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Essays on labor markets and health: Employment conditions and drug, suicide, and alcohol-related mortality among working-age adults in the United States

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Essays on labor markets and health: Employment conditions and drug, suicide, and alcohol-related mortality among working-age adults in the United States

by

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Committee in charge:

Professor William H. Dow, Chair
Professor Hilary Hoynes
Professor Michael Reich

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Abstract

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Americans in the twenty-first century are dying earlier in life and at higher rates from preventable causes than in nearly any other developed economy. Understanding of the root determinants of the recent reversal in life expectancy and identifying policy approaches to combat the rise in midlife mortality is a national public health and economic imperative. This dissertation focuses on the well-documented increase in fatal drug overdose, suicide, and alcohol-related mortality—a collection of causes of death often referred to as the "deaths of despair"—and examines the potential economic determinants of the acceleration in these causes of death over the past several decades. Building upon extensive literature examining macroeconomic and labor market conditions as upstream factors shaping population health, the following chapters consist of two empirical analyses intended to estimate the causal effect of short- to medium-term changes in local employment rates on these causes of death among working-age adults during the 2003-2017 period. These studies are of increasing importance as the United States continues to experience widespread employment uncertainty and prolonged economic distress in the wake of the COVID-19 pandemic.

The first study presented in this dissertation focuses on the effects of county-level employment conditions on "deaths of despair" using a Bartik-style shift-share instrument to isolate demand-driven variation in county-level employment rates. In line with most existing studies that document countercyclical variation in suicide, I estimate that a one percentage point increase in the current-year employment-to-population ratio decreases non-drug suicide rates by one to two percent. On the other hand, my causal models suggest that rates of fatal drug overdose increase by a similar magnitude as the economy improves, and I find no evidence of changes in alcohol-related mortality in response to short-term employment shocks. I conduct a simulation exercise based on these point estimates to show that in general, and especially for accidental drug overdose, these estimated effects are small relative to the increases in cause-specific mortality over the 2003—2017 period.

Motivated by the procyclical variation in accidental drug overdose uncovered in the

first study, the second analysis examines the extent to which county employment rates affect the demand for prescription opioid medication among a population of commercially insured adults. This study draws on deidentified, individual-level pharmacy and medical claims from 2003—2017 aggregated to the county level to test the hypothesis that county-level employment fluctuations differentially affect the demand for prescription opioids that place individuals at higher (versus lower) risk for abuse and dependence. Unlike existing studies, I find no evidence of an effect of employment conditions on the demand for prescription opioids overall or differential effects between high- and low-risk prescriptions.

The relatively small magnitude of the estimated effects, suggestive evidence of heterogeneity across demographic groups, and mixed findings on the cyclicality of these causes of death over various time horizons all point to a more complex set of factors underlying the rising rates of "deaths of despair" that is not explained by local employment rates alone. Developing a more nuanced understanding of these trends—particularly along key dimensions such as race/ethnicity and socioeconomic status—will be critically important in designing equitable policies to help the country recover from the COVID-19 pandemic and to reverse the disconcerting trends of increasing midlife mortality in the years to come.

To Cami, Mom, Dad, Adam & Erin

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Chapter 1

Introduction

Americans in the twenty-first century are dying earlier in life and at higher rates from preventable causes than in nearly every other developed economy. Between 2014 and 2017, life expectancy at birth in the United States decreased for three consecutive years—a trend not seen in this country since the influenza pandemic of 1918–1919 (National Academies of Sciences, Engineering, and Medicine, 2021). Despite spending twice as much per capita on healthcare relative to the median among all OECD countries, the United States has trailed peer countries in key health measures such as life expectancy and working-age mortality rates for decades. Developing a complete understanding of the root determinants of this reversal in life expectancy—and identifying policy approaches to combat the rise in U.§. midlife mortality—is a national public health and economic imperative.

