Labor Market Deregulation and Deaths of Despair

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Abstract

Employment protection legislation (EPL) deregulation has expanded labor market flexibility across advanced economies, with underexplored consequences for population health. This paper asks whether such deregulation contributes to deaths of despair (drug overdoses, alcohol-related disease, and suicide), a mortality pattern first documented in the United States. Using OECD employment protection indicators and WHO mortality data for 26 economies from 1998–2019, we apply local projection method to trace the dynamic mortality response to major EPL reforms. Drug use disorder and suicide mortality rise significantly within five years of deregulation, with no corresponding rise in alcohol-related mortality. This response is systematically moderated by institutional context: health-system effectiveness consistently attenuates it; income support protects against suicide and violence but, unexpectedly, amplifies the alcohol-mortality response; and poverty dampens the drug-mortality response, consistent with an affordability channel in which binding budget constraints limit the substance use that economic insecurity would otherwise drive. These findings indicate that labor market deregulation carries public-health costs, and that social safety nets and health-system capacity are important complements to flexibility-enhancing reforms.

Keywords: Deaths of Despair; Employment Protection Legislation; Labor Market Deregulation; Mortality; Social Safety Nets; OECD Economies
JEL Classification Numbers: I14; J65; J08; I18

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1 Introduction

Over the past three decades, advanced economies have experienced profound changes in the organization of work and the distribution of economic gains. A salient trend is the steady weakening of workers' bargaining power and job security, often attributed to deregulation of employment protection legislation (EPL), declining union density, and the rise of non-standard work. In parallel, researchers and policy-makers have grown increasingly concerned about "deaths of despair": mortality from drug overdoses, alcohol-related diseases, and suicide, which have contributed to stalled or reversed improvements in life expectancy in several high-income countries. These twin developments raise an urgent question: do labor-market institutions that shape job security and income stability have measurable consequences for despair-related mortality?

This paper examines that question in a cross-national setting by studying the dynamic health effects of major EPL deregulation. We assemble annual data for 26 OECD economies from 1998 to 2019 and focus on age-standardized death rates for drug use disorders, alcohol use disorders, and suicide. We also examine cirrhosis of the liver, violence, and inflammatory heart disease mortality, plausibly linked to the same substance-use and psychosocial-stress pathways that drive our primary outcomes. To trace the timing and persistence of effects, while avoiding strong parametric assumptions, we use local projections (Jordà, 2005) to estimate impulse-response functions (IRFs) following major reforms to EPL that relax dismissal protections for workers on regular contracts and alter procedures for collective dismissals. This approach allows us to characterize how deaths of despair evolve in the years immediately after reforms and to differentiate medium-run trajectories across causes of death. We identify major reforms from large discrete movements in the OECD's EPL indicator and embed them in a panel framework with country and year fixed effects, forward-looking reform controls, and lags of outcomes and treatments to mitigate reverse causality, a design well suited to policy shocks that occur at different times across countries. We then extend the baseline specification with interaction terms that capture how country characteristics (unemployment-benefit replacement rates, social-transfer effort, public health-insurance coverage, avoidable mortality, and poverty) amplify or dampen the mortality response.

Our analysis yields three core findings. First, major EPL deregulation is followed by a statistically and economically significant rise in drug-related mortality, with effects that build over several years; we also find a positive and significant increase in suicide mortality within five years of reform. Second, the average effect on alcohol-related mortality is not statistically different from zero, pointing to heterogeneous causal pathways across the components of deaths of despair. Third, we detect systematic institutional moderation of these effects, though not uniformly across every institution and cause of death, and not always in the direction a simple buffering account would predict. Countries with more effective health systems (proxied by lower avoidable mortality and, for drug mortality specifically, higher public health insurance coverage) exhibit significantly attenuated increases in drug and inflammatory heart disease mortality. Income-support institutions protect against suicide and violence mortality, but are associated with significantly *larger*, not smaller, increases in alcohol-related mortality, a reversal we can only partly explain; we also find that the mortality consequences of deregulation and tightening are not simple mirror images of one another, with a formal test rejecting exact symmetry at both the shortest and longest horizons we examine. We discuss these patterns, along with plausible explanations for the departures from a simple institutional-buffering story, in Section 6. We further document increases in violence and inflammatory heart disease mortality several years after deregulation, patterns plausibly linked to the direct and indirect harms of substance use as well as to prolonged socioeconomic stress.

These results contribute to several literatures. In labor economics, a large body of work evaluates the efficiency and distributional effects of job-protection reforms (Barbieri & Cutuli, 2016; Ciminelli et al., 2022), yet far fewer studies follow their downstream health consequences. By linking major EPL changes

to cause-specific mortality dynamics, we provide new evidence that labor-market flexibility carries measurable population-health trade-offs. In population health and social epidemiology, the paper builds on the deaths-of-despair literature developed primarily around U.S. within-country evidence (Case & Deaton, 2022; Case & Deaton, 2015, 2020; Gaydosh et al., 2019; Monnat, 2020; Ruhm, 2022; Ruhm, 2018; Stein et al., 2017) and complements individual-level studies connecting insecurity, job loss, and precarious work to mental health, substance use, and suicide (Glei et al., 2024; Hawkins et al., 2021; Jolivet & Postel-Vinay, 2025; Scheiring et al., 2020), showing that macro-institutional shifts can move despair-related mortality at the country level. Finally, by disaggregating deaths of despair and emphasizing timing, magnitude, and moderators, we speak to debates about whether rising mortality reflects supply-side shocks (e.g., synthetic opioids) or deeper structural distress (Case & Deaton, 2022; Ruhm, 2022; Ruhm, 2018): our results are most consistent with a view in which institutional erosion increases exposure and vulnerability, particularly where social protections are thin, even as drug supply conditions shape which harms are most salient.

The findings have direct policy relevance. If reforms that loosen dismissal protections increase deaths of despair, especially drug and suicide mortality, then labor-market institutions should be considered part of the public-health architecture. The moderation we observe in countries with stronger income supports and health systems suggests that policymakers contemplating flexibility-enhancing reforms should pair them with robust social insurance and health-system capacity, including accessible mental-health and addiction services. More broadly, the evidence cautions against evaluations of labor-market reform that focus narrowly on employment or wage aggregates while overlooking externalities borne by health systems, families, and communities.

The remainder of the paper proceeds as follows. Section 2 reviews the related literature, states our contribution relative to it, and develops the conceptual mechanisms linking deregulation to mortality through economic, psychosocial, and institutional channels. Section 3 describes the data sources, construction of outcomes, and identification of major reforms. Section 4 details the empirical strategy based on local projections and the heterogeneous-effects design. Section 5 reports the main impulse-response and moderator estimates, along with supplementary analyses of candidate mediating channels and the asymmetry between deregulation and tightening. Section 6 discusses mechanisms, alternative explanations, institutional buffering, policy implications, and limitations. Section 7 concludes.

2 Related Literature and Conceptual Motivation

This section reviews the three literatures that motivate our analysis, states our contribution relative to each, and develops the conceptual mechanisms linking employment protection (EPL) deregulation to deaths of despair that guide our empirical design.

2.1 Literature and Contribution

A first literature evaluates the labor-market consequences of employment protection legislation (EPL) deregulation, a central feature of labor market reform in advanced economies since the 1980s. Ciminelli et al. (2022) show, using a panel of 26 advanced economies from 1970–2013, that EPL deregulation significantly reduces the labor share of income, accounting for roughly 15% of its average decline. Barbieri and Cutuli (2016) find that partial, "marginal" EPL deregulation in Europe which relaxes protections for temporary contracts while leaving permanent employment largely untouched widens labor market dualism and produces at best short-lived employment gains, with temporary jobs rarely serving as stepping stones to stable employment. This erosion of job protection is one part of a broader decline in labor power across advanced economies, evident also in the sharp fall in union density and its contribution

to rising wage inequality (Peters, 2008; Western & Rosenfeld, 2011). This literature establishes that EPL deregulation carries real efficiency and distributional costs, but it has focused almost exclusively on labor-market outcomes; its downstream consequences for population health remain largely unexamined.

A second literature documents the rise of "deaths of despair" which is mortality from drug overdose, alcohol-related disease, and suicide and traces it to economic and social decline. Case and Deaton (2020) and Case and Deaton (2022) link the U.S. rise in these deaths, concentrated among adults without a bachelor's degree (U.S. Census Bureau, 2025), to stagnant wages, weakening attachment to stable employment, and eroding social institutions. Subsequent work extends this account along several dimensions: structural life-cycle evidence on the joint dynamics of job loss and mental health (Jolivet & Postel-Vinay, 2025); deindustrialization as a slow-acting driver of psychosocial stress and eroded social identity (Scheiring et al., 2020); occupation-specific exposure to injury, insecurity, and non-standard work (Hawkins et al., 2021); the interaction of economic precarity with opioid availability and community composition (Bjorklund, 2023); the compounding effects of income inequality and low social mobility (Kuo & Kawachi, 2023); causal evidence linking housing instability to substance-related mortality (Bradford & Bradford, 2020); direct evidence that labor-market policies such as minimum wages and the EITC can reduce specific despair-related deaths (Dow et al., 2020); causal and correlational evidence linking housing wealth and housing policy to despair-related mortality (Jou et al., 2023; Venkataramani & Tsai, 2020); financial losses as a mediating pathway between education and despair-associated risk behaviors (Fishman & Gutin, 2021); occupation- and employment-status-specific mortality risk together with life-course evidence connecting labor-market conditions to mental health (Gutin & Hummer, 2020; Hammarström et al., 2024); structural drivers of widening life-expectancy inequality (Geronimus et al., 2019); and a broader scoping review tying these deaths to widening social and economic inequality (Beseran et al., 2022). Reassessing this evidence, Glei et al. (2024) find that structural detachment from stable work and social ties predicts deaths of despair more robustly than subjective distress per se, reinforcing the view that these deaths reflect social and economic disconnection rather than purely psychological despair. This literature is overwhelmingly correlational and drawn from single-country, typically U.S., settings; it has not exploited a plausibly exogenous, cross-national policy shock to identify the labor-market channel directly.

A third literature debates whether the rise in these deaths mainly reflects changes in the drug-supply environment or demand rooted in economic distress. Ruhm (2018) argues that changes in the drug environment including the substitution from prescription opioids to heroin and, later, illicit fentanyl account for most of the rise in drug mortality, finding that economic decline explains only a small share. Ruhm (2022) shows that the post-1999 rise in midlife mortality is predominantly drug-overdose driven, with distinct age- and cause-specific patterns for suicide and alcohol-related disease, cautioning against treating deaths of despair as a single, homogeneous phenomenon. Reconciling these views, Case and Deaton (2022) argues that supply and demand interact rather than compete: the opioid epidemic would not have reached its scale absent underlying economic distress, even as the timing and severity of the crisis reflect drug-supply dynamics. This debate motivates two choices in our empirical design: we model each cause of death separately rather than pooling them, and we test whether institutional context systematically moderates the mortality response.

This paper's contribution is to bring these three literatures together within a single, plausibly exogenous research design. Relative to the labor economics literature, we extend the study of EPL deregulation to a health-related margin rarely part of the standard cost-benefit calculus. Relative to the deaths-of-despair literature, we move beyond single-country, correlational evidence by tracing the dynamic mortality response to a discrete, cross-national institutional shock across 26 economies over more than two decades. Relative to the supply-side/demand-side debate, we provide new evidence on how the mortality response varies with health-system effectiveness, income-support generosity, and poverty, offering a way

to assess whether labor-market institutions shape exposure to this risk independent of local drug-supply conditions. Our design also parallels a small set of studies that use quasi-experimental economic shocks to trace mortality effects, such as trade liberalization (Pierce & Schott, 2020) and automation (Mishra et al., 2024), but applies this approach to an institutional rather than a technological or trade-driven shock, across a cross-national panel of advanced economies.

2.2 Conceptual Motivation

Given this literature and the gap it leaves open, what should we expect when a country undertakes major EPL deregulation? We organize our expectations around three mechanisms linking deregulation to deaths of despair.

1. Economic Insecurity and Material Strain Deregulation increases job instability, unemployment risk, and income volatility. These forms of economic insecurity are established drivers of adverse health outcomes (Benach et al., 2014; Knapp et al., 2019; Link & Phelan, 1995; Wilkinson & Marmot, 2003), operating through financial strain, reduced future prospects, and heightened uncertainty, and increase the likelihood of risky or self-destructive behaviors.

2. Labor Market Disconnection and Psychosocial Stress Weaker job protection increases precarious employment and detachment from stable work, eroding social integration and undermining identity and purpose (Jolivet & Postel-Vinay, 2025; Putnam, 2000; Scheiring et al., 2020). These processes generate chronic stress and raise the likelihood of harmful coping behaviors such as substance use.

3. Institutional Buffering The mortality consequences of deregulation depend on the institutional context in which it occurs. Health-system capacity, income-support generosity, and exposure to harmful environments shape how economic and psychosocial shocks translate into mortality (Bjorklund, 2023); we refer to this moderating role as institutional buffering and focus on it directly in the empirical design below.

3 Data

This section describes the data sources, variable construction, and sample used in the empirical analysis. We combine cross-country information on employment protection legislation, mortality outcomes, and institutional characteristics for a panel of OECD economies over 1998–2019.

3.1 OECD Indicators of Employment Protection

We measure employment protection using the OECD Employment Protection Indicators, which capture the strictness of regulations governing dismissals and the use of temporary contracts on a scale from 0 (least strict) to 6 (most strict). Consistent with the conceptual framework, we focus on the dimension of job security, specifically regulations on individual and collective dismissals.

The individual dismissals component of the OECD indicators reflects procedural requirements such as advance notice periods, financial costs such as severance pay obligations, and legal enforceability such as employees' access to legal recourse. The collective dismissals component reflects thresholds for triggering collective dismissal rules, consultation obligations, notification requirements to public authorities, and additional procedural delays.

