dashed red line represents the first year of the expansion.

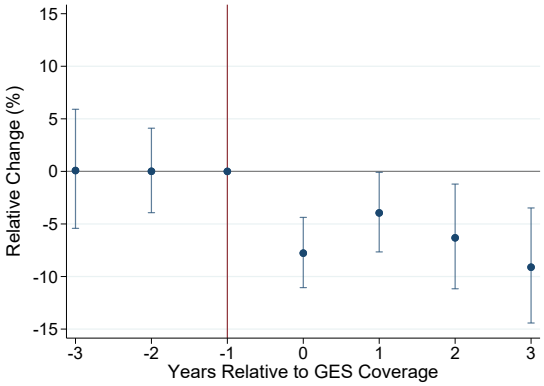
Figure 2: Event Studies: GES Impact on Deaths



(a) All Deaths



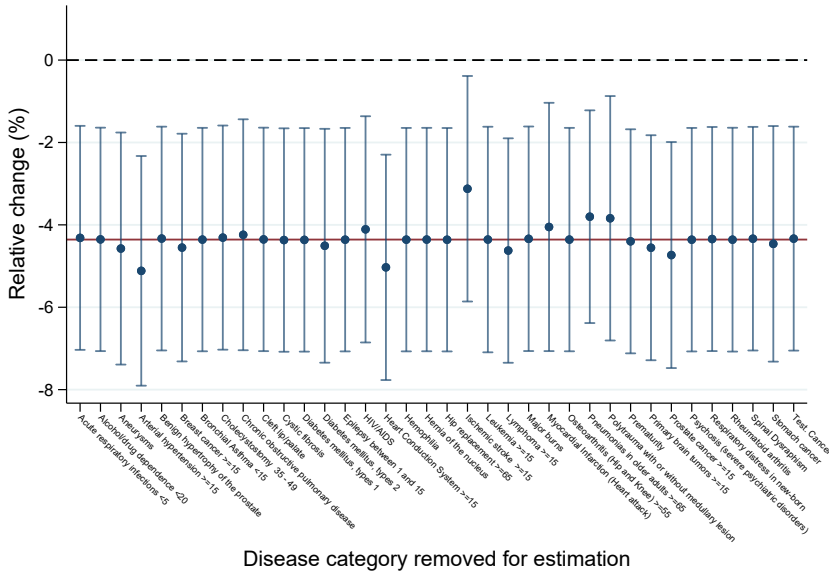
(b) All Deaths (7-year window)



(c) In-Hospital Deaths

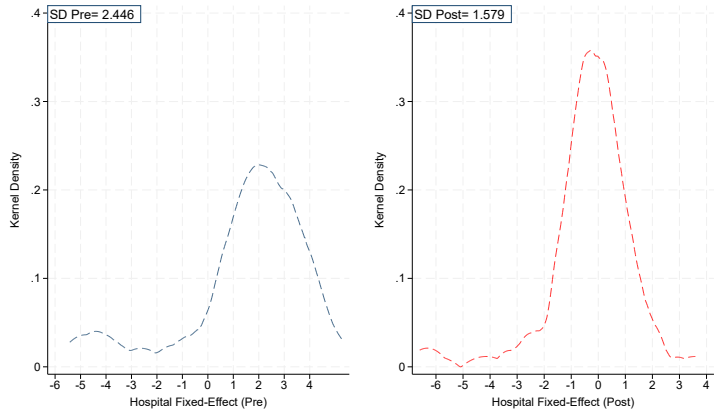
Notes: This figure shows the results obtained from estimating the dynamic difference-in-differences using the count of deaths as the dependent variable in a Poisson regression. For figures in panels (a) and (b), we used data from 1997-2014 available in the death records, and we binned up endpoints for (a). In panel (c), we use data from 2001-2010 that is available in the discharged records. 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, i.e., $\hat{\theta}_{ATT\%} = exp(\hat{\beta}) - 1$. Each estimate captures the effect in each period relative to one year before each group of diseases started to be covered. 95% confidence intervals for $\hat{\theta}_{ATT\%}$ are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression.

Figure 3: Sensitivity of the Impact on Death to Targeted Diseases

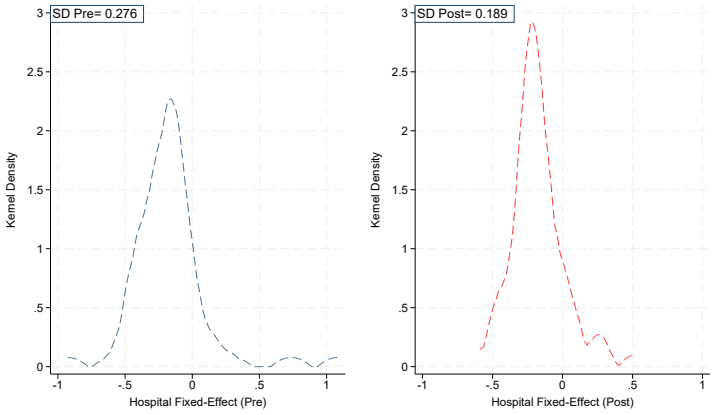


Notes: This figure shows the results obtained from estimating (several times) the dynamic difference-in-differences presented in equation (4) using the count of deaths as the dependent variable in a Poisson regression. Each point estimate and confidence interval comes from a regression in which we remove 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, i.e., $\hat{\theta}_{ATT\%} = exp(\hat{\beta}) - 1$. Each estimate captures the effect in each period relative to one year before each group of diseases started to be covered. 95% confidence intervals for $\hat{\theta}_{ATT\%}$ are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression.

Figure 4: Distribution of Risk-Adjusted Hospital Surgery Rates and Spending



(a) Risk-Adjusted Surgery Rates



(b) Risk-Adjusted Log Spending

Notes: This figure shows the risk-adjusted hospital fixed effects distribution. Estimates come from the logistic regression presented in equation (5), estimated separately in the pre- and post-reform periods. Panel (a) presents estimates obtained when we use surgery rates, and Panel (b) presents estimates obtained when we use spending.

Table 1: GES Impact on Deaths

	Main			Amenable Disease Interaction		
	All Deaths	Ever Covered	In Hospital	All Deaths	Ever Covered	In Hospital
	(1)	(2)	(3)	(4)	(5)	(6)
After GES Expansion	-0.044***	-0.040***	-0.069***	-0.022	-0.018	-0.033
	(0.014)	(0.010)	(0.020)	(0.016)	(0.013)	(0.024)
After GES Expansion × More Amenable				-0.086***	-0.095***	-0.128***
				(0.026)	(0.026)	(0.036)
No. Deaths	521,300	264,974	172,940	521,300	264,974	172,940
No. Deaths ∈ covered diseases (year before coverage)	38,129	38,129	10,773	38,129	38,129	10,773
Total No. disease-age cells (obs.)	99,146	24,906	81,654	99,146	24,906	81,654

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* and its interaction correspond to percent changes by subtracting 1 from the rate ratio, i.e., $\hat{\theta}_{ATT\%} = exp(\hat{\beta}) - 1$. The Poisson estimation drops disease-age cells with zero outcomes in the study period. Standard errors for $\hat{\theta}_{ATT\%}$ 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 Deaths Using WHO Mortality Database

	Diagnoses-Age Categories				
	All	Ever covered			Non-covered
	Chile	Chile	Other countries	All countries	All countries
	(1)	(2)	(3)	(4)	(5)
After GES Expansion	-0.036**	-0.039***	-0.010	-0.010	
	(0.015)	(0.011)	(0.009)	(0.009)	
After GES Expansion × Chile				-0.029**	
				(0.015)	
After 2004 × Chile					0.023
					(0.021)
Total No. disease-age cells (obs.)	83,390	16,520	125,678	142,198	1,045,860