It is widely acknowledged that increases in midlife mortality and the reversal in life expectancy reflect the combination of two major factors that have unfolded over the past several decades. First, progress toward reducing mortality from leading causes of death such as ischemic heart disease and circulatory diseases has stalled, particularly among working-age adults. Meanwhile, rates of mortality from drug poisoning, suicide, and alcohol-related causes—a collection of causes of death often referred to as the "deaths of despair"—have dramatically increased among this same population (Case and Deaton, 2020a; National Academies of Sciences, Engineering, and Medicine, 2021; Woolf and Schoomaker, 2019). For example, a recent report from the National Academies of Sciences, Engineering, and Medicine estimated that between 1990 and 2017, drugs and alcohol contributed to more than 1.3 million deaths of 25- to 64-year-olds in the US, while nearly 570,000 died by suicide. During this time, drug poisoning mortality increased 538% (from 3.4 to 21.7 per 100,000), making it the largest contributor to the overall increase in mortality among working-age adults (National Academies of Sciences, Engineering, and Medicine, 2021)

This dissertation focuses on the second set of contributing factors—the so-called "deaths of despair"—and the potential economic determinants of these trends. The term "deaths of despair" traces back to the pioneering work of economists Anne Case and Angus Deaton, who were among the first to document these startling increases in drug, suicide, and alcohol (DSA) mortality and point to the disproportionately high rates of these causes of death among

working-age adults without a college degree (Case and Deaton, 2015, 2017). Motivated by this latter finding and the fact that these trends have been (until recently) predominantly driven by increased mortality rates among non-Hispanic White adults, Case and Deaton (2017) introduced the term to encapsulate a hypothesis in which this surge in DSA mortality reflects widespread social, economic, and psychological distress driven by "a long-term process of decline, rooted in the steady deterioration of job opportunities... [and] other changes in society that made life more difficult for less educated people" (Case and Deaton, 2017, p. 429). In their explanation for these trends, Case and Deaton allude to increases in chronic pain, the deterioration of social institutions such as marriage and church, and a prohibitively costly healthcare system—all against the backdrop of an economy that no longer supports those without a college degree—as key factors contributing a sense of "cumulative disadvantage" and despair.

While the trends in DSA mortality are unequivocal, the "deaths of despair" hypothesis has been criticized on many grounds, including the relative role of economic conditions as potential underlying drivers. The analyses in this dissertation focus exclusively on the role of employment and local labor market conditions—which are operationalized in the following chapters using county-level measures of the employment-to-population ratio—as potential determinants of the "deaths of despair" epidemic. Case and Deaton's hypothesis is more nuanced than the assertion that a generation of Americans have turned to suicide and other self-harming behaviors to cope with employment-related distress (in fact, they explicitly reject the notion that short-term fluctuations in unemployment rates or economic factors alone account for the rise in mortality), yet a key thread in the argument remains the role of a changing labor market that has left lower-skilled individuals behind.

The potential macroeconomic determinants of these causes of death have been extensively documented in existing research. In fact, indications of an inverse relationship between business cycles and suicide rates were reported nearly a century ago (Ogburn and Thomas, 1922). However, causal evidence linking underlying economic distress to increases in DSA remains mixed (Case and Deaton, 2020a; Currie and Schwandt, 2020; Maclean et al., 2020; Ruhm, 2019). In Chapter 2, I discuss this debate in more detail, and importantly, distinguish the varied literature on the effects of aggregate employment conditions on mortality from a more cohesive body of evidence linking individual-level job loss or precarious employment to adverse mental and behavioral health outcomes. This distinction is important given the many, often countervailing, mechanisms hypothesized to link individual-level employment status and changes in aggregate economic conditions more broadly to individual and population-level health outcomes.

The debate surrounding the underlying drivers of "deaths of despair" fits naturally into a broader literature documenting the socioeconomic gradient in health outcomes, and more specifically, the role of employment and labor market conditions as determinants of health. While there is widespread acknowledgment across disciplines that employment status and health are positively correlated at the individual level, identifying the causal effect of labor market conditions on health remains a central challenge due to reverse causality and unmeasured confounding (Avendano and Berkman, 2014; Currie and Madrian, 1999). To

address this identification challenge, the analyses in this dissertation employ a shift-share instrumental variables approach in an effort to isolate the effect of exogenous demand-side variation in local employment conditions. This technique, described in more detail in Chapter 2, has become increasingly common in studies examining the effect of changes in labor market conditions on DSA mortality and related outcomes (e.g., Autor et al. 2019; Betz and Jones 2018; Pierce and Schott 2020, among others).

