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Research Statement

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My research examines how labor-market institutions, financial structure, and the distribution of income shape population health, and how the resulting health dynamics feed back into macroeconomic performance. This agenda sits at the intersection of labor economics, health economics, and macroeconomics, and centers on three dissertation chapters: an empirical study of employment protection deregulation and deaths of despair across advanced economies, a theoretical model that embeds substance-abuse dynamics into a macroeconomic growth-cycle framework, and an early-stage empirical project on local financialization, financial distress, and deaths of despair in the United States. Together, these chapters build a research program that treats population health not as a byproduct of economic conditions to be measured after the fact, but as a structural feature of labor markets, financial systems, and macroeconomic dynamics in its own right.

Chapter 1: Employment Protection Deregulation and Deaths of Despair (Job Market Paper)

My job market paper asks whether policies that make it easier for employers to dismiss workers carry measurable public-health costs. A large literature evaluates employment protection legislation (EPL) reforms in terms of employment and wage outcomes, while a separate literature documents the rise of "deaths of despair"—mortality from drug overdose, alcohol-related disease, and suicide—first identified in the United States by Case and Deaton (2020). These two literatures rarely intersect: the downstream health consequences of labor-market deregulation remain largely unexamined outside the U.S. context.

I address this gap using OECD employment protection indicators and World Health Organization cause-specific mortality data for 26 advanced economies from 1998 to 2019. I identify major EPL reforms as large discrete movements in the OECD's employment protection index and estimate their dynamic effects on age-standardized death rates using Jordà's (2005) local projection method, embedded in a panel framework with country and year fixed effects and controls for reverse causality. I find that major EPL deregulation is followed by a significant rise in drug-related mortality—approximately 35 percent higher than pre-reform levels within five years—and a smaller but significant rise in suicide mortality of roughly 5 percent over the same horizon; the effect on alcohol-related mortality is not statistically distinguishable from zero, pointing to heterogeneous causal pathways across components of despair-related mortality. I further show that these effects are not uniform: countries with more generous unemployment benefits, larger social transfers, stronger public health-insurance coverage, and more effective health systems (lower avoidable mortality) experience substantially smaller increases in deaths of despair. This pattern is consistent

with an institutional-buffering mechanism, in which protections outside the labor market shape how economic insecurity translates into mortality risk.

This paper contributes to labor economics by extending the evaluation of job-protection reforms beyond employment and wage aggregates to their population-health externalities, and to social epidemiology by showing that macro-institutional shifts, not only individual-level job loss, can move despair-related mortality at the country level. The results carry direct policy relevance: they suggest that employment protection functions not only as a labor-market institution but as a component of a country's public-health infrastructure, and that flexibility-enhancing reforms should be paired with robust income supports and health-system capacity to limit unintended harm.

Chapter 2: The Addicted Predator–Prey Model: How Substance Abuse Shapes Productivity and Growth-Cycle Dynamics (with Marwil J. Dávila-Fernández)

My second chapter turns from measuring the health consequences of labor-market institutions to modeling how those consequences feed back into aggregate economic dynamics. Standard macroeconomic growth-cycle models offer limited guidance on this feedback: in Goodwin's (1967) classic predator-prey model, the wage share and employment rate oscillate endogenously through the tension between capital accumulation and workers' bargaining power, but labor productivity growth is treated as exogenous and the workforce as homogeneous—assumptions that are difficult to sustain in an environment where income compression raises the prevalence of substance use disorder, which in turn depresses effective productivity.

This paper introduces substance abuse into the Goodwin framework as an endogenous channel linking income distribution to aggregate productivity. Using a continuous-time discrete-choice mechanism, workers transition between drug-abusing and drug-free states in response to the wage share: a declining wage share raises the probability of substance use disorder, while a rising wage share lowers it. This behavioral channel enters the economy through a productivity damage function that scales with the prevalence of substance use among workers, so that the standard Goodwin cycle is amplified by a health channel operating in the same direction as capital accumulation. We ground the model's key mechanisms empirically using U.S. state-level data from 1998 to 2019, documenting that positive labor-share gains—particularly of moderate severity—reduce drug-induced mortality over one-to-four-year horizons. On the theoretical side, we establish three results: the resulting three-dimensional nonlinear system admits a unique, economically meaningful interior equilibrium; applying the existence part of the Hopf bifurcation theorem, we show that as the autonomous wage-growth parameter crosses a critical threshold, the equilibrium loses stability and the system bifurcates into persistent, bounded fluctuations; and numerical simulations above this threshold reveal the coexistence of multiple stable limit cycles, so that the long-run amplitude of the cycle depends on initial macroeconomic conditions as well as structural parameters.