Notes: This table shows the results from different Poisson regressions using death counts from the WHO Mortality dataset. All regressions control for disease-age cell fixed effects and year fixed effects. In addition, columns (3) and (4) use disease-age cell fixed effects, and year-fixed effects interacted with country-fixed effects. Column (1) considers data for Chile, including covered and non-covered diseases. Column (2) considers data for Chile, including only ever-covered diseases. Columns (3), (4), and (5) also use data from other countries; columns (3) and (4) include only covered diseases, while column (5) includes only non-covered diseases. All coefficients correspond to percent changes by subtracting one from the rate ratio, i.e., $\hat{\theta}_{ATT\%} = exp(\hat{\beta}) - 1$. Standard errors are clustered at the level of treatment: disease-age in columns (1) and (2), diseases-age-country in columns (3) and (4), and disease-age-Chile indicator in column (5). Standard errors for $\hat{\theta}_{ATT\%}$ 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 3: GES Impact on Inpatient Surgeries

		Sex		Type of Hospital		Polytraumatized	
	All	Female	Male	Public	Private	Yes	No
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
After GES Expansion	0.151***	0.126***	0.200***	0.215***	0.004	0.339***	0.056**
	(0.032)	(0.028)	(0.041)	(0.036)	(0.030)	(0.065)	(0.029)
No. Surgeries	761,472	385,206	376,266	540,618	220,854	647,088	690,943
No. Surgeries ∈ covered diseases (year before coverage)	18,718	8,083	10,635	13,444	5,274	6,341	12,377
Total No. disease-age cells (obs.)	105,510	84,560	84,296	94,385	73,134	90,012	97,558

Notes: This table shows the results obtained from estimating the staggered difference-in-differences presented in equation (1) using Poisson regressions based 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 one from the rate ratio, i.e., $\hat{\theta}_{ATT\%} = exp(\hat{\beta}) - 1$. The Poisson estimation drops disease-age cells with zero outcomes in the study period. Standard errors for $\hat{\theta}_{ATT\%}$ 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.

Appendix

The Impact of Standardized Disease-Specific Healthcare Coverage

Felipe Menares and Pablo Muñoz

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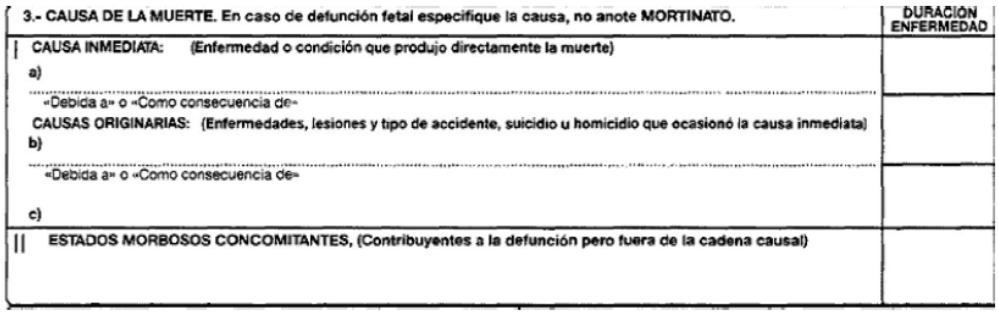
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Appendix A: Additional Figures and Tables

Figure A.1: Protocol to Record Deaths

Figura 1. Sección 3 "Causa de la muerte", del formulario del Certificado Médico de Defunción vigente en Chile.



3.- CAUSA DE LA MUERTE. En caso de defunción fetal especifique la causa, no anote MORTINATO.	DURACIÓN ENFERMEDAD
I CAUSA INMEDIATA: (Enfermedad o condición que produjo directamente la muerte) a)	
«Debida a» o «Como consecuencia de» CAUSAS ORIGINARIAS: (Enfermedades, lesiones y tipo de accidente, suicidio u homicidio que ocasionó la causa inmediata) b)	
«Debida a» o «Como consecuencia de» c)	
II ESTADOS MORBOSOS CONCOMITANTES, (Contribuyentes a la defunción pero fuera de la cadena causal)	

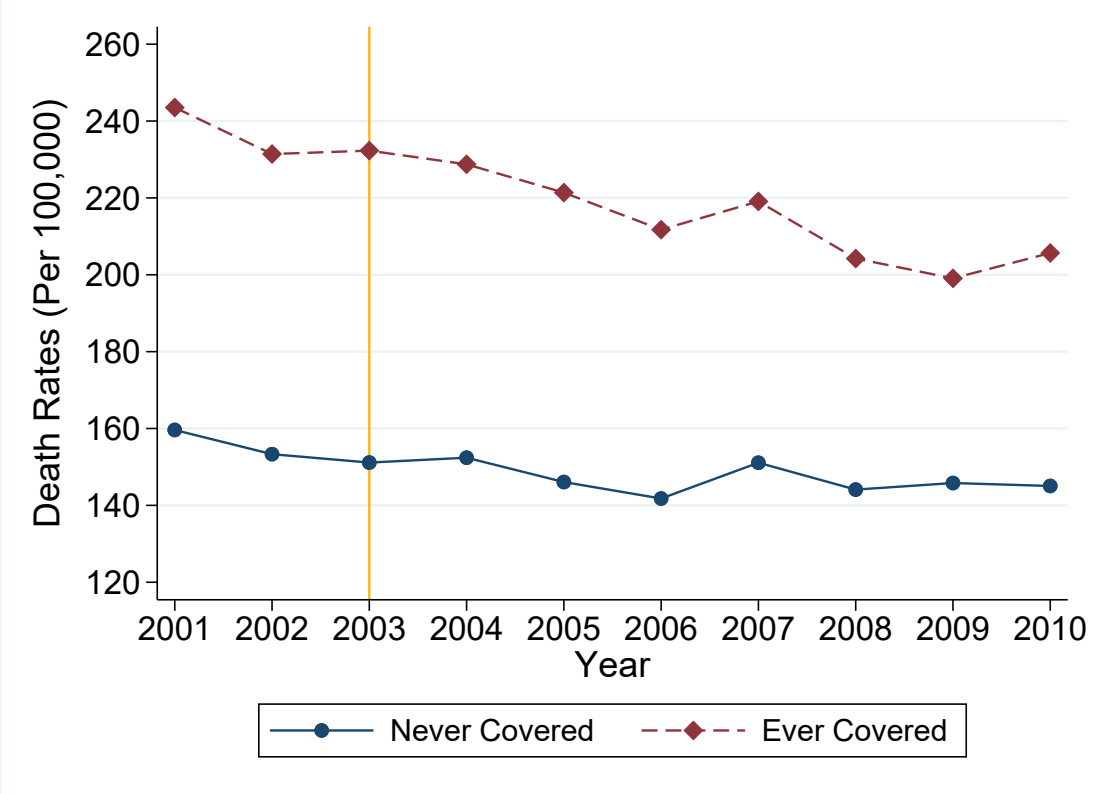
El fin de las estadísticas de causas de muerte es contar con información que permita prevenir la muerte, para lo cual es necesario identificar aquella causa que dio origen a la cadena de eventos que condujo a la muerte y que se tabulará como la *causa básica* de defunción, definida como:

"(a) la **enfermedad o lesión** que **inició** la cadena de acontecimientos patológicos que condujeron directamente a la muerte, o

(b) las circunstancias del **accidente o violencia** que produjo la lesión fatal".