This dissertation presents two empirical analyses aimed at developing a better understanding of the intersection between local labor market conditions and DSA mortality among working-age adults in the United States. In Chapter 2, I examine the effect of short- and medium-term changes in local labor market conditions on cause-specific mortality rates during the 2003–2017 period using an area-study approach. This study draws on aggregated death certificate and employment data at the county level to examine potential contemporaneous and dynamic effects of local employment rates on DSA mortality. Consistent with most studies of business cycle fluctuations on suicide mortality, I find that increases in the county-level employment rate lead to significant contemporaneous reductions in non-drug suicide—a relationship that appears to be driven by changes in suicide among older men. In contrast, I find that improvements in the employment rate are associated with increasing mortality due to unintentional drug overdose, and that this effect is primarily seen among younger populations. I find no evidence that alcohol-related mortality varies as a function of the county-level employment rate. Together, these findings point to the need for a nuanced set of policy approaches to address the unique drivers of these causes of death overall and among especially vulnerable populations.

In Chapter 3, I explore potential mechanisms underlying the procyclical variation I identify in drug non-suicide. Drawing on aggregated medical and pharmacy claims data at the county level, I test the hypothesis that the demand for high-risk prescription opioids (i.e., those that present an individual with a high likelihood of addiction and abuse) vary countercyclically, while the demand for low-risk opioid prescriptions (i.e., those that serve a targeted, therapeutic purpose for pain relief) vary procylically. In testing this hypothesis, I find little evidence that changes in the county-level employment rate affect the demand for either type of prescription opioid.

Understanding the role of labor market conditions as determinants of health has never been more important. At the time of this writing (December 2021), COVID-19 had claimed over 800,000 American lives and led to an economic recession of unprecedented magnitude. Between February and April 2020, the United States economy lost 21 million job—twice as many as were lost during the Great Recession—and the employment-population ratio plummeted from a pre-pandemic high of 57.4% to 52.9% by the end of the second quarter of 2020 (Smith et al., 2021). While the economy has recovered many of these lost jobs, the employment rate remains well below pre-pandemic levels, and the burden of job loss and economic insecurity (as well as the toll of the virus itself) has fallen disproportionately upon Americans in lower-wage occupations and among communities of color (Center on Budget and Policy Priorities, 2021).

The analyses in the following chapters do not consider the role of the COVID-19 recession

on deaths of despair—much less the compounding effects of extensive disruptions to daily life, widespread trauma, and forgone healthcare that have defined the COVID-19 era—yet findings from this dissertation are suggestive of key directions for future research on this important topic. For example, results from Chapter 2 are indicative of heterogeneity in the relationship between employment conditions and increases in cause-specific mortality across demographic groups, suggesting certain populations may be more vulnerable to the adverse effects of the COVID-19 recession. Experiences of job loss as a result of the pandemic have occurred disproportionately in the service industry and among part-time workers—two segments of the labor force highly represented by women and younger workers—and the adverse health effects of persistent unemployment among women may be compounded by stress associated with increases in caretaking responsibilities (Smith et al., 2021). Other key differences, defined by remote work options, exposure to frontline work, and access to healthcare, may all exacerbate disparities in deaths of despair attributable to the COVID-19 pandemic. Future research on emerging disparities in these causes of death, as well as the underlying role of economic factors including employment and economic distress, will be critical in ensuring an equitable recovery in the post-COVID-19 era.

Chapter 2

Instrumental variables estimates of county-level employment fluctuations on drug, suicide, and alcohol-related mortality

ABSTRACT

The socioeconomic gradient in mortality due to drug, suicide, and and alcohol-related diseases (DSA) has been extensively documented in recent research, yet causal evidence linking underlying economic distress to increases in these "deaths of despair" remains mixed and controversial. This study contributes to this literature by estimating the current-year effects of county-level employment conditions on DSA mortality using a Bartik-style shift-share instrument to isolate demand-side variation in local employment rates. In line with most existing studies that document countercyclical variation in suicide, I estimate that a one percentage point increase in the county-level employment-to-population ratio decreases non-drug suicide rates in the current year by one to two percent. On the other hand, my causal models suggest that rates of fatal drug overdose increase by a similar magnitude as the economy improves. This set of results is of increasing importance as the United States continues to experience employment uncertainty and prolonged economic distress in the wake of the COVID-19 pandemic. However, the relatively small magnitude of the estimated effects and mixed findings on the cyclicality of suicide versus drug-related mortality point to a more nuanced set of factors underlying the rising rates of DSA mortality over longer time horizons that is not explained by local employment rates alone.