To ensure consistency with mortality data classified under tenth revision of the International Classification of Diseases (ICD-10), we use version 2 of the OECD indicators, which provides annual data for

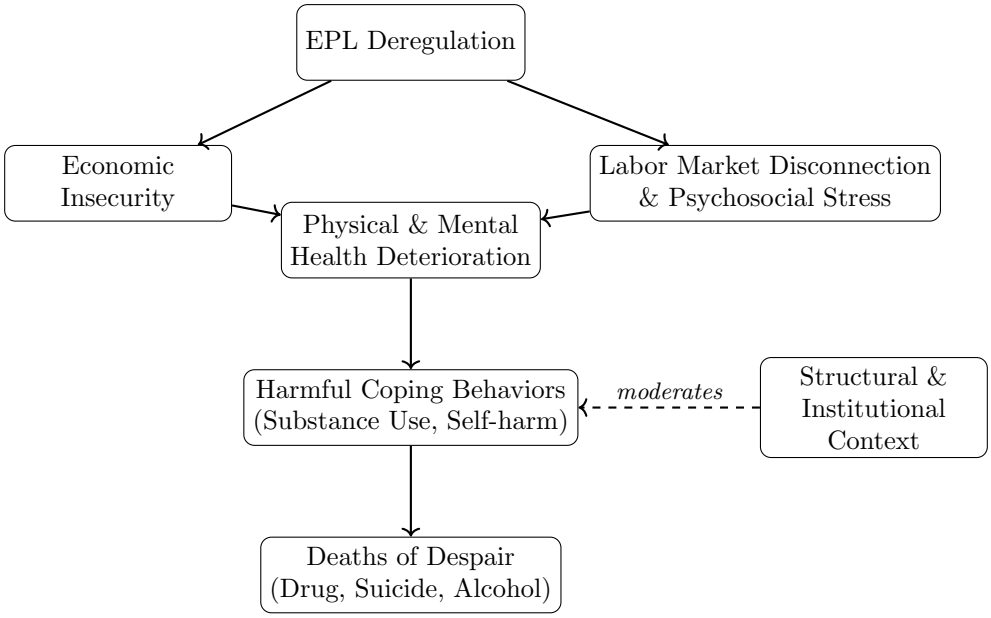


Figure 1: **Conceptual pathways linking employment protection deregulation to deaths of despair.** Solid arrows denote the hypothesized causal channels through which deregulation raises despair-related mortality; the dashed arrow denotes the moderating role of structural and institutional context, which shapes how the health consequences of economic insecurity and psychosocial stress translate into fatal outcomes, rather than itself constituting a direct causal channel.

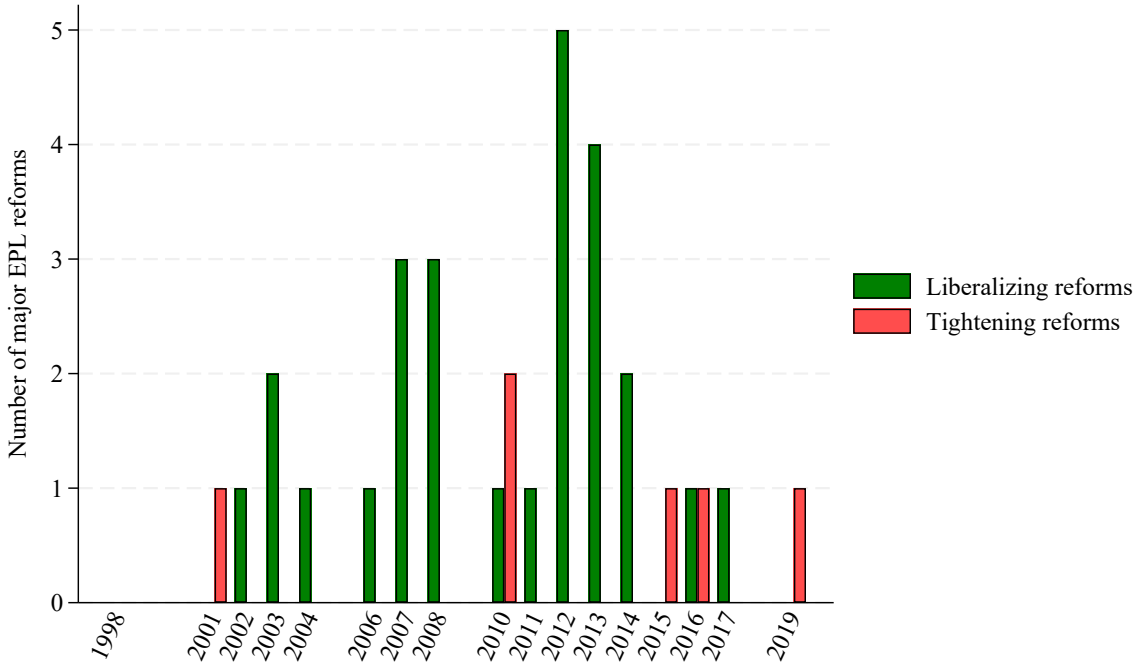
the period 1998–2019. Our analysis exploits large discrete changes in these indicators to identify policy reforms. Following Ciminelli et al. (2022), major EPL reforms are defined as changes in the indicator that fall within the top or bottom 5th percentile of the distribution of annual changes. We construct an indicator variable that takes the value 1 for major deregulation reforms, -1 for major tightening reforms, and 0 otherwise. This approach isolates substantial policy shifts that are plausibly exogenous to short-term fluctuations in mortality.

Figure 2 illustrates the timing and distribution of major EPL reforms across the sample period and countries. The top panel shows the aggregate number of liberalizing and tightening reforms by year. Reforms are unevenly distributed over time, with clear clustering in the mid-2000s and early 2010s. In particular, liberalizing reforms are most frequent between 2006 and 2013, with a pronounced peak around 2012, while tightening reforms are relatively infrequent and occur sporadically throughout the sample period. This pattern indicates that EPL changes occur in discrete reform episodes rather than as gradual adjustments.

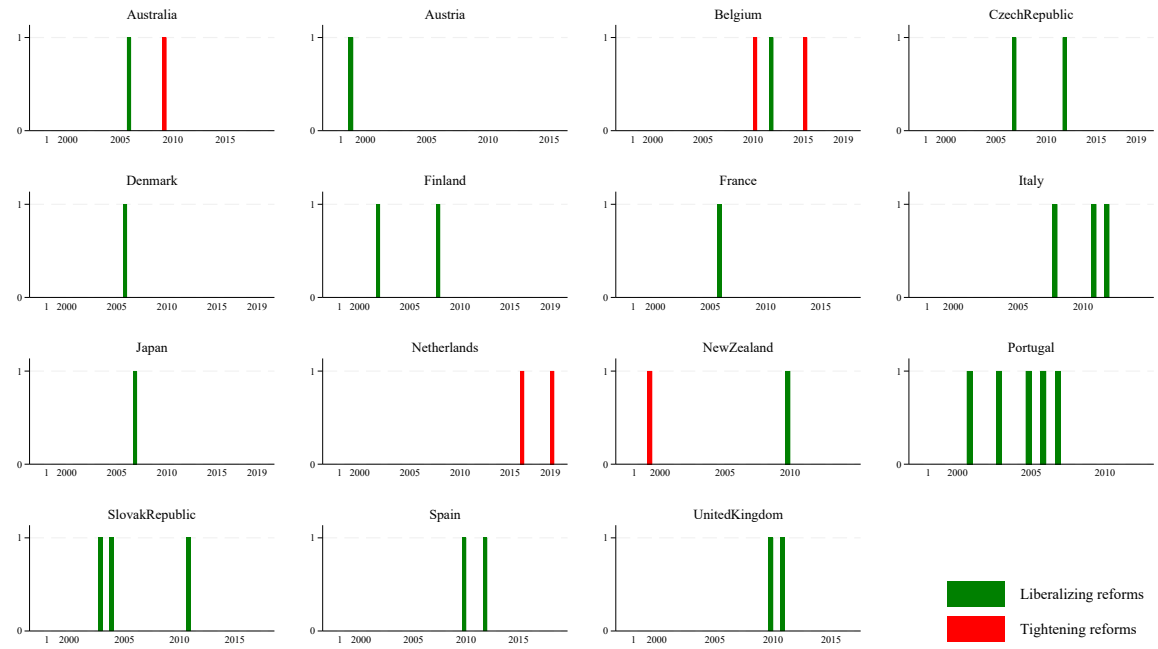
The bottom panel displays the distribution of reforms by country, revealing substantial cross-country heterogeneity. Several countries, such as Portugal, Italy, and Spain, experience multiple liberalizing reforms concentrated within relatively short periods, whereas others exhibit few or no major reforms over the sample. Tightening reforms are less common and limited to a smaller set of countries, including Australia, Belgium, the Netherlands, and New Zealand. The variation in the timing and intensity of reforms across countries provides the key source of identifying variation for estimating the dynamic effects of EPL deregulation.

3.2 WHO Mortality Database

Mortality data are drawn from the World Health Organization (WHO) Mortality Database, providing age-standardized death rates per 100,000 population. Age standardization ensures comparability across countries with different demographic structures.



(a) Distribution of reforms over time



Countries with no major EPL reforms during 1998–2019: Canada, Germany, Greece, Iceland, Ireland, Korea, Luxembourg, Norway, Sweden, Switzerland, UnitedStates.

(b) Reforms by country

Figure 2: **Major employment protection reforms.** The top panel shows the aggregate distribution of reforms over time, while the bottom panel presents the corresponding country-level patterns.

We focus on mortality causes associated with deaths of despair, using ICD-10 classifications. The main outcomes are deaths due to drug use disorders, alcohol use disorders, and self-inflicted injuries (suicide); we also examine violence and inflammatory heart diseases, which are plausibly linked to substance use and prolonged stress, and cirrhosis of the liver as a supplementary alcohol-related outcome.[1]

To ensure consistency and avoid measurement discontinuities, we restrict the sample to ICD-10 data. Many countries experienced substantial breaks in mortality series during the transition from ICD-9 to ICD-10, making pre-transition data less comparable. Since most OECD countries adopted ICD-10 around 1998, this restriction yields a balanced and reliable panel covering 26 countries over 1998–2019. Figure 11 in appendix shows the number of countries and areas with data in the WHO Mortality Database by ICD code and year, as of April 2022.

3.3 OECD Country-specific Characteristics

To examine heterogeneous effects, we incorporate country-specific characteristics capturing key institutional and structural conditions. These variables enter the empirical specification as interaction terms with the EPL reform indicator and are normalized to represent differences between the 25th and 75th percentiles.

We consider three broad categories of moderators. First, income support is measured using unemployment benefit replacement rates and government social transfer expenditures, which proxy the extent of economic insurance. Second, health system capacity is captured by avoidable mortality rates and public health insurance coverage, reflecting the effectiveness and accessibility of healthcare systems. Third, broader social conditions are proxied by poverty rates, which capture structural disadvantage.

Avoidable mortality can represent the effectiveness of public health and healthcare system, which includes both preventable and treatable deaths. Preventable deaths are defined as causes of death among people aged under 75 years that can be avoided through effective public health and prevention interventions (i.e. before the onset of disease/injury, to reduce incidence). Treatable deaths are defined as causes of death that can be avoided through timely and effective healthcare interventions, including secondary prevention and treatment (i.e. after the onset of disease, to reduce case fatality) (OECD, 2023).

These measures allow us to assess whether stronger social protection and health institutions mitigate the impact of EPL deregulation on deaths of despair, consistent with the institutional buffering mechanism outlined in Section 2.2.

4 Empirical Methodology

This section describes the empirical strategy used to estimate the dynamic effects of employment protection legislation (EPL) reforms on deaths of despair. To capture the timing and persistence of these effects while avoiding restrictive functional form assumptions, we employ the local projection method introduced by Jordà (2005).

4.1 Baseline Specification

For each horizon $k = 0, \ldots, 5$, we estimate the following specification:

[1] ICD-10 codes: drug use disorders (F11–F16, F18–F19, X41–X42, X44); alcohol use disorders (F10, X45); self-inflicted injuries (X60–X84, Y870); violence (X85–Y09, Y871); inflammatory heart diseases (I30–I33, I38, I40, I42); cirrhosis of the liver (K70, K74). Detailed description of these ICD-10 codes are in the appendix.

$$y_{i,t+k} - y_{i,t-1} = \alpha_i + \tau_t + \beta_k R_{i,t} + \theta \mathbf{X}_{i,t} + \sum_{h=1}^{k} \phi_h R_{i,t+h} + \sum_{l=1}^{4} (\delta_l \Delta y_{i,t-l} + \gamma_l R_{i,t-l}) + \varepsilon_{i,t}. \quad (1)$$

The dependent variable, $y_{i,t+k}$, denotes the natural logarithm of the age-standardized mortality rate for a given cause of death in country i at horizon k; expressing the outcome in logs lets the coefficients β_k be interpreted as semi-elasticities, or approximate percentage changes in mortality. The specification models cumulative changes in log mortality relative to the pre-reform period, allowing the coefficients β_k to be interpreted as impulse responses to EPL reforms.

The variable $R_{i,t}$ is an indicator of major EPL reform, capturing discrete policy changes identified from the OECD Employment Protection Indicators. Country fixed effects (α_i) control for time-invariant heterogeneity across countries, while year fixed effects (τ_t) capture global shocks affecting all countries simultaneously. The vector $\mathbf{X}_{i,t}$ includes standard macroeconomic controls.

To isolate the effect of reforms occurring at time t, the specification includes forward reform terms $\sum_{h=1}^{k} \phi_h R_{i,t+h}$, which account for additional reforms implemented within the response horizon. In addition, four lags of both the outcome variable and the reform indicator are included to mitigate concerns about reverse causality and dynamic adjustment.

The coefficients β_k trace the dynamic response of mortality to a major EPL reform over time. By estimating the model separately for each horizon k, we obtain impulse response functions (IRFs) that describe how mortality evolves in the years following a reform. Standard errors are clustered to account for serial correlation.

The identifying assumption is that, conditional on country and year fixed effects and the lagged dynamics built into equation (1), the timing of major EPL reforms is unrelated to the mortality trajectory that would have prevailed absent the reform. Two distinct threats to this assumption are worth separating. The first is reverse causality running from mortality to policy: reforms could, in principle, be timed in response to a mortality crisis. This is unlikely in practice, since major EPL reforms are the outcome of protracted political bargaining over labor-market flexibility (typically motivated by unemployment, competitiveness, or fiscal considerations) rather than being designed or timed around cause-specific mortality trends; the four lags of the reform indicator and the forward-reform controls in equation (1) further guard against any residual short-run feedback. The second, more subtle threat is confounding rather than reverse causality: even reforms that are not caused by mortality may coincide with broader economic or political shifts, such as fiscal consolidation, structural adjustment programs, financial crises, or changes in government, that independently affect deaths of despair through channels unrelated to job security. Country and year fixed effects absorb time-invariant country characteristics and common global shocks, but cannot by themselves rule out that a country undertaking EPL deregulation is, at the same time, undergoing other changes correlated with the mortality response we attribute to deregulation.

We take two complementary steps to assess this threat empirically rather than assume it away. First, we extend the baseline specification into an event-study design that adds six years of pre-reform leads to the six post-reform horizons, using Teulings–Zubanov-style controls for interim reforms in both directions (Figure 4). If EPL deregulation were confounded with a broader package of other reforms or a country-specific shock that also drives mortality, we would generally expect that shock to leave a footprint on mortality in the years just before or around the EPL reform, or to generate a differential trend already visible pre-reform. Finding instead that pre-reform coefficients are close to zero and show no systematic trend, followed by a post-reform response that rises gradually rather than jumping discontinuously at $h = 0$, is more consistent with a genuine dynamic response to the EPL reform itself than with a pre-existing or contemporaneously confounded trend, though it cannot rule out a confounding shock whose

effects happen to be timed to coincide exactly with the EPL reform.