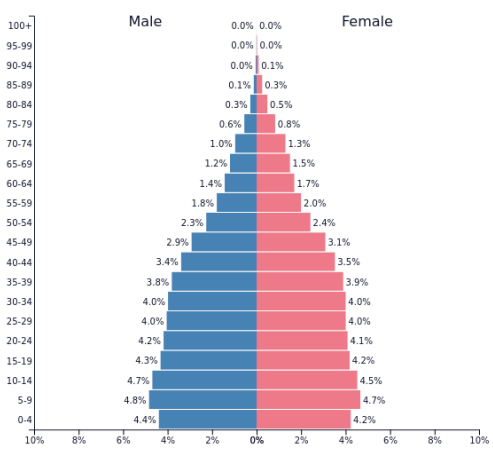
Notes: This figure shows an excerpt from the document "Manual of the Correct Filling of the Medical Certificate of Death" (Antini, 2020). It reads: *The purpose of statistics on causes of death is to have information that allows death to be prevented, for which it is necessary to identify the cause that gave rise to the chain of events that led to death and that will be tabulated as the basic cause of death.* Thus, causes (ICD-10 codes) recorded in our data should reflect the original cause of death.

Figure A.2: Age Standardized Cause-Specific Death Rate

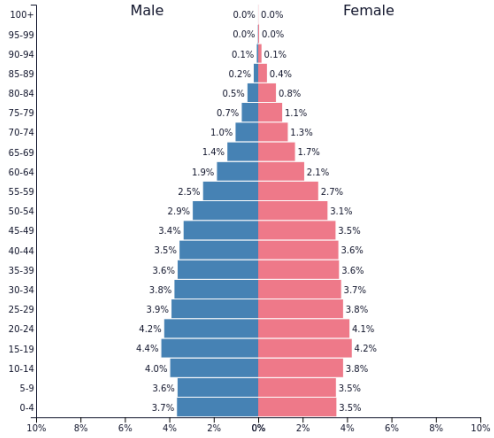


Notes: This figure shows the decrease in cause-specific death rates for ever-covered and never-covered diseases; in this case, all rates are standardized using the 2001 age distribution to account for the population's age structure. To adjust death rates, we proceed in the following way: i) we calculate 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 vertical solid yellow line represents one year before the expansion. This figure is constructed using data from the Death Registry, Vital Statistics, Census, and GES eligibility rules.