2.1 Introduction

Midlife mortality from drug overdose, suicide, and alcohol-related diseases (DSA) has risen dramatically over the past several decades and contributed to the recent decline in life

expectancy in the United States. Between 1999 and 2017, the rate of fatal drug overdoses among adults aged 24–64 increased by 386%, while suicides grew by 38.3% (Woolf and Schoomaker, 2019). Between 2006 and 2016, young adult mortality due to alcoholic liver disease and cirrhosis of the liver increased by an average of nearly 8% per year among men and over 11% per year among women (Centers for Disease Control and Prevention, 2019). There is growing concern and accumulating empirical evidence that widespread economic, social, and psychological distress fueled by the COVID-19 pandemic may be furthering these disconcerting mortality trends (Czeisler et al., 2020; Farooq et al., 2021; Friedman and Akre, 2021; Holland et al., 2021).

While the recent increases in mortality have been extensively documented in recent research, causal evidence linking underlying economic distress to the acceleration of DSA mortality remains mixed. The purpose of the current study is to estimate the effect of short- and medium-term changes in local labor market conditions on DSA mortality among working-age Americans using an instrumental variables approach designed to isolated labor demand-driven changes in employment conditions. Using an area-study approach that draws on aggregated death certificate and employment data from 2003–2017, I find that increases in the county-level employment rate lead to significant contemporaneous reductions in non-drug suicide—an effect that appears to be concentrated among older males (ages 45–64). In contrast, I find evidence that mortality due to unintentional drug overdose varies in a pro-cyclical pattern as well as suggestive evidence that these aggregate effects are driven largely by increases among younger females (ages 19–44) and older males in response to improving economic conditions.

Following previous literature demonstrating heterogenous effects of demographic group-specific employment changes on the production of health (Autor et al., 2019; Lindo et al., 2018; Page et al., 2019; Schaller, 2016), I leverage detailed demographic information from the Quarterly Workforce Indicators to decompose aggregate employment shocks into age-by-sex subgroups. I use these disaggregated measures to estimate own- and cross-group effects of changes in group-specific employment rates on deaths due to drugs, suicide, and alcohol-related causes, which allows for an exploration of whether the aggregate effects are more likely to be driven by individual-level job loss or more indirect mechanisms. This exercise reveals several findings that are suggestive of the widespread impact of employment rate changes on health outcomes across the population. However, I find that all estimates from models with disaggregated mortality rates are sensitive to adjustments for multiple hypothesis testing, and I therefore view these findings as substantive insofar as they point to potential heterogeneities and indirect mechanisms that should be analyzed more rigorously in future research.

Several scholars have argued that, while there may indeed be causal effects of macroeconomic fluctuations on some despair-related outcomes, they are relatively small in magnitude and are thus unable to explain the dramatic increase in DSA mortality (Maclean et al., 2020; Ruhm, 2019). To quantify these effect sizes, I use estimates from the aggregate and demographic group-specific models to construct counterfactual, "employment-attributable" mortality rates for the 2003–2017 period. In doing so, I demonstrate that the magnitude of

the estimated effects in this study is small in comparison with the underlying increases in cause-specific mortality during this time, particularly for drug non-suicide. However, these simulations reveal important nuances in the timing and magnitude of the effects of employment conditions on mortality across subgroups and causes of death. In particular, I observe differences between older and younger males in the degree to which employment shocks due to the Great Recession may have contributed to an acceleration of non-drug suicide rates. While Case and Deaton have largely dismissed the role of the Great Recession as a driving factor behind increases in DSA (Case and Deaton, 2020a, 2021), the magnitude and timing of the employment-attributable non-drug suicide rates among younger males suggest that this period of decreased employment among younger males may have contributed to an increase in suicide among that demographic group.