Second, we implement the local projections difference-in-differences (LP-DiD) estimator of Dube et al. (2025), applied to the subsample of 22 countries that experience at least one liberalizing reform (Figure 7). Unlike the two-way fixed-effects specification in equation (1), LP-DiD estimates each horizon-specific effect using only "clean" comparisons: newly deregulating countries against countries that are not-yet-treated or, under an effect-stabilization assumption, previously treated countries that have re-entered the control pool once their own effects have stabilized. This avoids the negative-weighting bias that can affect two-way fixed-effects estimates under staggered treatment timing. This is a useful check on the confounding concern above because it is a different estimator built on a more restrictive set of country-year comparisons than the baseline specification; a result that failed to survive this alternative construction of the control group would raise doubts about whether the baseline estimate reflects a general feature of deregulation episodes or an artifact of a specific comparison. Figure 7 reports five variants of the estimator that differ in how the control sample is constructed: 4a and 4b restrict controls to not-yet-treated countries, with 4a fixing the estimation sample across all horizons to rule out compositional changes in which countries are compared at each horizon, and 4b allowing that sample to vary horizon by horizon; 5a and 5b instead allow previously treated countries to re-enter the control pool once their effects have stabilized, again with (5a) and without (5b) the fixed-sample restriction; and 5c reweights the 5b sample using a regression-adjustment estimator to recover an equally weighted, rather than variance-weighted, average treatment effect. A consistent pattern across all five variants would indicate that the baseline drug-mortality result is not an artifact of the two-way fixed-effects estimator or of any single way of constructing the control group.

4.2 Heterogeneous Effects

To examine how institutional and structural conditions shape the impact of EPL reforms, we extend the baseline specification to include interaction terms:

$$y_{i,t+k} - y_{i,t-1} = \alpha_i + \tau_t + \beta_{1k}(R_{i,t} \times S_{i,t}) + \beta_{2k} R_{i,t} + \beta_{3k} S_{i,t} + \cdots + \varepsilon_{i,t}. \tag{2}$$

Here, $S_{i,t}$ represents country-specific characteristics, including measures of income support, health system capacity, and social conditions. These variables are normalized to reflect differences between countries at the 25th and 75th percentiles. For social transfer expenditure, public health insurance coverage, and the unemployment benefit replacement rate (each of which is naturally increasing in institutional generosity), $S_{i,t}$ is defined as the inverse of the underlying measure, so that, consistent with the avoidable death rate and the poverty rate, moving from the 25th to the 75th percentile of $S_{i,t}$ corresponds to moving from a more to a less protective institutional environment for every moderator considered.

The interaction coefficient β_{1k} captures how the effect of EPL reforms varies across institutional environments. Specifically, it can be interpreted as the differential impact of a reform between countries at the 25th percentile of $S_{i,t}$ (a more protective environment) and countries at the 75th percentile (a less protective environment); under this convention, a positive β_{1k} indicates that the mortality response to deregulation is larger where institutions are less protective, consistent with an institutional-buffering interpretation.

5 Results

5.1 Descriptive Statistics

Table 1 reports summary statistics for the main outcome, treatment, control and moderator, and candidate mediator variables used in the analysis.

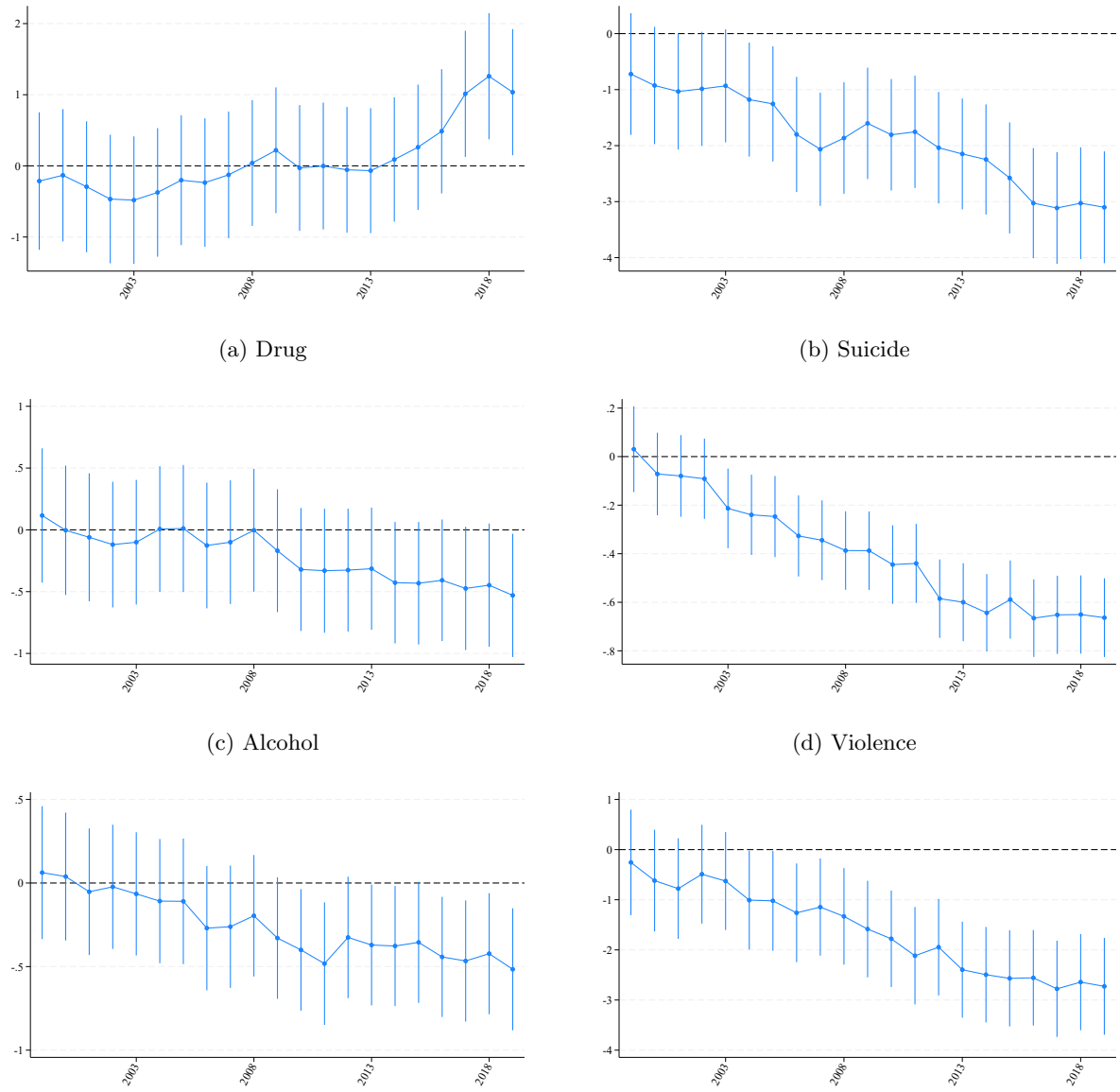
Table 1: Descriptive statistics

Variable	Count	Mean	SD	Min	Max
Outcome variables					
Drug death rate	486	2.561944	2.753695	0.000000	19.09016
Suicide death rate	484	10.895560	4.007673	3.321734	26.31062
Alcohol death rate	486	2.456993	2.144824	0.000000	10.89471
Violence death rate	486	1.140382	1.161581	0.000000	7.333786
Inflammatory heart diseases death rate	488	3.654734	1.323953	1.039139	8.607623
Cirrhosis of liver death rate	488	7.605482	4.283892	1.394737	22.82417
Treatment variable					
EPL reforms	486	0.041152	0.253540	−1	1
Control variables & moderators					
Avoidable death rate	458	220.906100	65.475420	124.000000	515.0000
Replacement rate of UB (60 months)	440	32.854550	16.366160	0.000000	69.0000
Gov. social transfers expenditure (% of GDP)	457	11.893550	3.417891	5.023348	19.67462
Nominal GDP per capita ($)	486	40205.440000	15698.440000	10665.070000	121073.8000
Manufacturing share of GDP	486	15.050760	5.528970	4.554389	34.89132
Unemployment rate	463	7.262532	4.024560	2.013950	26.49027
Poverty rate	267	10.398510	3.362702	4.545772	18.62064
Gov. health insurance coverage rate	462	95.635070	15.335840	22.000000	100.0000
Possible mediators					
Net family earnings, min. wage (% of reference type)	292	55.628770	12.880510	24.640000	80.34000
Disposable income below poverty line (% of population)	257	10.406180	3.354735	4.545772	18.50000
Involuntary part-time employment (% of employment)	458	3.905338	2.318894	0.176000	12.33600
Employment tenure of 1–5 months (%)	393	5.726652	1.808592	2.389000	11.78989

Notes: Mortality rates are per 100,000 population. EPL reforms is the signed reform indicator (−1 for major tightening, 1 for major deregulation, 0 otherwise). The unemployment rate appears both as a control variable and, in horizon-matched cumulative-change form, as a candidate mediator (Section 5.6); it is reported once here. Summary statistics are computed over available observations; counts reflect non-missing country-year observations for each variable, 1998–2019.

Figure 3 summarizes stylized facts on cause-specific mortality across OECD countries. It reports average changes in mortality rates by year, obtained from a specification with country and year fixed effects. Several common patterns emerge. Only drug use disorder mortality exhibits a marked upward trend over the sample period, with increases especially pronounced after the mid-2000s. In contrast, alcohol use disorder and inflammatory heart disease mortality displays a gradual decline over time, while suicide, violence mortality and cirrhosis of liver mortality show statistically significant and persistent downward trends. These aggregate patterns highlight substantial heterogeneity across causes of death, motivating their separate analysis in the empirical framework.

Appendix Figure 12 shows that these aggregate patterns mask substantial cross-country heterogeneity in the magnitude and direction of mortality trends for each cause of death, underscoring the value of exploiting both cross-country and time-series variation to identify the effects of EPL reforms.



(a) Drug

(b) Suicide

(c) Alcohol

(d) Violence

(e) Inflammatory Heart Disease

(f) Cirrhosis of Liver

Figure 3: **Average changes in cause-specific mortality rates across 26 OECD countries.** Each panel reports the coefficients on the year fixed effects from the regression $Mortality_{i,t} = \alpha + \tau_t + \gamma_i + \varepsilon_{i,t}$, where i and t index country and year, respectively. $Mortality$ denotes the cause-specific mortality rates, α is a constant, τ_t are year fixed effects, and γ_i are country fixed effects. Vertical lines indicate 95% confidence intervals. The estimates can be interpreted as the average change in mortality rates relative to 1998, the omitted base year.

5.2 Main Effects

Figures 4, 5, and 6 report the dynamic effects of major EPL deregulation on cause-specific mortality, estimated using the local-projection event-study design of equation (1) extended with six years of pre-reform leads (Section 4.1). Each figure plots both the pre-reform leads and the post-reform response, so the pre-trend evidence and the treatment effect can be read directly off the same plot. Two patterns emerge and structure the rest of this section.

First, as Figure 4 shows, EPL deregulation is associated with a statistically and economically significant increase in mortality from drug use disorders. Pre-reform coefficients are flat and close to zero, with no systematic upward or downward trend, while the post-reform response is not immediate: it builds gradually over the post-reform window and becomes both larger and more precisely estimated at longer horizons. By the five-year horizon, drug-related mortality is approximately 35 percent higher than its pre-reform level, indicating a substantial medium-run effect rather than a transient shock. This combination, a flat pre-trend followed by a gradually building post-reform response, is inconsistent with reverse causality or pre-existing divergence between reforming and non-reforming countries, and instead supports reading the post-reform estimates as a consequence of EPL reforms rather than an artifact of confounding.

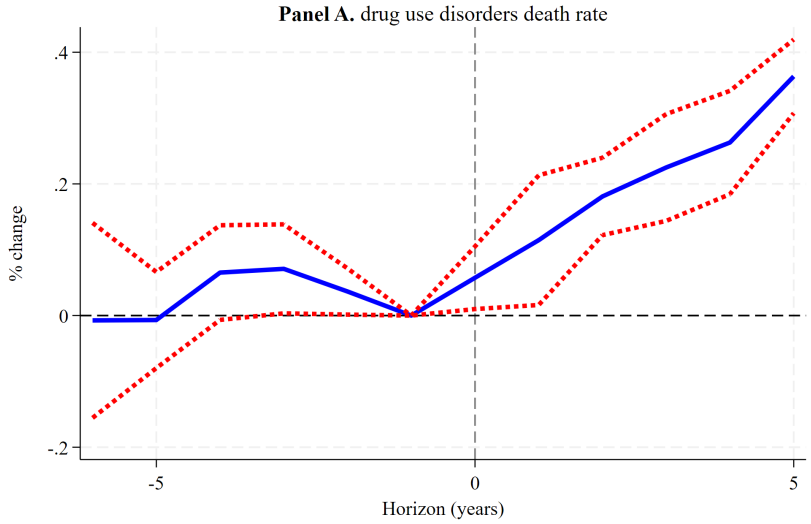


Figure 4: **Dynamic effects of major EPL deregulation on drug use disorder mortality with pre-trend.** The figure plots local-projection event-study estimates of the log mortality rate (per 100,000 population) response to major EPL deregulation for drug use disorder, including six years of pre-reform leads. The horizontal axis reports years relative to reform (horizons $h = -6, \ldots, 0, \ldots, 5$); the reference period is $h = -1$. The solid line shows point estimates; dotted lines show 95% confidence interval bounds. Standard errors are Driscoll–Kraay. Sample: 26 OECD countries, *[1998-2019]*.

Second, suicide mortality also rises following deregulation, with similarly flat pre-reform coefficients (Figure 5), though the magnitude is an order of magnitude smaller, reaching roughly 5 percent at the five-year horizon.

Mortality from alcohol use disorder, by contrast, shows no statistically significant response at any horizon, before or after reform (Figure 6).

This pattern points to meaningful heterogeneity across components of deaths of despair: labor market deregulation appears to affect causes of death linked to acute substance use and self-harm more than alcohol-related disease specifically. We treat the alcohol null result as substantively informative rather than as a nuisance to be explained away, and return to it in Section 5.4 together with a supplementary

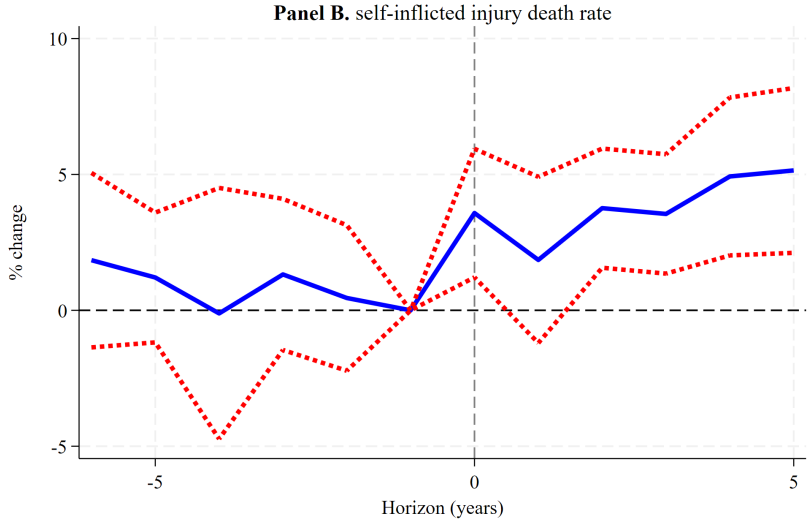


Figure 5: **Dynamic effects of major EPL deregulation on self-inflicted injury (suicide) mortality with pre-trend.** The figure plots local-projection event-study estimates of the log mortality rate (per 100,000 population) response to major EPL deregulation for self-inflicted injury (suicide), including six years of pre-reform leads. The horizontal axis reports years relative to reform (horizons $h = -6, \ldots, 0, \ldots, 5$); the reference period is $h = -1$. The solid line shows point estimates; dotted lines show 95% confidence interval bounds. Standard errors are Driscoll–Kraay. Sample: 26 OECD countries, *[1998-2019]*.