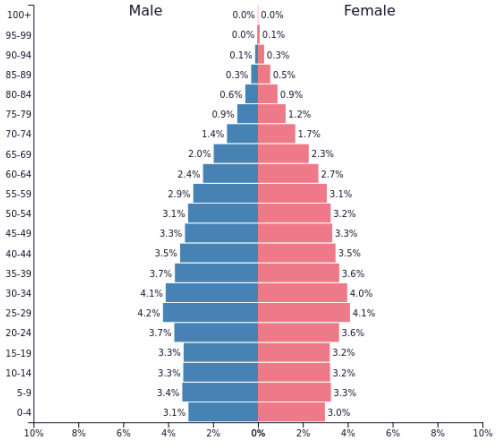
Figure A.3: Population Pyramids



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



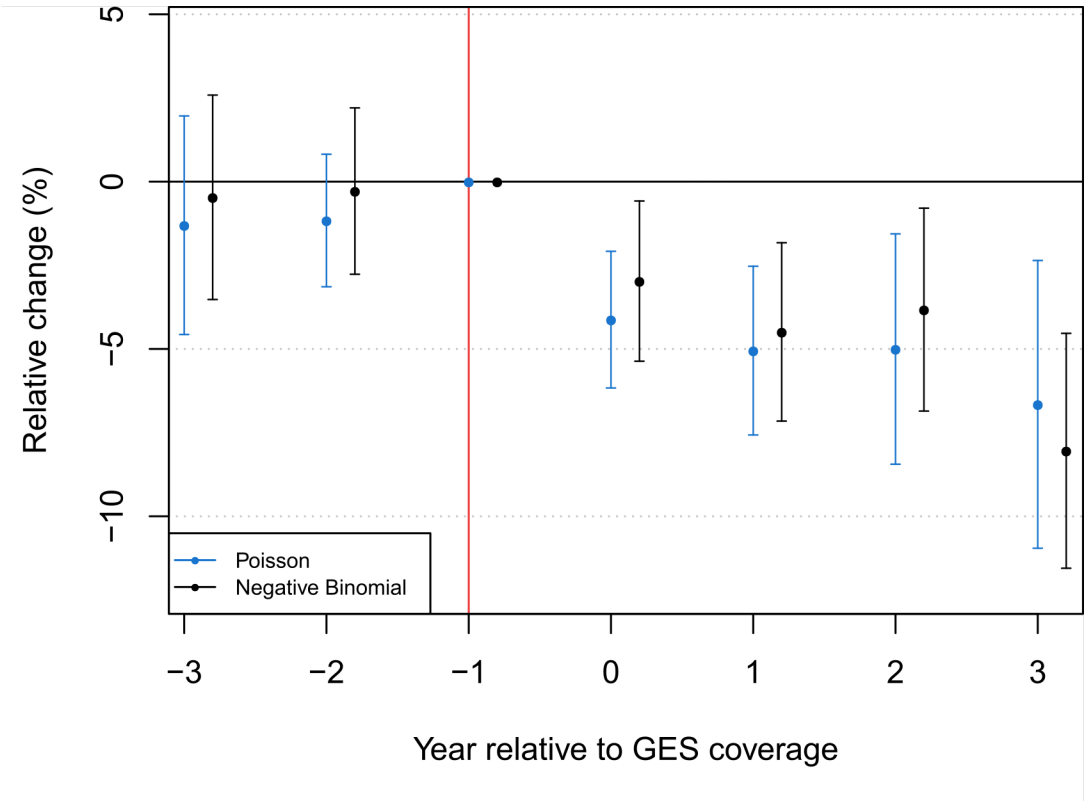
(b) 2010, Pop: 17,062,531



(c) 2020, Pop 19,611,208

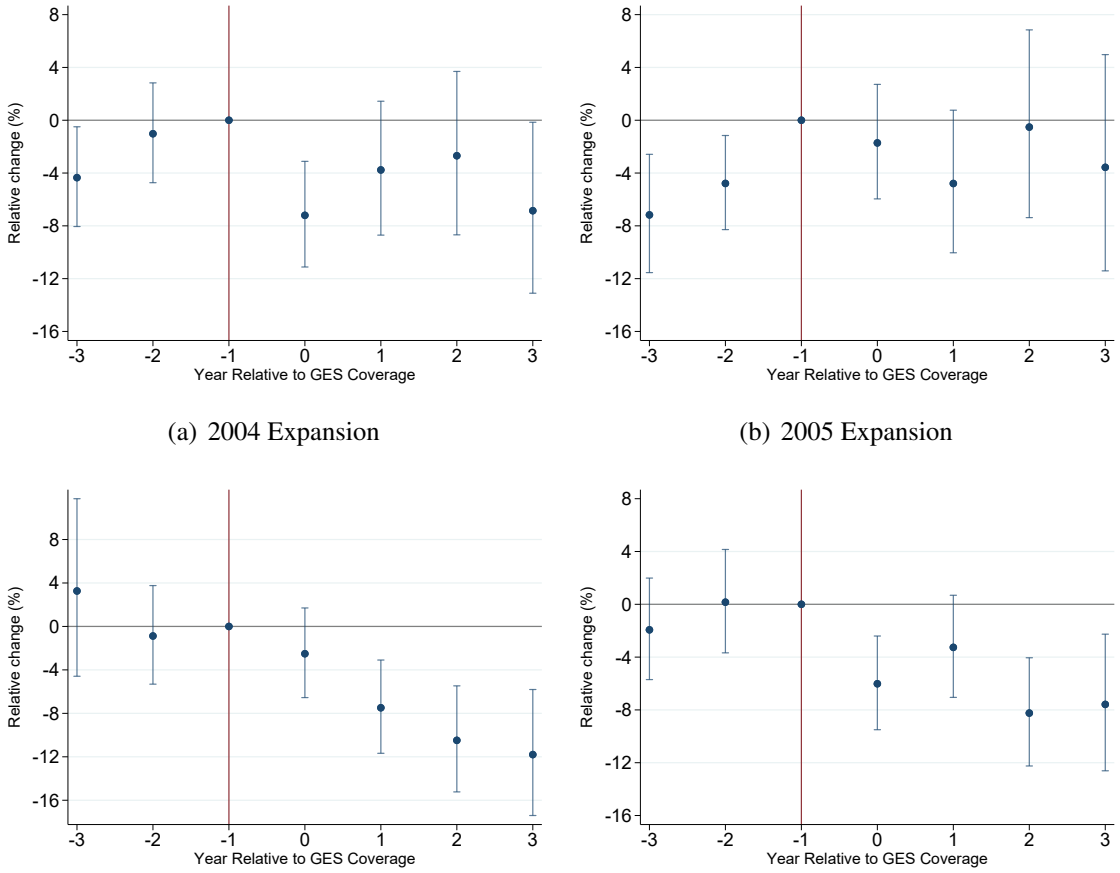
Notes: This figure shows Chile's population pyramids in 2000, 2010, and 2020. Source: Pyramids.net.

Figure A.4: 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 (4) 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 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. The negative Binomial model was estimated using R's fixest package

Figure A.5: Event Study: GES Impact on Deaths, by Expansion



(a) 2004 Expansion

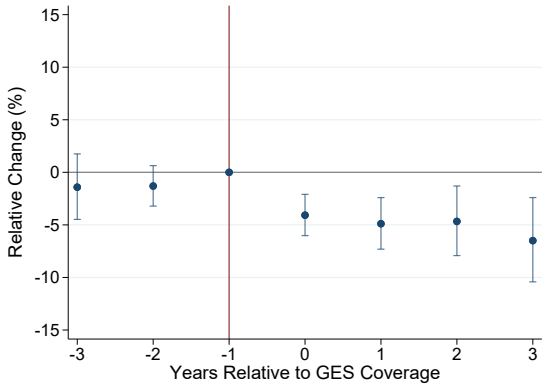
(b) 2005 Expansion

(c) 2006 Expansion

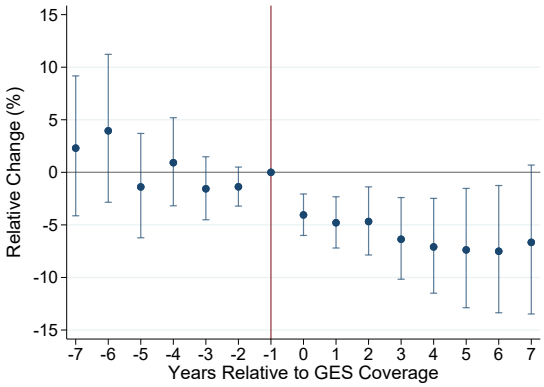
(d) 2007 Expansion

Notes: These figures show the coefficients obtained from estimating the dynamic difference-in-differences presented in equation (4). Each regression considers each expansion independently. 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 one 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.

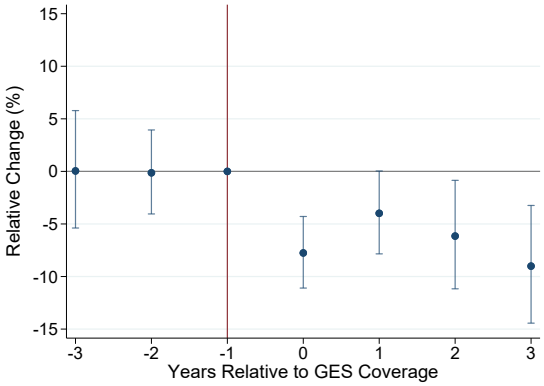
Figure A.6: Event Studies: GES Impact on Deaths (Non-Binning)



(a) All Deaths (3-year window)



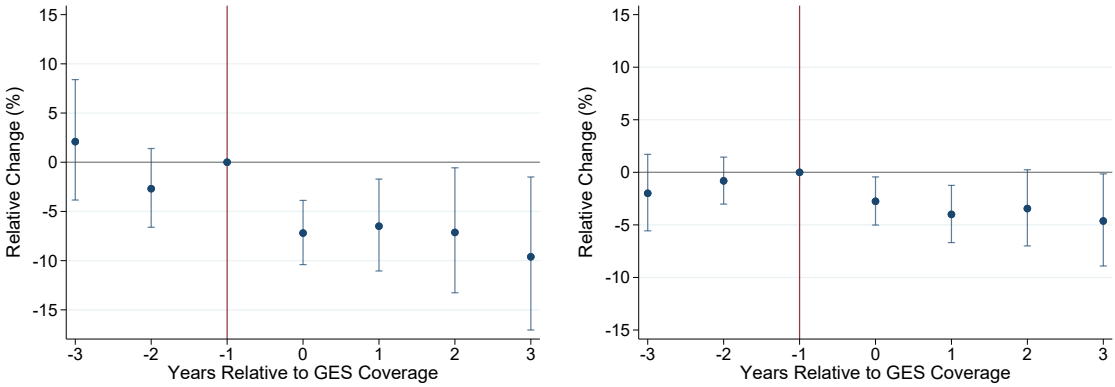
(b) All Deaths (7-year window)



(c) In-Hospital Deaths (3-year window)

Notes: This figure shows the results obtained from estimating the dynamic difference-in-differences using the count of deaths as the dependent variable in a Poisson regression. Following Borusyak et al. (2024), we do not bin up the endpoints for this exercise. Instead, we consider all the available leads and lags to allow the model to capture the full dynamic of the dependent variable and plot the coefficients within our window of interest, as in Atal et al. (2024). For the figure in panel (b), we used data from 1997-2014 in the death records. In panel (a) and (c), we use data from 2001-2010 that is available in the discharged records. 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, i.e., $\hat{\theta}_{ATT\%} = exp(\hat{\beta}) - 1$. Each estimate captures the effect in each period relative to one year before each group of diseases started to be covered. 95% confidence intervals for $\hat{\theta}_{ATT\%}$ are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression.