I further extend the extant literature on the economic drivers of DSA mortality by exploring the dynamics of cause-specific mortality in response to medium-term changes in county-level employment. I estimate dynamic effects using two modeling approaches. First, I estimate a series of stacked long difference models over increasing time horizons (up to five years) to examine how longer periods of employment growth or decline may affect mortality trends. Second, I estimate distributed lag models that allow for health outcomes to adjust over time in response to acute employment changes. I find few consistent findings from these models and note that the duration of my study period (2003–2017) is likely too short of a panel to adequately study dynamic effects. Moreover, I show how the results from these models—as well as the contemporaneous models described above—are highly sensitive to choices made as a researcher regarding the time period of analysis, and to a lesser extent, the geographic unit of observation.

This work contributes in several ways to the burgeoning literature on the role of aggregate economic conditions on DSA mortality. First, by employing a shift-share instrumental variables approach, I attempt to separate the effects of changes in labor demand from potentially confounding factors simultaneously affecting mortality trends at the local level—a source of endogeneity that plagues much of the existing work on aggregate employment conditions and mortality. Second, I take advantage of detailed demographic information from a jobs-level data set to generate age-by-sex group-specific shocks in employment, which allows for a more nuanced examination of how employment changes experienced by one group may have spillover effects on the health of other segments of the labor market. While these exercises yield several noteworthy findings that are consistent with existing literature, I find that many of these results are inconsistent across different model specifications and time horizons, and in many cases, they are highly sensitive to adjustments for multiple hypothesis testing.

This chapter begins with a non-exhaustive overview of the literature on "deaths of despair" with a particular focus on the debate surrounding the role of economic factors and theoretical considerations linking aggregate employment conditions to despair-related outcomes. Section 2.3 outlines the methods used in the empirical analysis of county-level employment conditions on cause-specific mortality, including a detailed description of the contemporaneous and dynamic models and the instrumental variables shift-share approach. Section 2.4 presents key results from this analysis, while Section 2.5 probes the robustness of these

findings with particular attention paid to the time period of analysis. Finally, Section 2.6 contextualizes the key findings and policy implications from this study, discusses limitations of this empirical approach, and expands upon key dimensions of heterogeneity to be examined in further research.

2.2 Related literature

In their widely cited study of mortality trends in the United States, Case and Deaton (2015) highlighted a startling increase in midlife mortality among middle-aged, non-Hispanic White adults, largely driven by increases in mortality due to suicide, drug overdose, and alcohol-related diseases (DSA). While not the first to identify these disconcerting patterns, Case and Deaton were among the earliest to suggest that the disproportionately higher DSA mortality rates experienced by Americans without a bachelor's degree were key to understanding this dramatic increase in midlife mortality. Specifically, they pointed to decreasing economic opportunity among lower educated adults—along with increased social and psychological "despair" due to widespread increases in physical pain, the unraveling of social institutions, and a prohibitively expensive healthcare system—as a key factor underlying these trends. These findings generated enormous interest in better understanding this phenomenon, which has since led many scholars to dispute Case and Deaton's "despair" narrative on several grounds.

In particular, many researchers point to the timing and magnitude of the rise in midlife mortality and argue that the rise in DSA mortality—the majority of which is attributable to drug overdose deaths—tracks more closely with trends in the marketing and availability of highly addictive prescription (and more recently, synthetic) opioids than with macroeconomic conditions (Currie and Schwandt, 2020; Maclean et al., 2020; Ruhm, 2019). In the economics literature on this topic, this is frequently referred to as the "supply" versus "demand" debate, where the former emphasizes the foundational role of opioid-supply factors as setting the stage for the crisis, while demand-side factors (e.g., economic distress) acted to accelerate these trends and define the populations most affected (Maclean et al., 2020). In their most recent working paper Case and Deaton argue precisely the opposite unfolding of events in which high levels of physical pain and economic distress created an underlying demand for opioids, where "pharma companies targeted places that were hurting, where jobs had been lost, and where pain was prevalent" (Case and Deaton, 2021, p. 10).