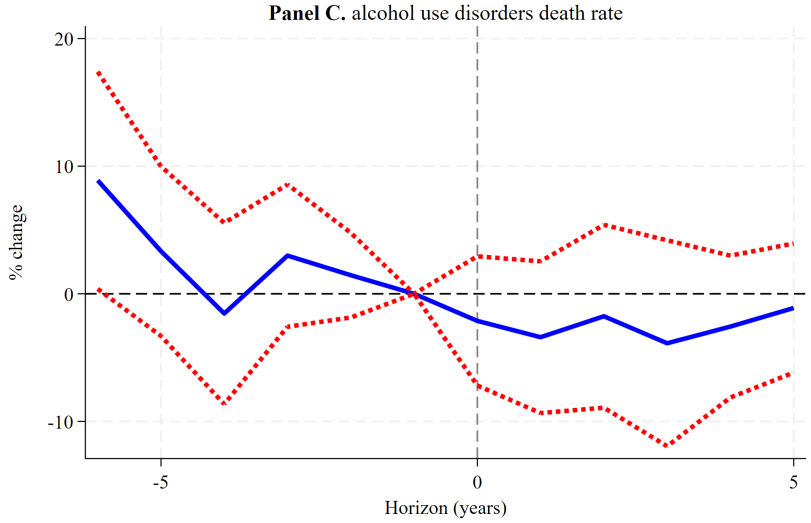


Figure 6: **Dynamic effects of major EPL deregulation on alcohol use disorder mortality with pre-trend.** The figure plots local-projection event-study estimates of the log mortality rate (per 100,000 population) response to major EPL deregulation for alcohol use disorder, including six years of pre-reform leads. The horizontal axis reports years relative to reform (horizons $h = -6, \ldots, 0, \ldots, 5$); the reference period is $h = -1$. The solid line shows point estimates; dotted lines show 95% confidence interval bounds. Standard errors are Driscoll–Kraay. Sample: 26 OECD countries, *[1998-2019]*.

analysis of alcohol-attributable cirrhosis mortality.

5.3 Robustness Checks

Figure 7 reports robustness checks based on the local projection difference-in-differences (LP-DiD) approach of Dube et al. (2025), described in Section 4.1, estimated using only countries experiencing liberalizing reforms. In brief, LP-DiD re-estimates each horizon-specific effect using only "clean" comparisons: newly deregulating countries against countries that are not-yet-treated or that have re-entered the control pool once their own effects have stabilized, rather than the full two-way fixed-effects sample used in the baseline specification. This addresses potential concerns related to treatment heterogeneity, staggered adoption, and contamination from tightening reforms, and in particular avoids the negative-weighting bias that can affect two-way fixed-effects estimates when treatment timing is staggered.

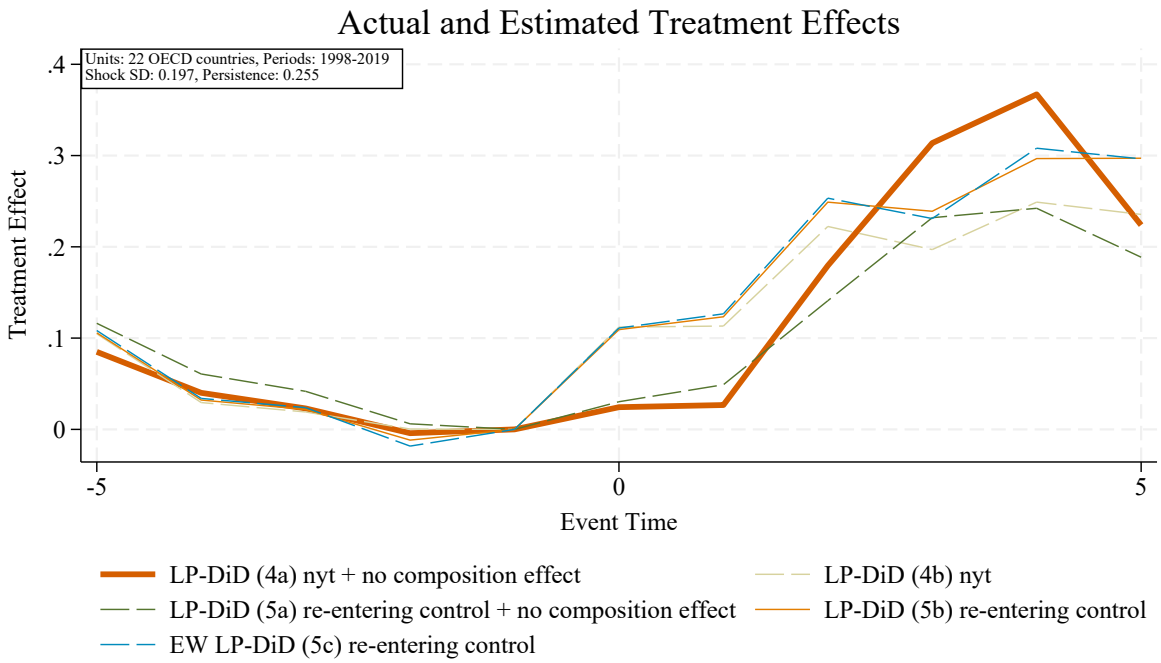


Figure 7: **Robustness check of the effects of major EPL deregulation on drug use disorder mortality with LP-DiD method.** This figure shows LP-DiD estimates of both pre-trend and post-reform response of the log mortality rate (per 100,000 population) for drug use disorder. The horizontal axis reports years before and after reform (horizons $h = -5, \ldots, 0, \ldots, 5$). Solid lines show point estimates; shaded bands are 95% confidence intervals. Standard errors are Driscoll–Kraay. Sample: 22 OECD countries, *[1998-2019]*.

Across all specifications, the results exhibit a consistent dynamic pattern. Pre-reform coefficients remain close to zero, providing additional support for the absence of differential pre-trends. Following reform, the estimated treatment effects increase steadily, reaching magnitudes comparable to those obtained in the baseline specification.

Importantly, the results are robust across alternative implementations of the LP-DiD estimator, including specifications that account for re-entering control groups and alternative weighting schemes. Although some variation in magnitude arises at longer horizons, the qualitative pattern of increasing post-reform effects remains stable. These findings indicate that the baseline results are not driven by specification choice or sample composition.

As a further check, Table 2 re-estimates the baseline drug mortality specification adding, one at a time,

controls for the global financial crisis, lagged unemployment, the unemployment benefit replacement rate, trade union density, the manufacturing share of GDP, and social transfer expenditure. The coefficient of interest is essentially unchanged by any single addition: point estimates at each horizon move by at most a few hundredths of a log point relative to the baseline row, and the pattern of increasing statistical significance from $h = 0$ through $h = 5$ is preserved throughout. This stability indicates that the baseline dynamic response is not an artifact of omitting any one of these commonly used macroeconomic or institutional controls.

Table 2: Dynamic effects of major EPL deregulation on mortality from drug use disorders

Controls for	**0y**	**1y**	**2y**	**3y**	**4y**	**5y**
Baseline	0.06**	0.11**	0.18***	0.22***	0.26***	0.36***
	(0.0224)	(0.0462)	(0.0274)	(0.0375)	(0.0360)	(0.0254)
Crisis dummy	0.05**	0.11**	0.19***	0.22***	0.25***	0.37***
	(0.0237)	(0.0412)	(0.0276)	(0.0330)	(0.0455)	(0.0283)
Lagged unemployment	0.05**	0.12**	0.19***	0.24***	0.27***	0.37***
	(0.0209)	(0.0480)	(0.0257)	(0.0394)	(0.0383)	(0.0312)
Replacement rate of UB	0.06**	0.11**	0.18***	0.22***	0.26***	0.36***
	(0.0223)	(0.0462)	(0.0272)	(0.0370)	(0.0357)	(0.0254)
Union density	0.05***	0.11**	0.18***	0.23***	0.24***	0.34***
	(0.0164)	(0.0474)	(0.0271)	(0.0350)	(0.0545)	(0.0431)
Manufacturing share of GDP	0.06**	0.11**	0.17***	0.20***	0.23***	0.30***
	(0.0217)	(0.0470)	(0.0251)	(0.0369)	(0.0321)	(0.0313)
Social transfers expenditure	0.06**	0.11**	0.18***	0.22***	0.27***	0.38***
	(0.0218)	(0.0443)	(0.0261)	(0.0343)	(0.0422)	(0.0445)

Notes: Outcome is the log mortality rate from drug use disorders (per 100,000 population). Entries report dynamic semi-elasticities at horizons $h = 0$ to 5 years after major EPL deregulation; standard errors in parentheses. Stars denote significance: $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$.

5.4 Effects on Other Causes of Death

We next examine effects on other causes of death, together with a supplementary analysis of alcohol-attributable cirrhosis of the liver that speaks directly to the null result for alcohol use disorder mortality reported in Section 5.2. Figures 8 (Panel D), 9 (Panel E), and 10 (Panel F) present local-projection event-study estimates, including six years of pre-reform leads, for three additional outcomes: cirrhosis of the liver, violence, and inflammatory heart disease.

Figure 8 shows that cirrhosis mortality is statistically indistinguishable from zero at nearly every horizon, both before and after reform. A single point estimate (at $h = 4$, approximately 3 percent) reaches conventional significance, but it is not part of a systematic pattern: it does not build steadily across horizons the way the drug and suicide effects do (Table 2), and it fades by $h = 5$. We read this as a basically null result for cirrhosis, consistent with the null result for alcohol use disorder mortality documented in Section 5.2, rather than as evidence that broadening the outcome to include alcohol-attributable liver disease (ICD-10 K70, K74, which our primary alcohol codes F10 and X45 omit) uncovers an effect that the narrower codes miss.

We view the joint null result for alcohol use disorder and cirrhosis mortality as genuinely informative rather than as an absence of findings, and as a result that disciplines the interpretation of our other estimates. Unlike drug and suicide mortality, which rise steadily and significantly through year 5, neither outcome shows a cumulative, persistent response to deregulation. This pattern indicates that the mortality response to EPL deregulation is not a generic "economic stress raises mortality" story, but is instead concentrated in, and most precisely estimated for, causes of death with comparatively rapid behavioral

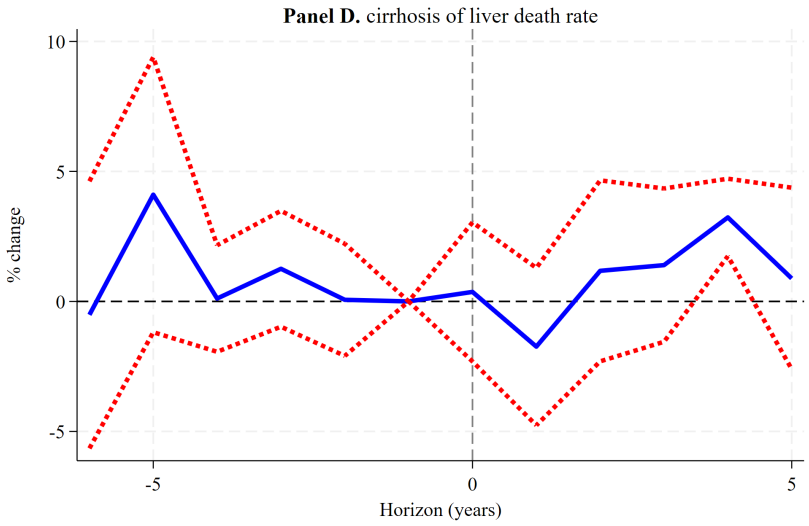


Figure 8: **Dynamic effects of major EPL deregulation on cirrhosis of the liver mortality with pre-trend.** The figure plots local-projection event-study estimates of the log mortality rate (per 100,000 population) response to major EPL deregulation for cirrhosis of the liver, including six years of pre-reform leads. The horizontal axis reports years relative to reform (horizons $h = -6, \ldots, 0, \ldots, 5$); the reference period is $h = -1$. The solid line shows point estimates; dotted lines show 95% confidence interval bounds. Standard errors are Driscoll–Kraay. Sample: 26 OECD countries, *[1998-2019]*.

and physiological onset (drug overdose and suicide) relative to the slower-accumulating alcohol-related pathway where 10 to 20 years are typically required for alcohol-related liver disease to develop. Five-year post-reform window may simply be too short to detect an effect even if one exists.

Turning to violence and inflammatory heart disease, Figures 9 and 10 show that EPL deregulation is associated with increases in mortality from both causes that become statistically significant only at the four- and five-year horizons, mirroring the delayed pattern documented for drug mortality. The timing and nature of this finding are consistent with indirect pathways linking economic insecurity to health outcomes. Increases in substance use and prolonged stress may contribute to both heightened exposure to violent environments and elevated cardiovascular risk.

5.5 Heterogeneous Effects by Institutional Context

The average responses reported in Sections 5.2 and 5.4 pool countries with markedly different social-insurance and health-system architectures. To test whether the mortality response to deregulation depends on this institutional context, we extend the baseline specification with an interaction between the reform indicator and each of five country characteristics: health-system effectiveness (the avoidable death rate), social transfer expenditure, public health insurance coverage, the unemployment benefit replacement rate, and the poverty rate (equation (2), Section 4.2). Each moderator is normalized so that the interaction coefficient β_{1k} is interpretable as the differential mortality response between a country at the 75th percentile of the moderator and one at the 25th percentile. Tables 3, 6, 7, 8, and 9 report the resulting coefficients at each horizon for drug use disorders, suicide, alcohol use disorder, violence, and inflammatory heart disease mortality, respectively.