Figure A.7: Event Study: GES Impact on *More* and *Less* Amenable Deaths

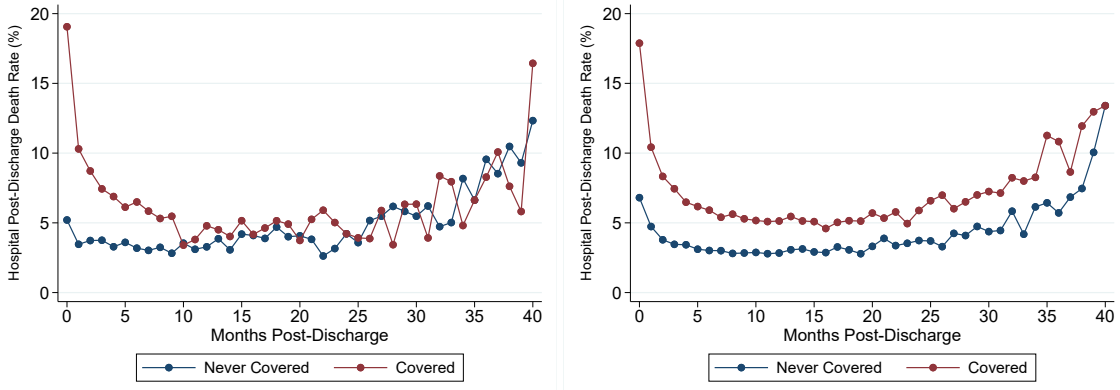


(a) More amenable

(b) Less amenable

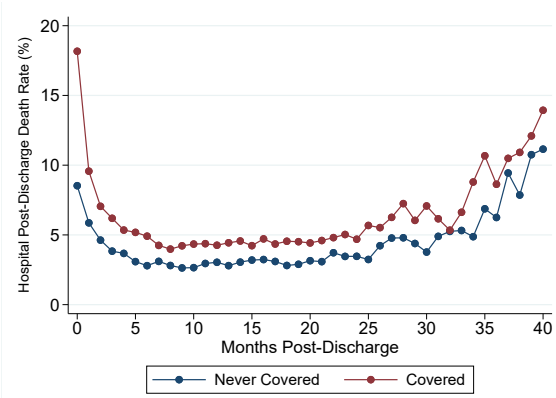
Notes: This figure shows the results obtained from estimating the dynamic difference-in-differences presented in equation (4) using the count of deaths as the dependent variable in a Poisson regression. Panel (a) shows the event study for deaths more amenable to health care (Nolte and McKee, 2011; Sommers et al., 2014). Panel (b) shows the event study for the deaths less amenable to health care. For details about the Amenable classification, see Online Appendix Table **??**. 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, i.e., $\hat{\theta}_{ATT\%} = exp(\hat{\beta}) - 1$. Each estimate captures the effect in each period relative to one year before each group of diseases started to be covered. 95% confidence intervals for $\hat{\theta}_{ATT\%}$ are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression.

Figure A.8: Hospital Post-Discharge Death Rate by Age Groups



(a) Ages 0 to 49

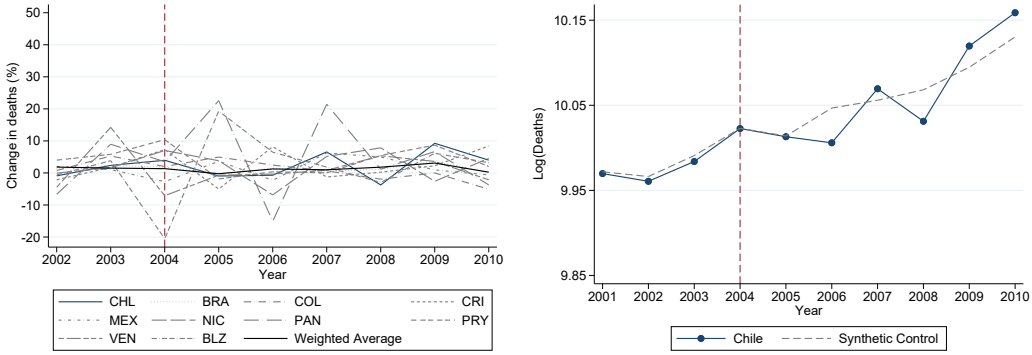
(b) Ages 50 to 79



(c) Ages above 80

Notes: This figure shows the 40-month post-intervention hospital post-discharge death rate. The numerator includes all deaths, both in-hospital and out-of-hospital. The denominator considers only patients discharged alive for diseases covered after the program implementation. We plot separate death rates for the covered and non-covered groups of diseases. We compute the rates for the 40 months as the ratio of the following counts: $\frac{Deaths_{g,a,m}}{Post-Discharges_{g,a,m}}$, where g = covered or never - convered; a= age groups, and m = months after discharge.

Figure A.9: Change in Deaths for Never-Covered Diseases in Chile and Latin America

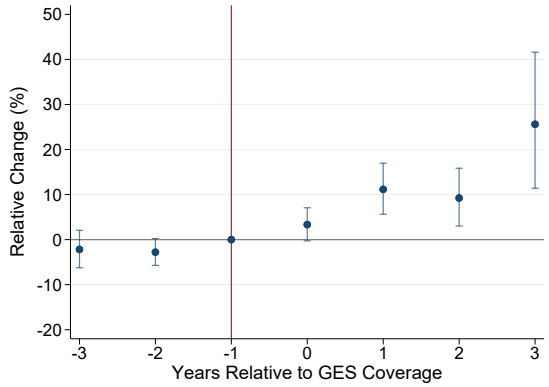


(a) Yearly change in deaths

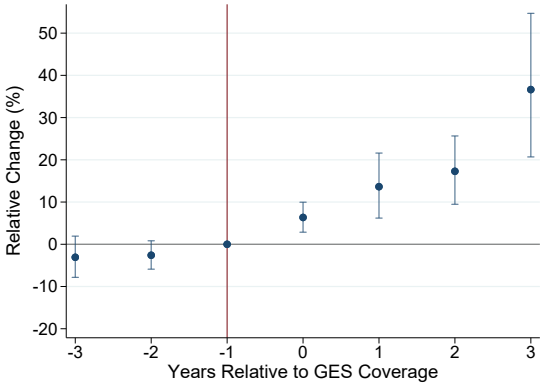
(b) Synthetic Control

Notes: This figure shows the time trends of deaths in uncovered diseases for different countries. Panel (a) reports the percentage changes in deaths. The weighted average line shows the sum of countries' deaths using their contribution to total deaths (across countries) as weights. Panel (b) shows the result from a synthetic control analysis that uses the log number of deaths in non-covered diseases as the main outcome. The vertical dashed red line represents the year the reform coverage started in Chile. Selected countries are those with high-quality mortality data under the World Health Organization classification. See the main text for details.

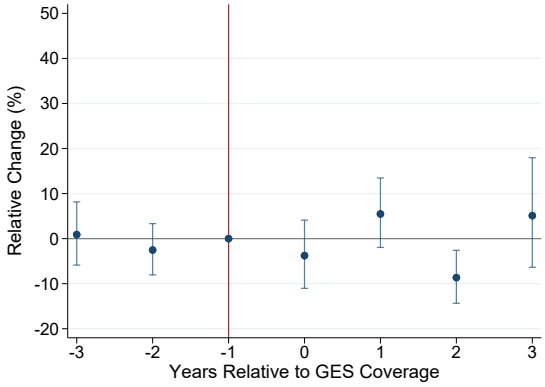
Figure A.10: Event Study: GES Impact on Inpatient Surgeries