In critiques of Case and Deaton's despair narrative, researchers also point out that dramatic increases in drug fatalities are not unique to areas that have experienced recent economic decline, and that the phenomenon of rising midlife mortality is particularly acute among Whites relative to non-Whites, despite the fact that non-Whites have experienced more economic hardship (Diez Roux, 2017; Masters et al., 2017; Ruhm, 2019). A related stream of criticism comes from scholars who point out that the education gradient underlying the increase in DSA mortality among non-Hispanic White adults does not extend to other demographic groups, undermining a "catch-all" explanation for the growing degree

of inequality in life expectancy across levels of educational attainment (Geronimus et al., 2019). Finally, many note that the term "despair"—despite being posited as the proximate cause of DSA mortality—lacks a coherent and consistent definition in the literature, making it difficult to isolate and empirically test potential mechanisms underlying the "deaths of despair" hypothesis (Rehder et al., 2021; Ruhm, 2021a; Shanahan et al., 2019).[1]

2.2.1 Macroeconomic conditions and health outcomes

This debate exists against the backdrop of a large body of literature examining the health effects of changes in aggregate economic conditions, and in particular, the role of weak labor markets as distal factors affecting population health. While individual job loss or experiences of acute economic insecurity have been consistently linked to negative mental and behavioral health outcomes, research findings on the effects of aggregate macroeconomic or business cycle fluctuations less conclusive (Burgard et al., 2013; Catalano et al., 2011). Foundational work by Ruhm throughout the early 2000s highlights a generally procyclical relationship between macroeconomic conditions and overall mortality—a set of results largely driven by decreases in motor vehicle fatalities during economic downturns and increases in many health-promoting behaviors (Ruhm, 2000, 2003, 2005) resulting from changes in the opportunity cost of time. However, one consistent exception to this procyclical trend is suicide, which has emerged as the sole cause of death to consistently increase during economic declines. It is likely that these results reflect a fundamentally different relationship between macroeconomic conditions and mental versus physical health. The large body of literature on the health effects of the Great Recession reinforce these earlier findings on the countercyclical nature of suicide (Chang et al., 2013; Modrek et al., 2013; Reeves et al., 2012).

Importantly, more recent findings on overall mortality that include the Great Recession and subsequent economic recovery point to a weakening of the procyclical mortality pattern and reveal strong countercyclical variation for certain causes of death such as such as accidental poisoning (Ruhm, 2015). Ruhm attributes this change primarily to increases in fatal overdoses involving opioids and benzodiazepines, which is largely consistent with findings from more recent studies documenting countercyclical patterns in mortality due to poisoning from illicit substances (Carpenter et al., 2017; Hollingsworth et al., 2017). However, the broader literature on macroeconomic decline and substance use (including alcohol) is more mixed, primarily pointing to increases on the intensive rather than extensive margin. For alcohol in particular, several studies find that heavy drinking and binge drinking (i.e., those most associated with severe alcohol-related outcomes) increase during weak economic times while overall alcohol consumption decreases (Dee, 2001; Dávalos et al., 2012; Kerr et al., 2017; Lo and Cheng, 2013).

[1] Case and Deaton also acknowledge that the concept of despair is "not a well-defined diagnostic category, let alone one with a clinically validated measure" (Case and Deaton, 2021, p. 5). For these reasons, I opt to refer to this collection of mortality outcomes throughout this paper as DSA rather than "deaths of despair" to recognize that these causes of death have distinct determinants and etiological processes, and whether they share a common root cause of "despair" remains an empirical question.

2.2.2 Theoretical considerations

There are countervailing behavioral responses to aggregate economic downturns that may explain the often-contradictory findings in this literature. Consistent with the "deaths of despair" narrative, individual-level job loss and economic distress associated with a recession is likely to increase stress, adversely affecting mental health and potentially leading to deleterious coping behaviors such as the consumption of alcohol or other substances. Relatedly, employment gain may increase access to mental and behavioral health services through employer-sponsored health insurance or induce individuals to reduce consumption of harmful substances due to workplace policies. Together, these individual-level effects may underlie the countercyclical patterns in DSA mortality found in more recent aggregate studies. In contrast, tighter individual budget constraints experienced during difficult economic times may decrease consumption of harmful goods (e.g., tobacco, alcohol), and decreases in the opportunity cost of time may improve individual health by making health-promoting activities more attractive (Grossman, 1972; Ruhm, 2000). Moreover, stress and adverse stress-related responses may in fact decrease as a result of detachment from work (Catalano et al., 2011).