For drug mortality, the cause of death with the largest and most precisely estimated average effect, institutional context matters along several dimensions (Table 3). The avoidable death rate interaction is positive and significant from the two-year horizon onward, rising from 0.34 to 0.50 log points ($p < 0.05$); the public health insurance coverage interaction is positive and significant at every horizon, growing from

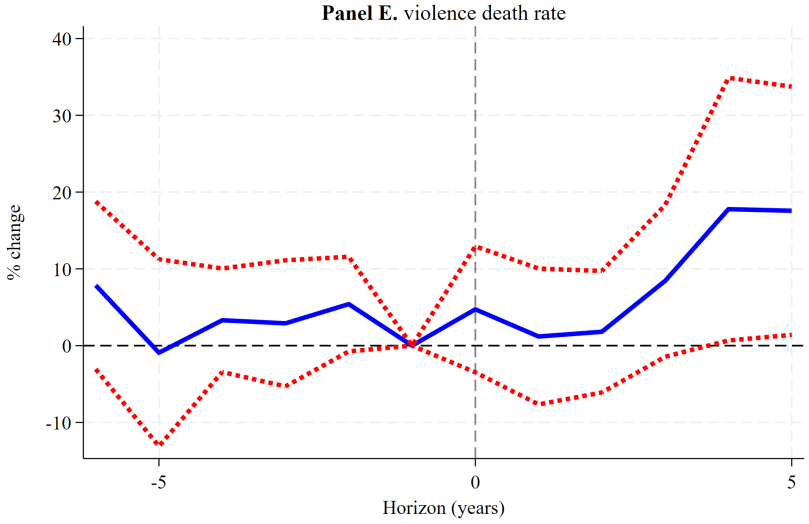


Figure 9: **Dynamic effects of major EPL deregulation on violence mortality with pre-trend.** The figure plots local-projection event-study estimates of the log mortality rate (per 100,000 population) response to major EPL deregulation for violence, including six years of pre-reform leads. The horizontal axis reports years relative to reform (horizons $h = -6, \ldots, 0, \ldots, 5$); the reference period is $h = -1$. The solid line shows point estimates; dotted lines show 95% confidence interval bounds. Standard errors are Driscoll–Kraay. Sample: 26 OECD countries, *[1998-2019]*.

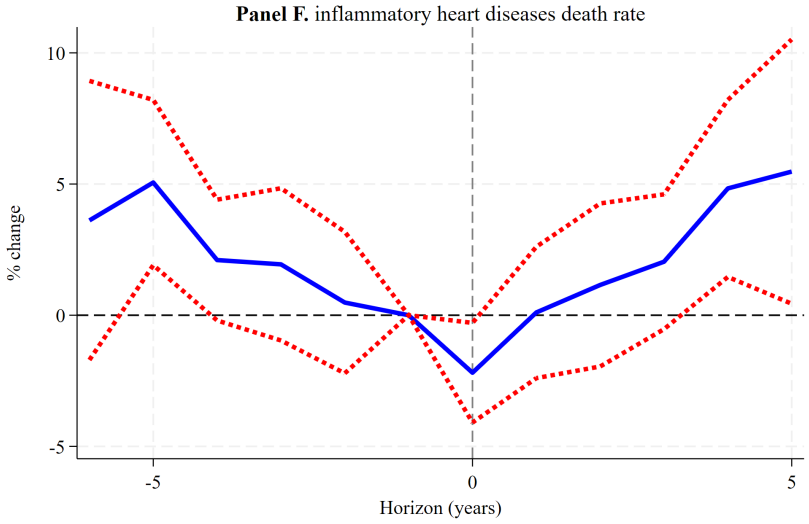


Figure 10: **Dynamic effects of major EPL deregulation on inflammatory heart disease mortality with pre-trend.** The figure plots local-projection event-study estimates of the log mortality rate (per 100,000 population) response to major EPL deregulation for inflammatory heart disease, including six years of pre-reform leads. The horizontal axis reports years relative to reform (horizons $h = -6, \ldots, 0, \ldots, 5$); the reference period is $h = -1$. The solid line shows point estimates; dotted lines show 95% confidence interval bounds. Standard errors are Driscoll–Kraay. Sample: 26 OECD countries, *[1998-2019]*.

0.01 to 0.17 log points; and the poverty rate interaction is negative and significant from impact through the four-year horizon, reaching −0.62 log points at $h = 3$ before losing significance at $h = 5$. Social transfer expenditure and the unemployment benefit replacement rate do not interact significantly with deregulation at any horizon for this outcome.

For the remaining four outcomes, full results are reported in Appendix Tables 6–9; here we summarize only the broad patterns, both because the individual coefficients are discussed further in Section 6.3 and because, with five outcomes and five moderators evaluated across six horizons, any single moderator-outcome-horizon combination should be read with caution given the scope for multiple comparisons. Suicide interactions are estimated far less precisely than those for drug mortality, consistent with the smaller average effect documented in Section 5.2, with social transfer expenditure the only moderator significant at more than one horizon. Alcohol use disorder mortality shows no significant *average* response to deregulation (Section 5.2), yet several institutional interactions are nonetheless significant, a pattern suggesting that offsetting responses across more and less generous welfare states may mask an average effect for this cause of death. Violence and inflammatory heart disease mortality, the two additional outcomes discussed in Section 5.4, display institutional interactions concentrated at the horizons where their average effects also emerge.

Two patterns cut across all five outcomes and are the ones we carry forward to the interpretation in Section 6.3. First, the avoidable death rate is the most consistently significant moderator, entering with a positive sign wherever it is significant, indicating that a less effective health system is associated with a larger mortality response. Second, the poverty rate is negative and significant for four of the five outcomes, making it the single most pervasive moderator in the analysis, though its sign runs counter to a naive prediction that higher poverty would amplify, rather than dampen, the mortality response.

Table 3: Dynamic effects of EPL deregulation on mortality from drug use disorders: interaction terms

Interaction term with	**0y**	**1y**	**2y**	**3y**	**4y**	**5y**
Avoidable death rate	0.14**	0.22	0.34**	0.50**	0.40**	0.40**
	(0.0544)	(0.1279)	(0.1372)	(0.1699)	(0.1785)	(0.1731)
Social transfers expenditure	0.05	0.14	0.05	0.17	0.35	0.49
	(0.0843)	(0.1615)	(0.2287)	(0.2829)	(0.3316)	(0.3240)
Public health insurance coverage	0.01***	0.01*	0.04**	0.08**	0.11*	0.17*
	(0.0036)	(0.0082)	(0.0156)	(0.0324)	(0.0573)	(0.0836)
Replacement rate of UB (60 months)	0.09	0.22	0.02	0.05	-0.06	0.06
	(0.0957)	(0.1887)	(0.2097)	(0.2237)	(0.2350)	(0.2046)
Poverty rate	-0.19*	-0.47**	-0.54***	-0.62**	-0.51**	-0.19
	(0.0947)	(0.1934)	(0.1593)	(0.2051)	(0.1812)	(0.1384)

Notes: Outcome is the **log mortality rate from drug use disorders** (per 100,000 population). Entries are interaction effects (P75–P25 style) at horizons $h = 0$ to 5 years after a major EPL deregulation; standard errors in parentheses. Significance: *$p < 0.10$, **$p < 0.05$, ***$p < 0.01$.

5.6 Candidate Channels

The results above are consistent with slow-accumulating economic insecurity and labor market disconnection channels operating through the mechanisms outlined in Section 2.2, but this interpretation so far rests on the shape of the aggregate mortality response rather than on direct evidence that either channel is operative. We probe the mechanism more directly by testing whether a set of candidate mediating variables (proxies for material strain and for labor market disconnection) respond to major EPL deregulation with a timing pattern comparable to the one estimated for drug mortality. For each candidate M, we estimate

$$M_{i,t+k} - M_{i,t-1} = \alpha_i + \tau_t + \beta_k R_{i,t} + \theta \mathbf{X}_{i,t} + \sum_{h=1}^{k} \phi_h R_{i,t+h} + \sum_{l=1}^{4} (\delta_l \Delta M_{i,t-l} + \gamma_l R_{i,t-l}) + \varepsilon_{i,t}$$

for $k = 0, \ldots, 5$, using the identical horizon-matched cumulative-change transformation, lag structure, and forward-reform correction used in equation (1) for mortality, applied instead to the candidate mediator, together with four lags of the mediator's own first difference. This puts the question "does M respond to deregulation, and on what timeline" on the same footing as the mortality regression itself, rather than testing mediator relevance in levels, which we found in preliminary work to give a misleading picture of first-stage relevance because it does not distinguish a genuine dynamic response to reform from a persistent, confounded association between a country's mediator level and its propensity to reform. Table 4 reports results for five candidates spanning both proposed mechanisms: net family income for a low-wage family and the poverty rate (material strain); the short-tenure employment share (labor market disconnection); and, as an informative counterpoint, two conventional labor-market-slack measures, the aggregate unemployment rate and the involuntary part-time employment share.

Two candidates move with a timing and sign consistent with the conceptual framework. The poverty rate rises following deregulation, with the response building over time and reaching statistical significance only at the four- and five-year horizons (0.82 and 1.43 percentage points, $p < 0.05$ and $p < 0.01$, Table 4), the same horizons at which the drug mortality response in Table 2 is largest and most precisely estimated. The short-tenure employment share (the fraction of employees with one to five months of job tenure) rises significantly from the two-year horizon onward (0.36 to 0.45 percentage points, $p < 0.10$ or better), consistent with deregulation increasing job churn and reducing the sense of stable labor market attachment that the psychosocial-disconnection channel in Section 2.2 emphasizes. Net family income for a minimum-wage couple with two children moves in the theoretically expected (negative) direction from the second horizon onward and is directionally consistent throughout, though the response is noisier and only marginally significant at $h = 2$ and $h = 5$ ($p = 0.066$ and $p = 0.088$), likely reflecting the smaller sample of countries for which this measure is available.

The remaining two candidates move in the direction opposite to a naive prediction, which we view as informative rather than simply as a null result. Both the aggregate unemployment rate and the involuntary part-time employment share *decline*, not rise, following deregulation, and increasingly so: the unemployment rate falls by up to 1.16 percentage points by the five-year horizon ($p < 0.05$), and involuntary part-time employment falls by up to 0.91 percentage points ($p < 0.05$). This is not, on reflection, surprising: a large literature on employment protection notes that lower dismissal costs raise both separations and hires, so the net effect on the aggregate unemployment rate is theoretically ambiguous even where effects on job security and turnover are not (Ciminelli et al., 2022). Taken together with the short-tenure result above, the pattern across all five candidates argues against a crude story in which deregulation raises drug mortality simply by raising joblessness or involuntary underemployment; if anything, conventional labor-market-slack measures improve after deregulation. Instead, the evidence points toward rising material strain (poverty) and rising job instability (short-tenure churn), independent of the aggregate quantity of employment, as the more plausible channels.

We report these results as suggestive screening evidence, not as a formal causal mediation decomposition. Confirming that a candidate variable responds to deregulation with plausible timing is a necessary but far from sufficient condition for it to be a genuine mediator of the mortality effect: a formal decomposition into natural direct and indirect effects would additionally require sequential ignorability of the mediator conditional on treatment and observed confounders, an assumption we regard as implausible in a 26-country annual panel where mediators and mortality outcomes plausibly share unobserved

country-year confounders not captured by our controls. We view this exercise as motivating, rather than substituting for, future mediation analysis using individual- or household-level panel data with richer information on employment transitions, income, and health-seeking behavior.

Table 4: Timing of candidate mediating variables following major EPL deregulation

Candidate mediator	**0y**	**1y**	**2y**	**3y**	**4y**	**5y**
Net family income (minimum-wage couple, 2 kids)	-0.27	-1.41	-2.49*	-3.11	-4.20	-5.27*
	(0.369)	(0.898)	(1.248)	(2.416)	(2.881)	(2.837)
Poverty rate	0.51	0.45	0.62	0.45	0.82**	1.43***
	(0.503)	(0.317)	(0.560)	(0.490)	(0.358)	(0.340)
Short-tenure employment share (1–5 mo.)	-0.01	0.15	0.36**	0.40**	0.45*	0.44*
	(0.087)	(0.148)	(0.158)	(0.168)	(0.227)	(0.228)
Unemployment rate	-0.01	-0.22	-0.50*	-0.66**	-1.01*	-1.16**
	(0.169)	(0.259)	(0.270)	(0.303)	(0.508)	(0.480)
Involuntary part-time employment	-0.01	-0.15	-0.11	-0.48	-0.74*	-0.91**
	(0.123)	(0.165)	(0.278)	(0.286)	(0.378)	(0.356)

Notes: Each row reports $\hat{\beta}_k$ from a separate regression of the candidate mediator's own horizon-matched cumulative change on the EPL deregulation indicator, following the specification described in the text (identical lag structure and forward-reform correction to equation (1), applied to the mediator rather than to mortality). Standard errors, clustered by country, in parentheses. Significance: $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$.

5.7 Asymmetry Between Deregulation and Tightening

The baseline specification codes major EPL reforms with a single signed indicator (+1 for deregulation, −1 for tightening; Section 3.1), which imposes by construction that tightening reverses the mortality consequences of deregulation exactly: a single coefficient β_k at each horizon applies with the opposite sign to both reform types. This symmetry restriction need not hold. If job protections operate partly through channels with hysteresis, for instance if workers who lose employment during a period of weaker protection do not simply regain it once protections are restored, or if the substance-use and mental-health consequences of a deregulation episode persist independently of subsequent policy reversals, then tightening would be expected to have a smaller, or even a same-signed, effect rather than a mirror-image one.

We test this directly by decomposing the signed indicator into two non-negative dummies, one for major deregulation episodes and one for major tightening episodes, and entering both jointly, with their own lags and horizon-specific forward-term corrections, in place of the single signed indicator. Table 10 and Figure 13 (Appendix) report the results for drug use disorder mortality. The deregulation coefficients closely track the baseline estimates in Table 2 and are significant at every horizon. The tightening coefficients are negative from year 2 onward, consistent with tightening partially reversing the mortality consequences of deregulation, but they are estimated far less precisely, unsurprising given that tightening episodes are considerably rarer in our sample and concentrated in a handful of countries (Section 3.1), and are statistically distinguishable from zero only at the three- and five-year horizons.

We find evidence against exact symmetry, but it points in two different directions at different horizons rather than toward a single, uniform pattern. At impact ($h = 0$), the tightening coefficient (0.077) is positive rather than negative (the same sign as the deregulation coefficient, 0.092), and a formal test of H_0: $\beta_{dereg} + \beta_{tighten} = 0$ rejects exact symmetry at the 5 percent level ($p = 0.020$). Taken at face value, this is consistent with a short-run one-way ratchet: tightening does not immediately lower drug mortality the way deregulation immediately raises it. At the five-year horizon we again reject symmetry ($p = 0.016$), but in the opposite direction: the tightening coefficient (−0.955) is more than twice the magnitude of the deregulation coefficient (0.396), so tightening is associated with a substantially larger

mortality decline than deregulation is with a mortality increase, rather than a proportionate reversal. At the intervening horizons ($h = 1$ through $h = 4$) point estimates are of the theoretically expected negative sign but imprecisely estimated, and we cannot reject symmetry.