(a) All



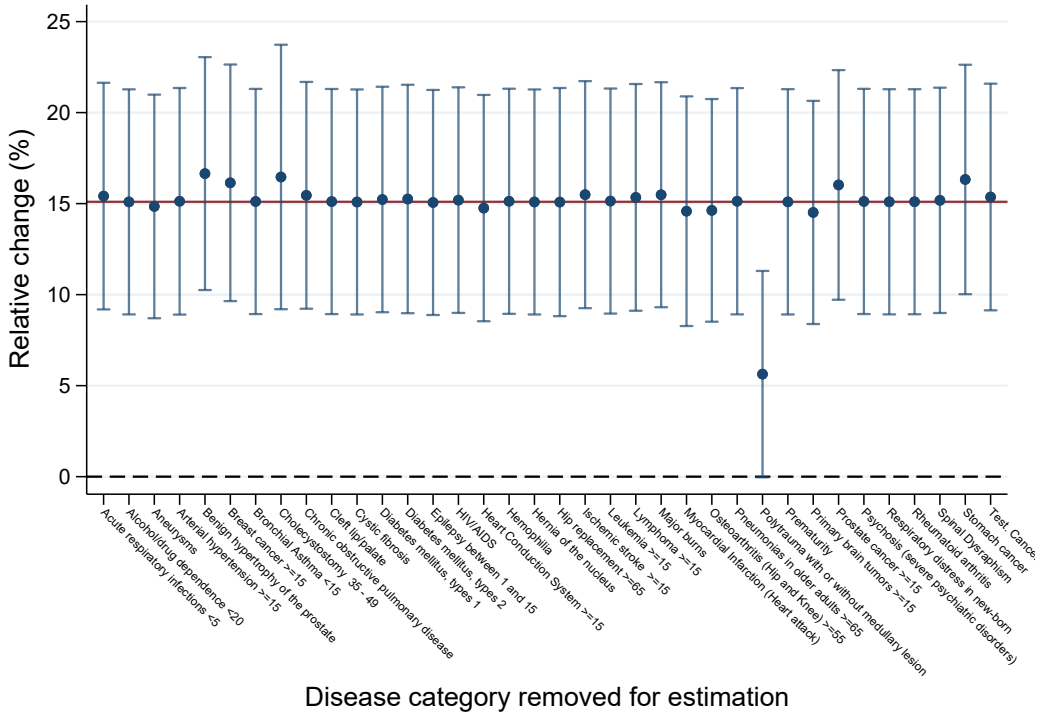
(b) Public Hospitals



(c) Private Hospitals

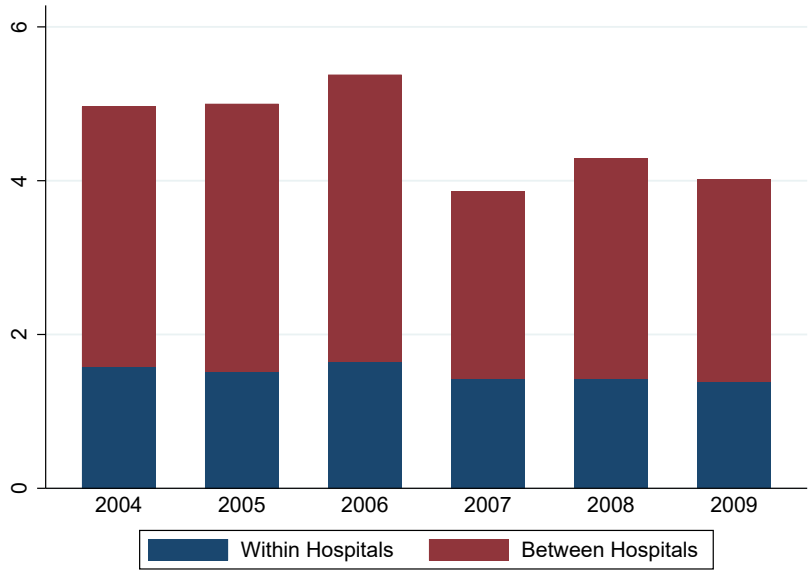
Notes: These figures show the results obtained from estimating the dynamic difference-in-differences presented in equation (4) using the count of in-hospital deaths and in-hospital surgeries as dependent variables in Poisson regressions. All regressions control for disease-age cell and year-fixed effects. 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. 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.

Figure A.11: Sensitivity of the Impact on Surgeries to Targeted Diseases



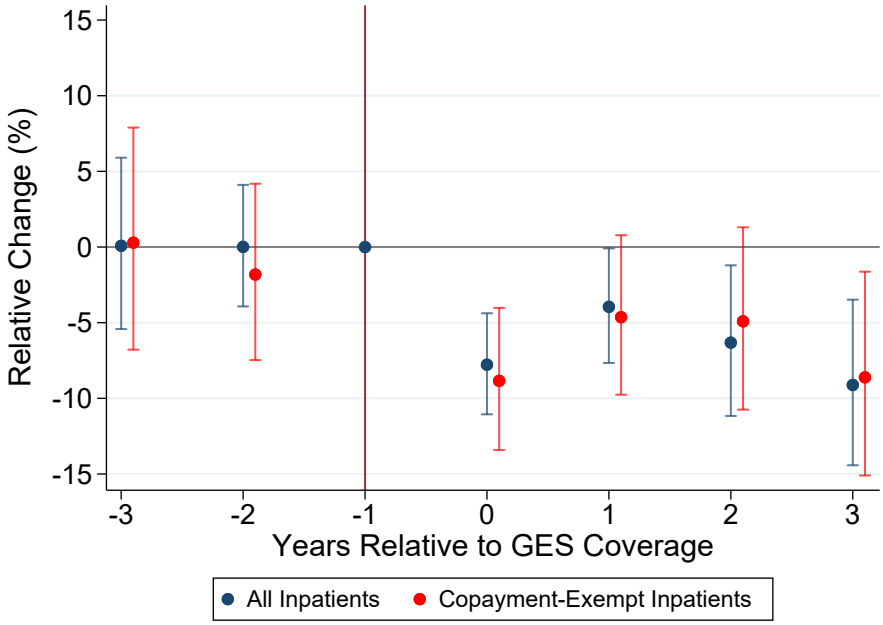
Notes: This figure shows the results obtained from estimating (several times) the dynamic difference-in-differences presented in equation (4) using the count of surgeries as the dependent variable in a Poisson regression. Each point estimate and confidence interval comes from a regression in which we 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 A.12: Variance Decomposition of the Risk-Adjusted Likelihood of Surgery



Notes: This figure shows the variance decomposition of the inpatient-level residual obtained from a variant of the logistic model given by equation (5) that removes hospital fixed effects and adds regional indicators instead.

Figure A.13: Impact of the GES Program on In-Hospital Deaths by Type of Co-payment



Notes: This figure shows the results obtained from estimating the dynamic difference-in-differences using the count of deaths as the dependent variable in a Poisson regression. All Inpatients point estimates replicates Figure 2 (c), for In-Hospital deaths. Copayment-Exempt Inpatients point estimates are for low-income patients already covered without copayment before the intervention. 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, i.e., $\hat{\theta}_{ATT\%} = exp(\hat{\beta}) - 1$. Each estimate captures the effect in each period relative to one year before each group of diseases started to be covered. 95% confidence intervals for $\hat{\theta}_{ATT\%}$ are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression.

Table A.1: Health Related Problems: Pilot 2004

Health Related Problem	Deaths	%
Myocardial Infarction (Heart attack)	41,358	72.32
Breast cancer (15+ years old)	7,753	13.56
Lymphoma (15+ years old)	3,813	6.67
HIV/AIDS	2,948	5.15
Testicular cancer (15+ years old)	665	1.16
Diabetes mellitus, types 1	219	0.38
Psychosis (severe psychiatric disorders)	176	0.31
Spinal Dysraphism	161	0.28
Hip replacement (65+ years old)	50	0.09
Cleft lip/palate	45	0.08
Total	57,188	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)	19,559	27.61
Diabetes mellitus, types 2	19,589	27.65
Arterial hypertension (15+ years old)	18,418	26.00
Heart Conduction System (15+ years old)	10,666	15.06
Prematurity	1,823	2.57
Acute respiratory infections (5- years old)	700	0.99
Epilepsy (between 1 and 15 years old)	88	0.12
Total	70,843	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)	24,402	30.60
Stomach cancer	21,851	27.40
Chronic obstructive pulmonary disease	19,586	24.56
Prostate cancer (15+ years old)	11,115	13.94
Respiratory distress in new-born	1,171	1.47
Cholecystostomy (between 35 to 49 years old)	1,034	1.30
Benign hypertrophy of the prostate	557	0.70
Hemophilia	25	0.03
Bronchial Asthma (15- years old)	9	0.01
Total	79,750	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	30,096	52.62
Aneurysms	16,252	28.42
Primary brain tumors (15+ years old)	4,133	7.23
Leukemia (15+ years old)	3,811	6.66
Major burns	2,000	3.50
Rheumatoid arthritis	773	1.35
Cystic fibrosis	118	0.21
Alcohol/drug dependence (20- years old)	5	0.01
Osteoarthritis (Hip and Knee) (55+ years old)	3	0.01
Total	57,191	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 More 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 more 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. We follow Nolte and McKee (2003) an set an age limit of 75 for those without it.