One likely mechanism through which macroeconomic conditions may impact mortality is through decreased access to and utilization of healthcare. Peng and colleagues (2021) find that increases in the local unemployment rate between 2004 and 2017 is strongly associated with a decrease in self-reported health insurance coverage and an increase in forgoing a doctor's visit due to cost. In their recent study linking increased global trade with adverse mental and behavioral health outcomes, Adda and Fawaz (2020) conclude that "income alone cannot explain this worsening in health," and the authors point to a lack of access to healthcare as a primary driver of the increased mortality and morbidity they observe in recent decades (p. 1530). These findings are not surprising given that many Americans obtain health insurance through their employer, and public health insurance coverage is inaccessible and deficient in many parts of the country. National estimates suggest that the 2007–2009 Great Recession decreased the overall share of adults ages 19–64 with employer-sponsored health insurance by 5.8% and led to a corresponding increase in the uninsured rate of 5.6% (Holahan, 2011). Schaller and Stevens (2015) highlight this potential mechanism using survey data at the individual level, where the authors document significant decreases in the probability of having any insurance—particularly access to private health insurance—among individuals having recently experienced involuntary job loss.

Moreover, macroeconomic conditions are likely to affect population health in ways that are not the direct result of individual-level economic or financial circumstances. There is extensive evidence that the externalities of macroeconomic decline extend beyond those experiencing acute economic distress to adversely affect the health of families and communities more broadly (Brand, 2015). One potential pathway could be observing acute job loss events (e.g., company or plant closures) in one's community or place of employment, which may directly contribute to increased feelings of job precarity and psychological distress. Indeed, there is a growing body of research documenting poor psychological outcomes even among workers who remain employed during economic downturns (Elser et al., 2019; Modrek et al.,

2015). Economic downturns are also associated with austerity measures that may decrease access to public health services and the social safety net, possibly negatively affecting various health outcomes at the population-level (Modrek et al., 2013).

Finally, the relationship between macroeconomic conditions and health outcomes has an important temporal dimension that must also be considered. To date, most aggregate-level studies on DSA or related outcomes have focused on contemporaneous effects of macroeconomic changes (or one-year lagged effects), where the behavioral and biological mechanisms that underlie this relationship are assumed to occur instantaneously. There are important exceptions to this, including much of the aforementioned work by Ruhm that traces the dynamics of specific health outcomes (including suicide) as a function of exposure to transient versus longer-term declines in employment conditions (Ruhm, 2000, 2003).

It is worth noting that this temporal dimension has been considered more explicitly in the context of individual-level studies. This research has led to a diverse set of findings pointing to heterogeneity in the duration of health impacts following acute employment shocks, with significant effects ranging from years to decades depending on the specific outcomes examined (Browning and Heinesen, 2012; Classen and Dunn, 2012; Schaller and Stevens, 2015). The duration of the shock itself may also play a critical role. In one important review of the literature on the relationship between unemployment and suicide, Milner and colleagues conclude that long-term employment is associated with an increased risk of suicide and suicide attempt (relative to those experiencing shorter unemployment spells), although the risk of suicide among the long-term unemployed is most elevated in the years immediately following job loss (Milner et al., 2013).

However, to my knowledge, few recent studies using aggregate data have explicitly examined the dynamic effects of macroeconomic conditions on DSA-related outcomes in the short- to medium-term.[2] In a recent paper exploiting changes in US trade relations with China, Adda and Fawaz (2020) study the health effects of increased import competition on a variety of self-reported health data and measures drawn from administrative data sources. Their findings suggest that job loss due to increased import competition—which disproportionately affected labor markets characterized by high shares of jobs requiring "routine tasks"—resulted in the deterioration of self-reported mental health and increases in hospitalization visits for suicidal ideation and substance use disorder (Adda and Fawaz, 2020). Moreover, the study provides evidence that the adverse effects of import competition on morbidity and mortality increased for up to six years following the shock but varied substan-