We interpret these results cautiously rather than as establishing a specific form of asymmetry. The tightening coefficients are identified from a much smaller number of reform episodes than the deregulation coefficients: tightening reforms in our sample are limited to a handful of countries, including Australia, Belgium, the Netherlands, and New Zealand (Section 3.1). The wide confidence intervals at most horizons likely reflect low power as much as a genuinely weak or absent tightening effect. What we can say with more confidence is that the data reject the specific symmetry restriction embedded in the baseline specification at two horizons, in two substantively different ways (a short-run ratchet and a long-run overshoot), which is itself informative: it suggests that a single symmetric coefficient understates the complexity of how EPL reforms of either sign propagate into mortality. Characterizing this asymmetry more precisely than our data allow is a natural direction for future research, ideally with a larger or more disaggregated set of tightening episodes drawn from a longer sample period or a broader set of countries.

6 Discussion

6.1 Interpretation of Mechanisms

The results speak directly to the three mechanisms outlined in the conceptual framework (Section 2.2): economic insecurity and material strain, labor market disconnection and psychosocial stress, and interaction with structural and institutional context. The dynamic pattern documented in Table 2 is itself informative about which channels are likely operative: the baseline semi-elasticity of drug mortality rises monotonically from 0.06 log points at impact to 0.36 log points at the five-year horizon, a gradual, cumulative pattern that is difficult to reconcile with a purely mechanical or short-lived shock and instead points to a slow accumulation of economic and psychological strain. This timing is consistent with a broader pattern in the related literature: Jolivet and Postel-Vinay (2025) document that the mental-health consequences of job loss unfold gradually over the life cycle rather than resolving quickly, Scheiring et al. (2020) show that deindustrialization operates as a slow-acting, cumulative driver of population health, and Glei et al. (2024) find that structural detachment from the labor force predicts deaths of despair more robustly than subjective distress alone.

The fact that suicide responds more modestly (roughly 5 percent at five years) than drug mortality (roughly 35 percent) is also informative. Both causes plausibly share the same underlying economic-insecurity channel, but drug mortality is also sensitive to the local drug-supply environment (the availability, potency, and price of illicit substances), which can amplify the health consequences of a given increase in psychosocial distress; suicide has no comparable supply-side amplifier. The same shared pathway is visible in the timing of the results for these additional causes of death: violence and inflammatory heart disease mortality rise only at the four- and five-year horizons (Section 5.4), mirroring the delayed drug mortality response.

The clinical and epidemiological literature offers a direct account of why sustained substance use would raise mortality from inflammatory heart disease and violence with a lag rather than immediately. Chronic heavy alcohol consumption is strongly associated with toxic myocarditis (Piano & Phillips, 2023); stimulants are linked to myocarditis through catecholamine surge and direct toxicity (Virmani et al., 2001); and opioid and intravenous drug use are leading causes of infective endocarditis in young adults (Schmidt et al., 2022). Because these cardiac conditions take time to develop into fatal events, this pathway is consistent with the delayed emergence of the inflammatory heart disease response. Alcohol and certain drugs also directly elevate the risk of homicide and assault through impaired judgment,

aggression, and involvement in illicit drug markets (Darke, 2010; Foran & O'Leary, 2008; Kuhns et al., 2014; World Health Organization, 2018), supporting a reading of the delayed violence and inflammatory heart disease responses as downstream consequences of the same rise in substance use that drives the primary drug mortality result.

This interpretation rests on the shape of the aggregate mortality response itself, rather than on direct evidence that a specific channel is operative. Section 5.6 probes the mechanism more directly by testing whether candidate mediating variables respond to deregulation with a timing pattern comparable to the mortality response. That evidence points toward rising material strain (the poverty rate) and rising job instability (the short-tenure employment share) as the more plausible channels, and specifically away from the aggregate unemployment rate, which if anything *falls* following deregulation, arguing against a crude story in which deregulation raises drug mortality simply by raising joblessness. We treat this as suggestive screening evidence, motivating rather than substituting for future mediation analysis with individual- or household-level data.

6.2 Alternative Explanations

A natural alternative explanation for the observed rise in drug mortality is a supply-side account, in which changes in the illicit drug environment (the substitution from prescription opioids to heroin and, after roughly 2013, to illicitly manufactured fentanyl), rather than labor market conditions, drive the increase (Ruhm, 2022; Ruhm, 2018). Several features of our results speak to this possibility, though none is fully dispositive on its own. First, the identification strategy exploits the staggered timing of major EPL reforms across 26 countries (Figure 2), whereas the opioid-to-fentanyl transition is heavily concentrated in North America with limited penetration in Southern and Eastern European labor markets; because our specification estimates the mortality response relative to each country's own pre-reform path, a common supply shock affecting all countries simultaneously cannot mechanically generate the estimated dynamic response. Second, the event-study evidence in Figure 4 shows flat, statistically insignificant pre-reform coefficients, which would not obtain if the drug environment happened to deteriorate systematically just before reforms. Third, the age-specific results in Table 11 show effects concentrated among prime-age and older workers (ages 20–69), with no significant increase among the youngest cohort (15–19), a profile more consistent with a channel operating through employment and its attendant economic security than with a supply-driven fentanyl narrative that has disproportionately affected younger users in North America.

That said, we cannot fully rule out that reforms are correlated with unobserved, time-varying local drug-market conditions, particularly where major deregulation episodes and the fentanyl-driven overdose wave coincided in the 2010s. We view the evidence as most consistent with Case and Deaton (2022)'s synthesis, in which demand-side distress and supply-side drug availability interact rather than operate as competing explanations: weakening labor market institutions plausibly increase the population at risk of problematic substance use, while the potency and availability of the local drug supply shape how that latent vulnerability translates into fatal outcomes, an interpretation reinforced by the institutional heterogeneity discussed next, which a purely supply-driven account would not predict.

6.3 Institutional Buffering

Building on the patterns reported in Section 5.5, we ask whether institutional context moderates the mortality response to EPL deregulation in a way that admits a coherent causal interpretation. The evidence supports an institutional-buffering interpretation, but one more nuanced than a single moderator operating uniformly across causes of death. Health-system effectiveness is the clearest and most consistent buffer: countries with weaker health-system capacity, less able to prevent, treat, or otherwise mitigate the consequences of substance use and cardiovascular stress before they become fatal, experience significantly

larger mortality responses to deregulation, for both drug and inflammatory heart disease mortality. Income-support generosity buffers the two outcomes most plausibly tied to acute psychological crisis, suicide and violence, as a simple protective story would predict, but for alcohol use disorder mortality it runs the opposite way, with more generous transfers and unemployment benefits associated with a *larger* response, a reversal we flag as a genuine puzzle rather than a resolved finding. One plausible reading is that, unlike suicide and violence, alcohol consumption may be constrained less by psychological distress than by purchasing power: income support need not reduce the propensity to drink under economic stress so much as it preserves the disposable income that job loss and income instability would otherwise curtail, sustaining consumption that a binding budget constraint would suppress in less generous welfare states. Under this affordability channel, weaker income support would dampen the alcohol-mortality response not through psychological buffering but simply by limiting the resources available to act on it, a reading loosely consistent with the negative, if only marginally significant, poverty-rate interaction for alcohol mortality reported below. We regard this as a more parsimonious explanation than an unobserved policy confound, though we cannot test it directly without individual- or household-level spending data, and note it as a direction for future work.

The poverty rate interaction, by contrast, is negative for four of the five outcomes, the opposite of a naive buffering prediction, though the pattern's strength varies considerably across them. It is most pronounced for drug mortality, negative and significant at five of six horizons (Table 3); added-variable and leave-one-country-out checks confirm this specific coefficient is not driven by any single country (Appendix Figures 14 and 15). We read this as consistent with the same affordability channel discussed above for income support and alcohol, operating here in the opposite direction: poverty constrains the purchasing power needed to act on the psychological and material pressures that deregulation generates, so a rise in economic insecurity translates less readily into drug use, and hence drug mortality, where households already face a binding budget constraint. This logic plausibly extends to violence and inflammatory heart disease mortality, whose poverty interactions are also negative and significant at several horizons (Appendix Tables 8 and 9): to the extent these outcomes are downstream consequences of substance use rather than independent channels (Section 6.1), a binding budget constraint on substance use would be expected to dampen their mortality response too. The alcohol interaction is weaker and less consistent, significant only on impact and changing sign thereafter (Appendix Table 7), but directionally consistent with the same affordability logic. Taken together, we read the heterogeneity results as most consistent with a layered story: health-system capacity as the most robust and theoretically coherent buffer; income-support generosity and the poverty rate as two sides of the same affordability channel, with income support buffering the acute-crisis outcomes (suicide, violence) but amplifying alcohol mortality, and poverty dampening drug, violence, and inflammatory heart disease mortality, and more weakly alcohol mortality, by constraining rather than enabling the resources needed to act on distress. We nonetheless recognize that several of these moderator–outcome combinations remain imprecisely estimated, and that individual- or household-level spending data would be needed to test the affordability channel directly.

6.4 Policy Implications

These findings have direct implications for how labor market reforms are evaluated and designed. Standard cost-benefit assessments of EPL deregulation typically weigh efficiency gains (reduced hiring frictions, improved allocative efficiency, lower structural unemployment) against distributional costs measured in wages or the labor share of income (Barbieri & Cutuli, 2016; Ciminelli et al., 2022). Our results suggest that this accounting is incomplete: a five-year, 35 percent increase in drug mortality and a 5 percent increase in suicide mortality following major deregulation episodes represent a public-health

externality that falls outside conventional labor market metrics but is arguably comparable in welfare terms to the employment gains such reforms are designed to achieve.

The heterogeneity results offer a constructive, rather than purely cautionary, policy lesson. Because the adverse mortality response is significantly attenuated in countries with more effective health systems, and because suicide- and violence-related harms are offset in countries with more generous income support (though alcohol use disorder mortality is an exception to this buffering pattern, Section 6.3), the evidence points toward specific, actionable complements to flexibility-enhancing reforms rather than a blanket argument against labor market deregulation. Countries pursuing EPL reform could pair it with expanded access to addiction treatment and overdose-reversal services, continuity of health coverage for displaced workers, and adequately funded unemployment insurance, particularly given the concentration of estimated effects among prime-age and older workers who are more likely to have dependents and less time to recover from labor market displacement (Table 11). More broadly, the results support treating employment protection legislation, unemployment insurance, and health-system capacity as jointly determined components of a country's social-insurance architecture rather than as independent policy levers, echoing calls in the literature to integrate labor market and public-health policy design (Beseran et al., 2022; Kuo & Kawachi, 2023).

6.5 Limitations

Several limitations qualify these conclusions and suggest directions for future research. First, and most fundamentally, the analysis is conducted at the country-year level; we cannot observe which individuals lose employment protection, whether they experience job loss, or the pathway from labor market events to fatal outcomes. Our estimates therefore capture aggregate population-level responses rather than individual-level treatment effects, and the effect on directly affected workers is likely substantially larger than the aggregate semi-elasticities reported here.

Second, the definition of a "major" reform (changes in the OECD EPL indicator falling in the top or bottom 5th percentile of the annual-change distribution, following Ciminelli et al. (2022)) is a useful but somewhat arbitrary threshold, and it collapses considerable heterogeneity in reform content (e.g., changes to individual versus collective dismissal rules, notice periods versus severance costs) into a single binary indicator. Reforms of similar magnitude on the OECD index may differ substantially in their practical labor market consequences, and our design cannot speak to which specific regulatory margins are most consequential for health.

Third, while the local projection design with country and year fixed effects, lagged outcomes and treatments, and forward reform controls is intended to mitigate reverse causality and confounding from contemporaneous shocks, and while the pre-trend evidence in Figure 4 is reassuring, we cannot rule out that unobserved, time-varying country-specific shocks, such as political transitions, macroeconomic crises, or shifts in the drug-supply environment, coincide with reform timing in ways not fully captured by our controls. The LP-DiD robustness check (Figure 7) addresses concerns about staggered-adoption bias and contamination from tightening reforms specifically, but shares this broader limitation.

Fourth, cross-country comparability of cause-specific mortality coding is an important caveat. Restricting the sample to the ICD-10 era (post-1998) improves comparability relative to spanning the ICD-9/ICD-10 transition, but countries continue to differ in coroner and medical examiner practices, and misclassification between suicide, accidental poisoning, and undetermined intent is well documented in the mortality literature; to the extent that such misclassification correlates with reform timing or with our institutional moderators, it could bias our estimates in either direction. Relatedly, our sample of 26 OECD economies over 1998–2019 excludes the most recent fentanyl-driven surge in North American overdose mortality after 2019 and is not necessarily representative of labor markets outside the OECD;

caution is warranted in extrapolating our estimates to lower- and middle-income countries with different labor market institutions and drug environments.

Fifth, statistical power varies across the heterogeneous-effects specifications: the poverty rate interaction, for example, is estimated on 267 country-year observations compared with up to 486 in the baseline specification (Table 1), reflecting more limited data availability for this moderator. Combined with the fact that we examine five outcomes and five institutional moderators across six horizons, some caution is warranted regarding multiple comparisons, and the moderator results discussed in Section 6.3 should be read as suggestive of institutional heterogeneity rather than as precisely estimated structural parameters.

7 Conclusion

This paper asks whether labor-market institutions that shape job security and income stability have measurable consequences for despair-related mortality. Using a panel of 26 OECD economies over 1998–2019 and a local-projection design applied to large, discrete reforms of the OECD Employment Protection Indicators, we find that major EPL deregulation is followed by a statistically and economically significant, gradually accumulating rise in mortality from drug use disorders and suicide: drug-related deaths increase by approximately 35 percent and suicide mortality by approximately 5 percent within five years of reform, with no detectable average effect on alcohol-related or cirrhosis mortality, and delayed increases in violence and inflammatory heart disease mortality. These headline findings survive an extensive set of robustness checks, including event-study evidence of flat pre-reform trends, an alternative local-projection difference-in-differences estimator, controls for a range of contemporaneous macroeconomic and institutional factors, and a test for asymmetry between deregulation and tightening episodes.

The results are consistent with labor-market institutions affecting health through economic insecurity and job instability, but the country-level design cannot establish individual mechanisms and cannot completely separate labor-market effects from contemporaneous country-specific shocks, particularly drug-supply conditions. Institutional heterogeneity provides suggestive evidence that health-system capacity may moderate the mortality response.

More broadly, evaluations of labor-market flexibility generally focus on its consequences for employment, productivity, and wages. Our results suggest there may also be health consequences worth incorporating into how such policies are evaluated.