Table A.6: Amenable Deaths by Health-Related Problems

	Amenable Deaths				All
	Less		More		Deaths
	No. (1)	**Col. %** (2)	**No.** (3)	**Col. %** (4)	**No.** (5)
All GES Diseases	202,904	76.58	62,070	23.42	264,974
All Non GES Diseases	221,430	86.39	34,896	13.61	256,326
All Diseases	424,334	81.40	96,966	18.60	521,300
Panel A: 2004 Expansion					
Myocardial Infarction (Heart attack)	20,146	48.71	21,212	51.29	41,358
Breast cancer (15+ years old)	2,352	30.34	5,401	69.66	7,753
Lymphoma (15+ years old)	3,556	93.26	257	6.74	3,813
HIV/AIDS	2,948	100.00	-	0.00	2,948
Testicular cancer (15+ years old)	41	6.17	624	93.83	665
Diabetes mellitus, types 1	169	77.17	50	22.83	219
Psychosis (severe psychiatric disorders)	176	100.00	-	0.00	176
Spinal Dysraphism	161	100.00	-	0.00	161
Hip replacement (65+ years old)	50	100.00	-	0.00	50
Cleft lip/palate	45	100.00	-	0.00	45
Total	29,644	51.84	27,544	48.16	57,188
Panel B: 2005 Expansion					
Pneumonias in older adults (65+ years old)	17,168	87.78	2,391	12.22	19,559
Diabetes mellitus, types 2	18,972	96.85	617	3.15	19,589
Arterial hypertension (15+ years old)	13,677	74.26	4,741	25.74	18,418
Heart Conduction System (15+ years old)	6,577	61.66	4,089	38.34	10,666
Prematurity	-	0.00	1,823	100.00	1,823
Acute respiratory infections (5- years old)	-	0.00	700	100.00	700
Epilepsy (between 1 and 15 years old)	-	0.00	88	100.00	88
Total	56,394	79.60	14,449	20.40	70,843
Panel C: 2006 Expansion					
Ischemic stroke (15+ years old)	16,654	68.25	7,748	31.75	24,402
Stomach cancer	21,851	100.00	-	0.00	21,851
Chronic obstructive pulmonary disease	19,503	99.58	83	0.42	19,586
Prostate cancer (15+ years old)	11,115	100.00	-	0.00	11,115
Respiratory distress in new-born	215	18.36	956	81.64	1,171
Cholecystostomy (between 35 to 49 years old)	979	94.68	55	5.32	1,034
Benign hypertrophy of the prostate	442	79.35	115	20.65	557
Hemophilia	25	100.00	-	0.00	25
Bronchial Asthma (15- years old)	-	0.00	9	100.00	9
Total	70,784	88.76	8,966	11.24	79,750
Panel D: 2007 Expansion					
Polytrauma with or without medullary lesion	30,096	100.00	-	0.00	30,096
Aneurysms	5,978	36.78	10,274	63.22	16,252
Primary brain tumors (15+ years old)	4,133	100.00	-	0.00	4,133
Leukemia (15+ years old)	2,974	78.04	837	21.96	3,811
Major burns	2,000	100.00	-	0.00	2,000
Rheumatoid arthritis	773	100.00	-	0.00	773
Cystic fibrosis	118	100.00	-	0.00	118
Alcohol/drug dependence (20- years old)	5	100.00	-	0.00	5
Osteoarthritis (Hip and Knee) (55+ years old)	3	100.00	-	0.00	3
Hernia of the nucleus	2	66.67	-	0.00	3
Total	46,082	80.57	11,111	19.43	57,193

Notes: This table shows the number of more and less amenable deaths by the health-related problem for each of the waves of expansion in the GES program.

Table A.7: Targeted Diseases, Targeted Cells (Disease-Age Groups), and the Total Number of Deaths

	Deaths			In-Hospital	
	All	Amenable	Less amenable	Deaths	Surgeries
	(1)	(2)	(3)	(4)	(5)
Panel A: Diseases (ICD-10)					
Total	1,027	317	944	1,017	1,001
Covered	315	132	284	308	309
Uncovered	763	227	668	756	741
Panel B: Disease-Age Cells					
Total	10,982	2,057	8,925	9,027	11,555
Covered	3,558	778	2,780	2,872	3,349
Uncovered	7,424	1,279	6,145	6,155	8,206
Panel C: No. of Deaths					
Total	521,300	96,966	424,334	172,940	761,376
Covered	264,974	62,070	202,904	77,104	184,901
Uncovered	256,326	34,896	221,430	95,836	576,475
Total No. of disease-age cells (obs.)	99,146	18,236	80,910	81,654	105,543

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.8: 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.9: 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.10: Deaths Covered by Age Group

	All		Never Covered		Covered in Expansion:							
					2004		2005		2006		2007	
Age 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.11: Robustness of GES Impact on Deaths to Alternative Models

	Non-linear		Linear	
	Poisson	Neg-Bin	Log	IHS
	(1)	(2)	(3)	(4)
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

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 represents 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 one 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.12: 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
% 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

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 each characteristic's linear growth between 2001 and 2003 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) presents the p-values associated with the column (5) coefficients.

Table A.13: GES Impact on Deaths by GES Expansion and Among Ever Covered

	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)
No. Deaths	264,974	313,514	327,169	336,076	313,519
No. Deaths ∈ covered diseases (year before coverage)	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.14: 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)
No. Deaths	96,966	424,334	86,324	434,976	134,481	386,819	106,780	414,520
No. Deaths ∈ covered diseases (year before coverage)	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 more amenable and less 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.15: Heterogeneous Impact on Deaths

	Sex		Age Group			Inpatients by Type of Hospital		
	Female	Male	0-49	50-79	80+	All	Public	Private
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
After GES Expansion	-0.052***	-0.038***	-0.082***	-0.047**	-0.022	-0.069***	-0.073***	-0.023
	(0.017)	(0.014)	(0.022)	(0.018)	(0.029)	(0.020)	(0.021)	(0.029)
No. Deaths	226,327	294,973	89,850	252,845	178,605	172,940	155,097	17,843
No. Deaths ∈ covered diseases (year before coverage)	16,819	21,310	5,611	19,015	13,503	10,773	9,683	1,090
Total No. disease-age cells (obs.)	77,145	80,558	42,145	36,415	20,586	81,654	78,139	30,850

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, i.e., $\hat{\theta}_{ATT\%} = exp(\hat{\beta}) - 1$. The Poisson estimation drops disease-age cells with zero outcomes in the study period. Standard errors for $\hat{\theta}_{ATT\%}$ 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.16: GES Impact on In-Hospital Deaths by Type of Health Care Provider Removing the 2004 (Pilot) Expansion