The model yields policy lessons that a purely empirical approach cannot. Policies that weaken the

sensitivity of substance-use behavior to income fluctuations—expanded social safety nets, universal access to mental-health and substance-use treatment, and housing security programs—compress the amplitude of growth cycles and reduce macroeconomic instability, carrying a stabilizing dividend beyond their direct health benefits. By contrast, policies that reduce the on-the-job productivity cost of substance use disorder without addressing its underlying drivers have an ambiguous effect on cyclical stability, since they can weaken a self-correcting feedback loop that runs from productivity damage back through employment and wages to recovery. This paper contributes to the post-Keynesian growth-cycle literature by identifying a novel behavioral-health channel of macroeconomic instability, and connects to public-health policy by showing that safety-net and treatment investments have macroeconomic stabilization properties, not only direct welfare benefits.

Chapter 3: Local Financialization, Financial Distress, and Deaths of Despair (Work in Progress)

My third chapter turns to a second macro-institutional channel behind rising mortality: the expansion of finance and real estate within local economies. Motivated by the parallel rise of financialization and deaths of despair in the United States, I ask whether local financialization causally affects mortality through its effect on household financial distress. Building on evidence that mortgage delinquency, foreclosure, and credit constraints raise psychological distress and suicide risk independently of employment and income, I propose a conceptual chain running from local financialization to credit-market fragility, to financial distress, to socioeconomic stressors, to deaths of despair.

I plan to identify this channel using a two-stage least squares design at the state and county level: the first stage relates measures of financial distress (mortgage delinquency and foreclosure rates, business lending distress) to local financialization (the shares of finance, insurance, and real estate in state GDP, with FIRE employment share, household debt-to-income, and bank assets per capita as alternative measures); the second stage relates mortality to the instrumented distress measure. Identification will rely on state and year fixed effects with state-specific trends, lagged instruments to rule out contemporaneous reverse effects, interactions of pre-period financialization with national credit shocks to isolate plausibly exogenous local credit exposure, and county-level replication to hold state policy fixed. This chapter is at an early stage—I have developed the conceptual framework, empirical design, and candidate data sources, and am now assembling the panel—but it extends the dissertation's core question to a second institutional channel: financial structure, alongside labor-market regulation, as a determinant of population health.

Other Future Directions

Beyond the dissertation, I plan to extend Chapter 1's cross-country design to worker-level and regional data to identify the mechanisms underlying the aggregate mortality response, and to

examine how EPL reforms interact with other structural policies, such as housing protections, in shaping health outcomes. On the theoretical side, I plan to extend the growth-cycle model in Chapter 2 to incorporate Minsky's financial instability hypothesis, in which firms finance investment through debt and debt accumulation interacts with the health-behavior channel already embedded in the model—connecting that chapter's labor-health nexus to the real-financial interactions studied in Chapter 3 and in the financial-instability literature more broadly. Other extensions of Chapter 2 include relaxing the assumption of a fixed labor force to let substance-use prevalence affect labor supply directly, and moving from a closed-economy to a multi-region setting that speaks to the geographic concentration of labor-share shocks observed in the data.

Across both empirical and theoretical work, my methodological toolkit includes local projection methods for dynamic causal estimation, instrumental-variables and two-stage least squares designs, nonlinear dynamic systems and bifurcation analysis, and standard panel-data econometrics, implemented in Stata, R, and Python. I am committed to a research agenda that treats health and macroeconomic outcomes as jointly determined, and I look forward to continuing this work in ***[department/institution type, e.g., a research-intensive economics department]***.