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9 Appendix

9.1 Data Appendix

Table 5: ICD-10 Codes and Descriptions Grouped by Cause of Death

Cause of Death	**ICD-10 Code**	**Description**
Drug Use Disorders	F11	Mental and behavioral disorders due to use of opioids
	F12	Mental and behavioral disorders due to use of cannabinoids
	F13	Mental and behavioral disorders due to use of sedatives or hypnotics
	F14	Mental and behavioral disorders due to use of cocaine
	F15	Mental and behavioral disorders due to use of other stimulants, including caffeine
	F16	Mental and behavioral disorders due to use of hallucinogens
	F18	Mental and behavioral disorders due to use of volatile solvents
	F19	Mental and behavioral disorders due to multiple drug use and other psychoactive substances
	X41	Accidental poisoning by antiepileptic, sedative-hypnotic, antiparkinsonism and psychotropic drugs
	X42	Accidental poisoning by narcotics and hallucinogens
	X44	Accidental poisoning by other and unspecified drugs
Alcohol Use Disorders	F10	Mental and behavioral disorders due to use of alcohol
	X45	Accidental poisoning by and exposure to alcohol
Self-Inflicted Injuries	X60–X84	Intentional self-harm
	Y870	Sequelae of intentional self-harm
Violence	X85–Y09	Assault
	Y871	Sequelae of assault
Inflammatory Heart Diseases	I30	Acute pericarditis
	I31	Other diseases of pericardium
	I32	Pericarditis in diseases classified elsewhere
	I33	Acute and subacute endocarditis
	I38	Endocarditis, valve unspecified
	I40	Acute myocarditis
	I42	Cardiomyopathy

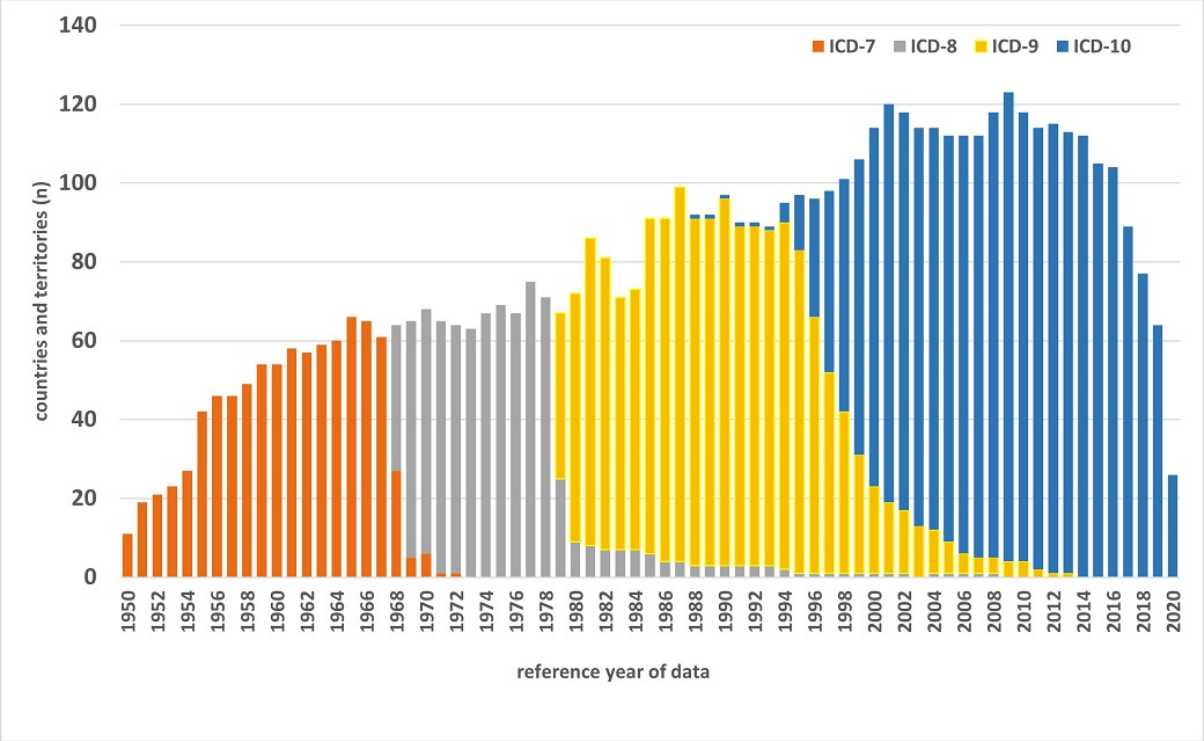
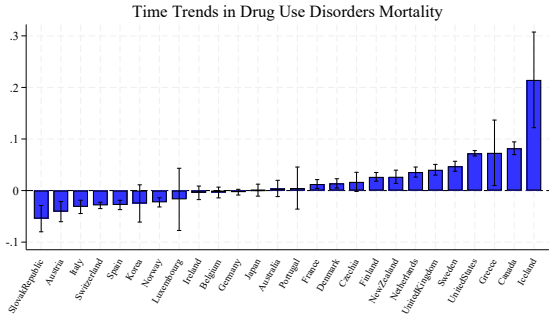
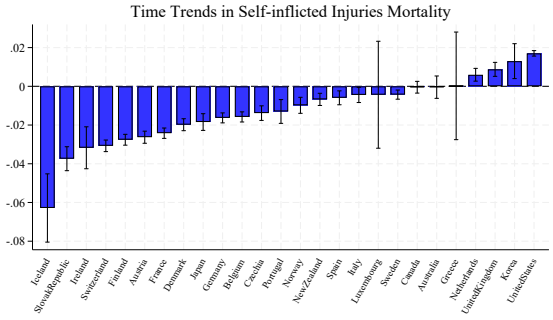


Figure 11: Number of Countries in ICD Transition

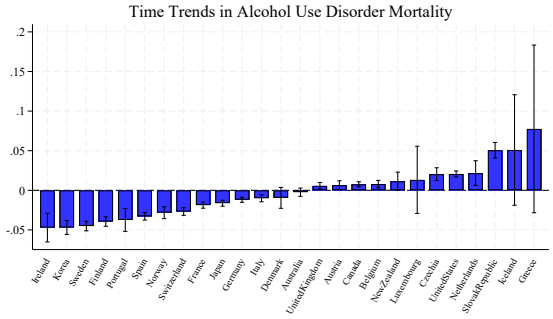
9.2 Descriptive Statistics: Country-Specific Trends



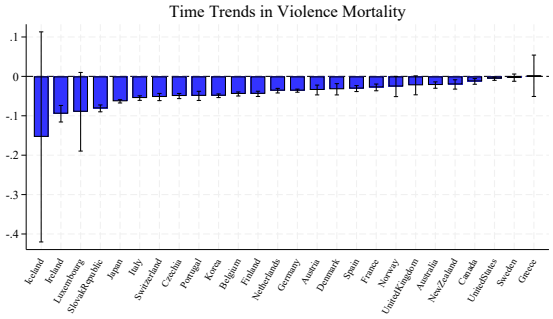
(a) ln(Drug)



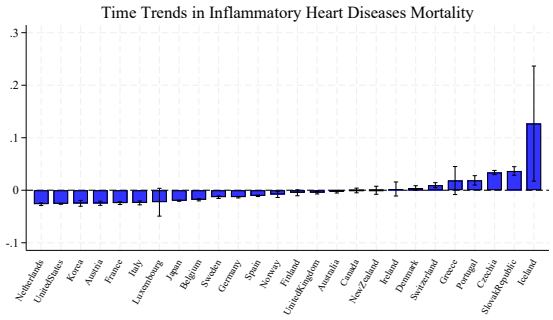
(b) ln(Suicide)



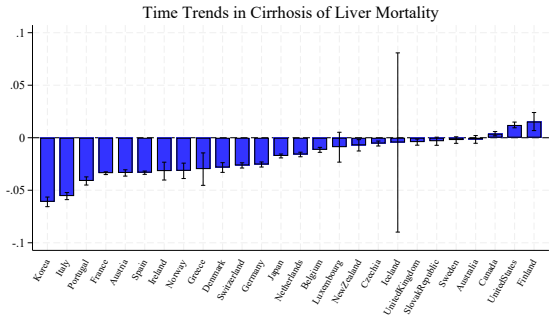
(c) ln(Alcohol)



(d) ln(Violence)



(e) ln(Inflammatory Heart Disease)



(f) ln(Cirrhosis of Liver)

Figure 12: **Country-specific time trends in log mortality rates by cause.** Each panel shows estimated linear trends in log age-adjusted mortality for the indicated cause of death of each country. Trends are obtained from the regression $Mortality_t^i = \alpha^i + \tau^i t + \varepsilon_t^i$, where the subscript t and superscript i denote year and country, respectively. $Mortality$ denotes the cause-specific mortality rates, α^i is a country-specific constant, τ^i is the country-specific linear time trend, and ε_t^i is an error term. Capped spikes indicate 90 percent confidence intervals. The estimates can be interpreted as the average annual change in a cause-specific mortality rate for each country over the sample period, which for most countries spans 1998–2019.

9.3 Additional Heterogeneity Results by Institutional Context

Table 6: Dynamic effects of EPL deregulation on mortality from self-inflicted injury: interaction terms

Interaction term with	**0y**	**1y**	**2y**	**3y**	**4y**	**5y**
Avoidable death rate	-0.0001	-0.02	-0.01	-0.03	-0.01	-0.02
	(0.0177)	(0.0306)	(0.0301)	(0.0528)	(0.0695)	(0.0789)
Social transfers expenditure	0.04	0.09**	0.06	0.10	0.16**	0.18**
	(0.0286)	(0.0403)	(0.0506)	(0.0763)	(0.0662)	(0.0782)
Public health insurance coverage	-0.0018	-0.0049**	-0.0031	-0.0039	-0.0038	0.0008
	(0.0011)	(0.0020)	(0.0030)	(0.0051)	(0.0129)	(0.0184)
Replacement rate of UB (60 months)	0.02	0.04	0.02	-0.01	-0.01	0.06
	(0.0187)	(0.0324)	(0.0397)	(0.0467)	(0.0594)	(0.0582)
Poverty rate	0.01	0.00	-0.04	-0.01	-0.04	-0.01
	(0.0320)	(0.0570)	(0.0675)	(0.0691)	(0.0790)	(0.0557)

Notes: Outcome is the **log mortality rate from self-inflicted injury (suicide)** per 100,000 population. Entries are interaction effects at horizons $h = 0$ to 5 years after a major EPL deregulation; standard errors in parentheses. Significance: $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$. If figures report percent changes, coefficients can be transformed as $100\,[\exp(\hat{\beta}_h) - 1]\%$.

Table 7: Dynamic effects of EPL deregulation on mortality from alcohol use disorder: interaction terms

Interaction term with	**0y**	**1y**	**2y**	**3y**	**4y**	**5y**
Avoidable death rate	-0.02	0.03	0.03	-0.01	0.04	0.11
	(0.0379)	(0.0540)	(0.0743)	(0.0955)	(0.1192)	(0.1170)
Social transfers expenditure	-0.18***	-0.21**	-0.12	-0.28***	-0.33***	-0.24
	(0.0411)	(0.0832)	(0.0971)	(0.0741)	(0.0985)	(0.1420)
Public health insurance coverage	-0.01	0.03***	0.02***	0.03*	0.001	-0.003
	(0.0083)	(0.0064)	(0.0061)	(0.0158)	(0.0162)	(0.0261)
Replacement rate of UB (60 months)	-0.06	-0.12*	-0.13*	-0.10	-0.04	-0.01
	(0.0448)	(0.0639)	(0.0717)	(0.0794)	(0.1417)	(0.0979)
Poverty rate	-0.12***	-0.09	0.10	0.07	0.15	-0.10
	(0.0387)	(0.0488)	(0.0617)	(0.0704)	(0.1671)	(0.0969)

Notes: Outcome is the **log mortality rate from alcohol use disorder** (per 100,000 population). Entries are interaction effects at horizons $h = 0$ to 5 years after a major EPL deregulation; standard errors in parentheses. Significance: $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$. If figures report percent changes, coefficients can be transformed as $100\,[\exp(\hat{\beta}_h) - 1]\%$.

Table 8: Dynamic effects of EPL deregulation on mortality from violence: interaction terms

Interaction term with	**0y**	**1y**	**2y**	**3y**	**4y**	**5y**
Avoidable death rate	0.08*	0.09	0.07	0.03	-0.13	0.04
	(0.0395)	(0.0756)	(0.0762)	(0.0846)	(0.1000)	(0.1450)
Social transfers expenditure	-0.02	0.03	-0.14	-0.12	-0.21	0.07
	(0.0612)	(0.0613)	(0.1603)	(0.1534)	(0.2052)	(0.2606)
Public health insurance coverage	0.01	-0.01	0.01	-0.01	-0.03	-0.06
	(0.0045)	(0.0056)	(0.0107)	(0.0167)	(0.0339)	(0.0359)
Replacement rate of UB (60 months)	0.06	0.12	0.07	0.28**	0.39***	0.54***
	(0.0684)	(0.0831)	(0.1137)	(0.0984)	(0.1204)	(0.1166)
Poverty rate	-0.04	-0.07	-0.29**	-0.10	-0.07	-0.20
	(0.0928)	(0.1088)	(0.1043)	(0.1484)	(0.1439)	(0.1843)

Notes: Outcome is the **log mortality rate from violence** (per 100,000 population). Entries are interaction effects at horizons $h = 0$ to 5 years after a major EPL deregulation; standard errors in parentheses. Significance: $^{*}p < 0.10$, $^{**}p < 0.05$, $^{***}p < 0.01$. Coefficients can be transformed to percent changes using $100[\exp(\hat{\beta}_h) - 1]\%$.

Table 9: Dynamic effects of EPL deregulation on mortality from inflammatory heart diseases: interaction terms

Interaction term with	0y	1y	2y	3y	4y	5y
Avoidable death rate	-0.004	0.003	0.02	0.07***	0.08**	0.07**
	(0.0159)	(0.0197)	(0.0175)	(0.0203)	(0.0287)	(0.0266)
Social transfers expenditure	-0.06**	-0.02	-0.04	0.02	-0.02	0.02
	(0.0234)	(0.0317)	(0.0539)	(0.0490)	(0.0721)	(0.0469)
Public health insurance coverage	-0.002***	0.001	-0.005	-0.001	-0.02**	-0.03*
	(0.0004)	(0.0010)	(0.0027)	(0.0043)	(0.0098)	(0.0177)
Replacement rate of UB (60 months)	-0.02	-0.01	0.01	0.002	0.0002	-0.02
	(0.0286)	(0.0416)	(0.0572)	(0.0537)	(0.0602)	(0.0585)
Poverty rate	-0.05	-0.06	-0.10**	-0.19**	-0.42***	-0.30***
	(0.0318)	(0.0603)	(0.0427)	(0.0658)	(0.0351)	(0.0551)

Notes: Outcome is the **log mortality rate from inflammatory heart diseases** (per 100,000 population). Entries are interaction effects at horizons $h = 0$ to 5 years after a major EPL deregulation; standard errors in parentheses. Significance: *$p < 0.10$, *$p < 0.05$, *$p < 0.01$. Percent-change interpretation (if used in figures): $100[\exp(\hat{\beta}_h) - 1]\%$.