	All	Type of Hospital	
	inpatients	Public	Private
	(1)	(2)	(3)
After GES Expansion	-0.074***	-0.079***	-0.031
	(0.023)	(0.024)	(0.034)
No. Deaths	161,269	145,224	16,045
No. Deaths ∈ covered diseases (year before coverage)	6,078	5,541	537
Total No. disease-age cells (obs.)	78,343	75,042	29,291

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 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. 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 A.17: GES Impact on Deaths by Major Geographic Areas

	North	Center	R.M.	Center-South	South	Austral
	(1)	(2)	(3)	(4)	(5)	(6)
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)
No. Deaths	34,038	80,661	192,498	132,338	73,371	8,394
No. Deaths ∈ covered diseases (year before coverage)	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

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. 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 A.18: GES Impact on In-Hospital Deaths by Hospital Usage Pre-Reform

	All	Surgery	By Hospital Usage	
	Hospitals	Hospitals	High	Low
	(1)	(2)	(3)	(4)
After GES Expansion	-0.069***	-0.063***	-0.030	-0.106***
	(0.020)	(0.019)	(0.021)	(0.022)
No. Deaths	172,940	150,373	85,794	64,579
No. Deaths ∈ covered diseases (year before coverage)	10,773	9,387	5,360	4,027
Total No. disease-age cells (obs.)	81,654	78,676	65,295	55,496

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. Column (1) are all deaths in health care facilities. Column (2) considers only hospitals with at least one surgery before the intervention. Columns (3) and (4) stratifies hospitals with at least one surgery between high and low average surgery rates before the intervention. High usage are hospitals with more than 50% of average surgeries rate before the intervention. Standard errors are clustered at the level of treatment: disease-age cell. *After GES Expansion* and its interaction correspond to percent changes by subtracting 1 from the rate ratio, i.e., $\hat{\theta}_{ATT\%} = exp(\hat{\beta}) - 1$. The Poisson estimation drops disease-age cells with zero outcomes in the study period. Standard errors for $\hat{\theta}_{ATT\%}$ 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.19: GES Impact on Deaths Using Alternative Time Trend Specifications

	Main (All Deaths)						
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
After GES Expansion	-0.044***	-0.045***	-0.038***	-0.037***	-0.046***	-0.039***	-0.041***
	(0.014)	(0.013)	(0.010)	(0.010)	(0.013)	(0.011)	(0.011)
No. Deaths	521,300	521,300	521,300	521,300	521,300	521,300	513,381
No. Deaths ∈ covered diseases (year before coverage)	38,129	38,129	38,129	38,129	38,129	38,129	38,129
Total No. disease-age cells (obs.)	99,146	99,146	99,146	99,146	99,146	97,908	80,888
Year FE	Y	Y	Y	Y	Y	Y	Y
Cell (Diseases-Age Group) FE	Y	Y	Y	Y	Y	Y	Y
Age-Group x Year FE	N	Y	N	N	N	N	N
Diseases x Year FE	N	N	Y	N	N	N	N
Cell x Year FE	N	N	N	Y	N	N	N
Age-Group x Year x Post FE	N	N	N	N	Y	N	N
Diseases x Year x Post FE	N	N	N	N	N	Y	N
Cell x Year x Post FE	N	N	N	N	N	N	Y

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, i.e., $\hat{\theta}_{ATT\%} = exp(\hat{\beta}) - 1$. We include additional fixed effects to control for time trends specific to the 22 age groups, diseases, and cells (disease-age), respectively. We also added a set of specifications that included an indicator that allowed for a different trend before and after intervention. The Poisson estimation drops disease-age cells with zero outcomes in the study period. Standard errors for $\hat{\theta}_{ATT\%}$ 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.

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: Period Life expectancy at birth was 77.33 years in 2003, the pre-reform year for which official data is reported in detail. 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. 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 0.39 years in terms of life expectancy as of 2003.[50] Such a decline would have taken people 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 workers' extra wages 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, in U.S. dollars of 2022, there are numbers ranging from $0,50 to $6,33 million depending on the method and purpose (Mardones and Riquelme, 2018; Parada-Contzen, 2019). Using Chile's halfway point estimates -$USD 3,419,185-, which represents two thirds of the GDP per capita in 2004, we can say that the 1,678 lives saved thanks to the GES reform (in one year) would be valued at USD $4,182,772,414, approximately 8% of the GDP in 2003.[51]

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 USD $1,224,506,697 in additional revenues after one year of its implementation. Therefore, we can say that the cost of the reform was approximately a third of the benefits that were brought because of the lives saved.

[50] We compared our results using Table 1, column (1), and Table A.15 columns (3-5) age-specific coefficients to compute the total and age-specific relative decrease in deaths, finding a 0.01 difference between them.

[51] All values in U.S. dollars of 2022. The exchange rate used to convert from Chilean pesos to U.S. currency corresponds to the market-observed dollar rate exchange published by the Chilean Central Bank.

Appendix C: 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 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.[52] Figure C.1 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 expansions on healthcare 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:

$$Y_{it} = \alpha + \rho_t + \beta \text{GES}_{it} + \gamma X_{it} + \varepsilon_{it}, \tag{6}$$

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

[52] 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.

level.

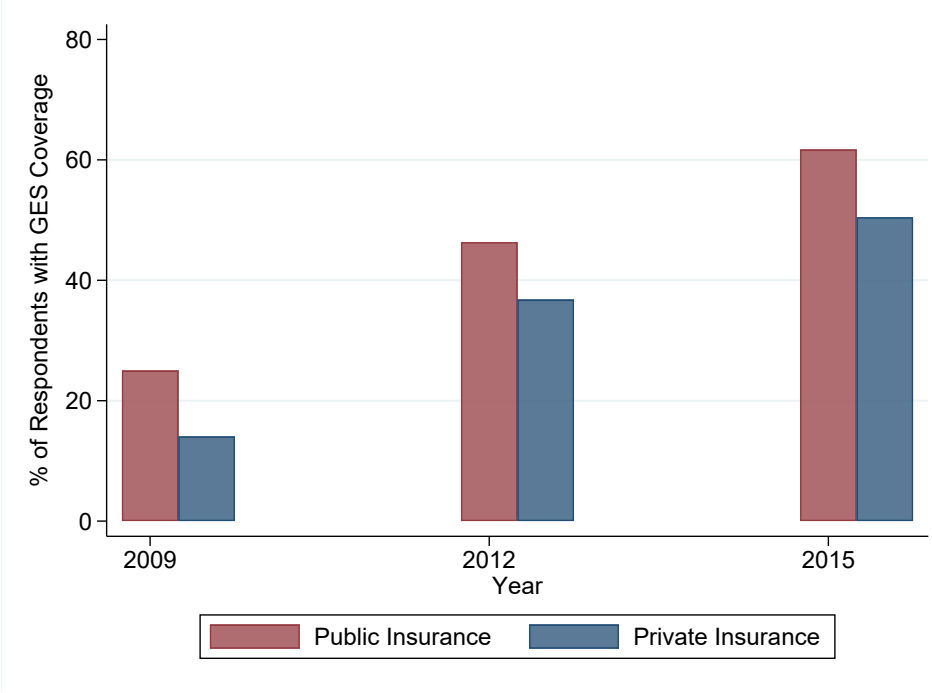
The parameter of interest is β, which, under a (strong) conditional independence assumption, can be interpreted as the effect of GES coverage on the outcome of interest. Taking advantage of the panel structure of our data, we also estimate a more stringent specification, including person-fixed effects. 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 C.1 presents our estimates of equation (6). 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 C.1, 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 healthcare. All in all, these results suggest that the reform led to more medical care and less out-of-pocket spending among its beneficiaries.

Figure C.1: 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 C.1: 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.

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