9.4 Asymmetry Between Deregulation and Tightening

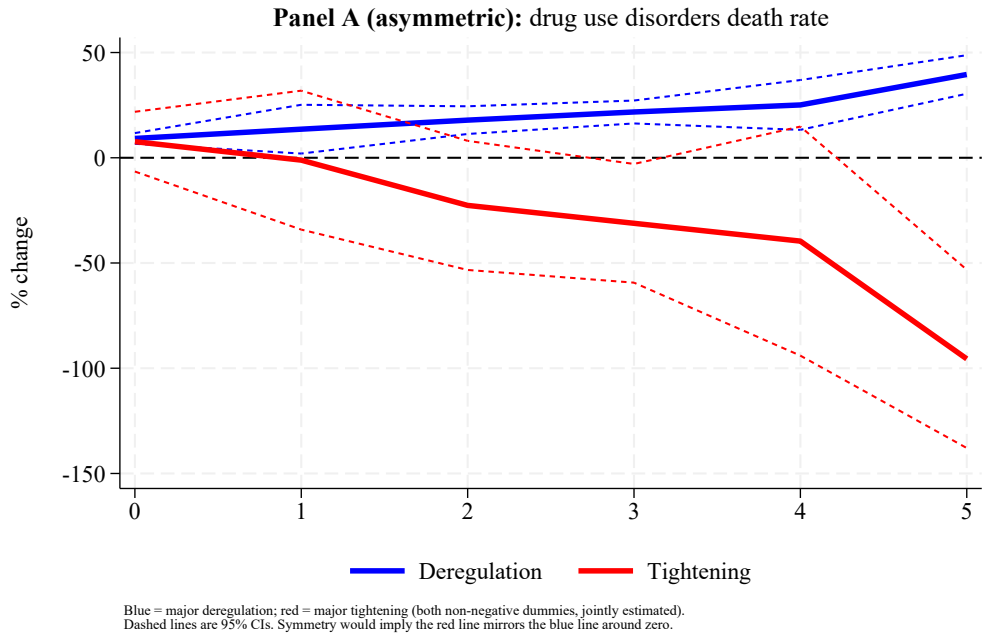


Figure 13: **Asymmetry between EPL deregulation and tightening: drug use disorder mortality.** The signed reform indicator used in the baseline specification is decomposed into two non-negative dummies, one for major deregulation episodes and one for major tightening episodes, entered jointly with their own lags and forward-term corrections. The blue line plots the deregulation coefficient (nearly identical to the baseline estimates in Table 2); the red line plots the tightening coefficient. Dashed lines are 95% confidence intervals. Under a symmetric response—the restriction imposed by construction in the baseline specification—the red line would mirror the blue line around zero. Coefficients and the corresponding horizon-by-horizon test of that restriction are reported in Table 10.

Table 10: Asymmetry between EPL deregulation and tightening: drug use disorder mortality

Horizon	**0y**	**1y**	**2y**	**3y**	**4y**	**5y**
Deregulation, β_{dereg}	**0.092**	**0.136**	**0.179**	**0.218**	**0.251**	**0.396**
	[0.066, 0.117]	[0.020, 0.252]	[0.113, 0.245]	[0.163, 0.272]	[0.133, 0.369]	[0.304, 0.487]
Tightening, $\beta_{tighten}$	0.077	−0.011	−0.226	**−0.311**	−0.396	**−0.955**
	[−0.065, 0.219]	[−0.341, 0.319]	[−0.533, 0.080]	[−0.593, −0.029]	[−0.940, 0.148]	[−1.379, −0.532]
Symmetry test p**-value**[a]	0.020**	0.466	0.761	0.517	0.608	0.016**

Notes: Outcome is the log mortality rate from drug use disorders (per 100,000 population). β_{dereg} and $\beta_{tighten}$ are the coefficients on non-negative dummies for major deregulation and major tightening episodes, respectively, jointly estimated in place of the signed `epl_flag` indicator used in the baseline specification, with their own lags and horizon-specific forward-term corrections; 95% confidence intervals in brackets. Bold coefficients indicate a 95% confidence interval that excludes zero.

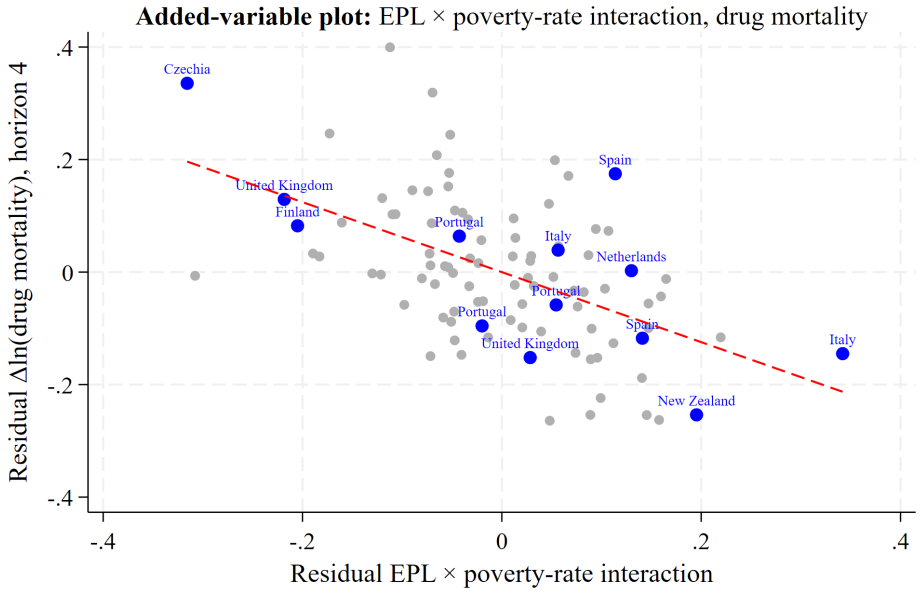
[a] Tests H_0: $\beta_{dereg} + \beta_{tighten} = 0$, i.e., that tightening reverses the mortality effect of deregulation exactly—the restriction imposed by construction in the baseline symmetric specification. *$p < 0.10$, *$\hat{p} < 0.05$.

9.5 Additional Robustness Checks

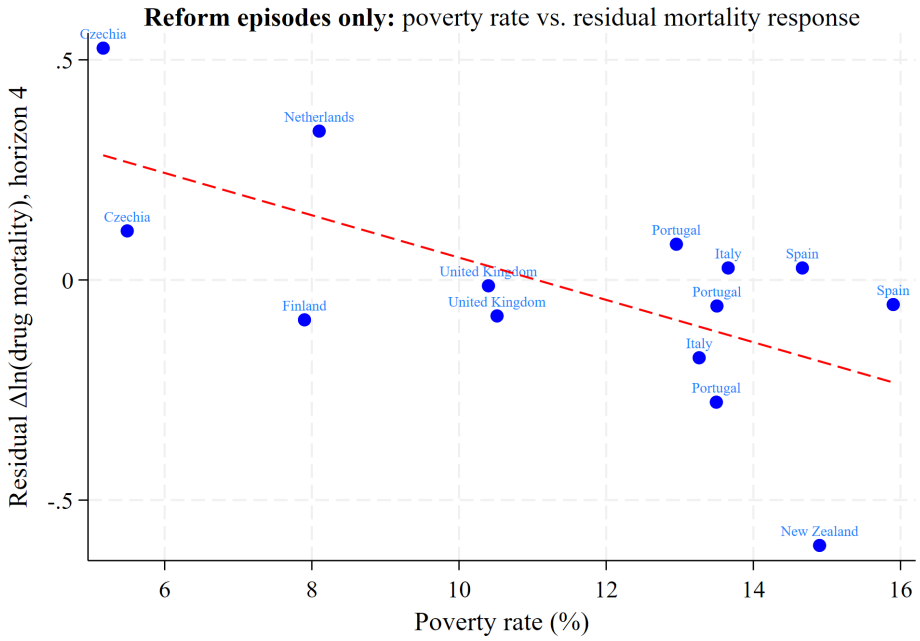
Table 11: Dynamic effects of EPL deregulation on mortality from drug use disorders by age group

Age group	**0y**	**1y**	**2y**	**3y**	**4y**	**5y**
15–19	0.03	-0.03	-0.09	-0.05	-0.08	-0.55***
	(0.1147)	(0.1774)	(0.2286)	(0.1927)	(0.1464)	(0.1176)
20–24	0.00	0.14***	0.17*	0.40***	0.16	0.36***
	(0.1006)	(0.0375)	(0.0949)	(0.0700)	(0.1140)	(0.0668)
25–29	-0.07	0.07	0.38***	0.22**	0.35***	0.50***
	(0.1093)	(0.0665)	(0.0874)	(0.0825)	(0.0510)	(0.1179)
30–34	0.08	0.12	0.31***	0.16	0.44***	0.42***
	(0.0935)	(0.1545)	(0.0557)	(0.0904)	(0.1067)	(0.0691)
35–39	0.04	0.11***	0.23***	0.20***	0.32***	0.29***
	(0.0456)	(0.0341)	(0.0657)	(0.0557)	(0.0700)	(0.0500)
40–44	0.01	-0.10	0.12	0.17**	0.22***	0.26***
	(0.0614)	(0.0824)	(0.0778)	(0.0565)	(0.0533)	(0.0615)
45–49	0.17**	0.12**	0.17**	0.11	0.07	0.25***
	(0.0668)	(0.0550)	(0.0604)	(0.0722)	(0.0982)	(0.0631)
50–54	-0.04	0.22**	0.13**	0.19	0.23***	0.48***
	(0.1236)	(0.0915)	(0.0538)	(0.1333)	(0.0531)	(0.0554)
55–59	0.01	0.11	0.08	0.15*	0.13*	0.34**
	(0.1226)	(0.0861)	(0.0963)	(0.0787)	(0.0704)	(0.1245)
60–64	0.19*	0.24**	0.35***	0.40**	0.27***	0.41***
	(0.0973)	(0.0961)	(0.0750)	(0.1317)	(0.0749)	(0.1029)
65–69	0.07	0.25*	0.12	0.32***	0.20***	0.38***
	(0.0961)	(0.1327)	(0.1489)	(0.0937)	(0.0641)	(0.0893)
70–74	-0.30**	0.06	-0.05	-0.24***	0.13	-0.17
	(0.1207)	(0.1104)	(0.0935)	(0.0772)	(0.1118)	(0.1126)
75–79	-0.10	0.11	0.14	0.06	0.16	0.14
	(0.1498)	(0.0986)	(0.0913)	(0.0879)	(0.1804)	(0.1384)

Notes: Outcome is the **log mortality rate from drug use disorders** (per 100,000 population). Entries are age-group–specific dynamic effects at horizons $h = 0$ to 5 years after a major EPL deregulation; standard errors in parentheses. Significance: *$p < 0.10$, *$\hat{p} < 0.05$, *$\hat{p}$*< 0.01. For percent-change interpretation (used in figures if applicable), transform as $100[\exp(\hat{\beta}_h) - 1]\%$.



(a) Added-variable plot



(b) Poverty rate vs. residual response, reform episodes only

Figure 14: **Is the EPL × poverty-rate interaction driven by outliers?** Panel (a) plots the residual of the horizon-3 drug mortality response against the residual of the EPL × poverty-rate interaction term, after partialling out all other regressors in the specification (country and year fixed effects, controls, lags, and forward reform terms); by the Frisch–Waugh–Lovell theorem, the slope of the fitted line reproduces the interaction coefficient reported in Table 3. Blue markers denote country-years with a major EPL reform, labeled by country (repeated country names indicate multiple reform episodes for that country); gray markers denote non-reform country-years. Panel (b) plots the same residualized mortality response against the raw poverty rate, restricted to reform episodes only. Both panels indicate that the negative interaction reflects a pattern shared across roughly a dozen reform episodes spanning the poverty distribution, rather than one or two extreme observations.

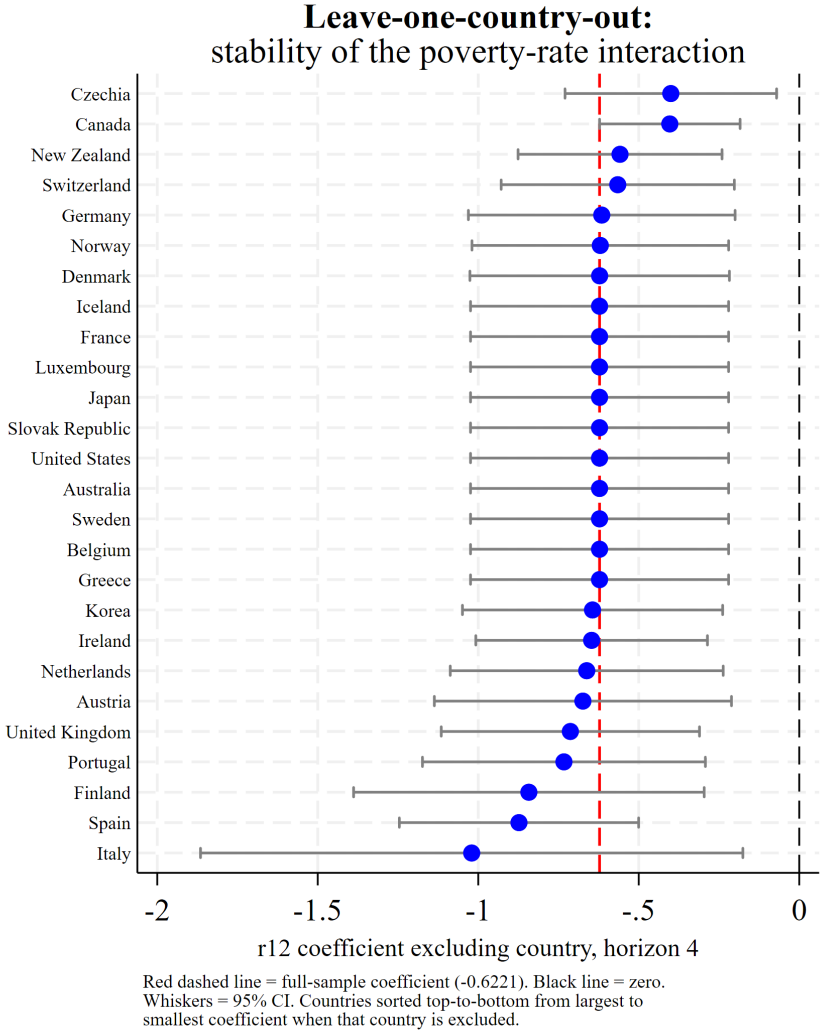


Figure 15: **Leave-one-country-out estimates of the EPL × poverty-rate interaction.** Each row re-estimates the horizon-3 interaction coefficient from Table 3 after excluding the named country from the sample. Whiskers are 95% confidence intervals; the red dashed line marks the full-sample coefficient (-0.622). The estimated coefficient remains negative, with a confidence interval that excludes zero, regardless of which single country is dropped, ranging from -0.35 (excluding Czechia) to -1.05 (excluding Italy).