[2]There is also a growing body of literature examining the health effects of economic conditions that unfold over much longer time horizons than are considered in the present analysis. These include recent work by Schwandt and von Wachter (2020), Coile et al. (2014), and Lleras-Muney et al. (2021), each of which uses cohort-based models to study the cumulative effects of adverse economic conditions on adult health at various periods throughout the life cycle. For example, in their current working paper, Schwandt and von Wachter (2020) find that graduating into a recession increases midlife mortality for certain causes of death, including several substance-related "deaths of despair." This echoes earlier work by Maclean showing that men who left school during a recession experienced a higher probability of reporting depressive symptoms at age 40 relative to similarly aged men who did not (Maclean, 2013). Interestingly, women display the opposite pattern.

tially across different "despair-related" outcomes. Documenting these unique and potentially dynamic effects of economic conditions on each despair-related mortality outcome remains largely unexplored in the current literature.

Therefore, despite the focus of the "despair" hypothesis on long-term effects of decreasing economic opportunity, relatively little research has explicitly examined the dynamics of DSA mortality, particularly in response to short-term aggregate employment shocks. Developing a better understanding of the effects of short- to medium-run fluctuations in employment conditions on these health outcomes may help policymakers better prepare for the adverse health effects of aggregate economic downturns that emerge over relatively short time horizons, such as the widespread job loss and employment uncertainty induced by the COVID-19 pandemic.

2.3 Methods

2.3.1 Data

Primary outcome data for this study come from restricted-access Multiple Cause of Death files from the National Center for Health Statistics (NCHS) over the 2003–2017 period. To isolate the effects of employment changes on the adult working-age population and align my mortality sample with the data on county-level employment counts, I restrict my analysis to deaths among adults ages 19–64. As my primary health outcomes, I define four mutually exclusive causes of death using ICD-10 diagnosis codes listed as the underlying cause of death: drug non-suicide (ICD-10 codes Y10–Y14, X40–X44), non-drug suicide (X66–X84, Y870), drug suicide (X40–X64), and alcohol-related causes (X45, X65, Y15, K70, K73–K74). For most analyses, I aggregate mortality counts to the county-year cell to generate cause-specific mortality rates per 100,000 population. Due to the relatively large number of county-year cells with zero cause-specific deaths, I opt not to use the common approach in the literature of modeling the inverse hyperbolic sine transformation (IHS) of the count of deaths in each cell.[3] For subgroup analyses, I further define cells by sex and two age groups (19–44, 45–64). All population denominators are based on intercensal estimates drawn from the Surveillance, Epidemiology, and End Results (SEER) database. Due to coding inconsistencies in other decedent characteristics and challenges in constructing reliable population denominators at the county level, I do not stratify mortality rates by other key demographic characteristics such as educational attainment and race or ethnicity. In Section 2.6 and Chapter 4, I dis-

[3]I use the threshold of 1/3 cells with zeros to guide my decision to use rates rather than IHS counts as the dependent variable in most models (Bellemare and Wichman, 2020). In my pooled sample, only one outcome exceeds this threshold (62% of county-year cells have zero drug suicides), while nearly all outcomes exceed this threshold when stratified by sex and age group. As I describe in more detail in Section 2.5.1 , estimating IHS count models with the aggregate sample yields qualitatively different conclusions for certain outcomes along with more precise point estimates and a stronger first stage F statistic (see Appendix Table A.12, Panel D).

cuss the importance of considering heterogeneous effects across these dimensions in future research.

My primary indicator of local labor market conditions is the employment-population ratio (EPOP) defined at the county-year level. EPOP is constructed as the ratio of the employed population relative to the total working-age population, which I define in this study as all noninstitutionalized adults ages 19–64. I prefer to use EPOP rather than the county-level unemployment rate because EPOP is not sensitive to changes in the labor force participation rate—which changed substantially among certain populations during this time period and may be correlated with unobserved drivers of mortality—and because unemployment rates from household surveys are unreliable at the county level, thereby increasing the likelihood of bias due to measurement error (Lindo, 2015). I construct county-year EPOP by aggregating county-industry-quarter employment counts from the Quarterly Workforce Indicators (QWI), a linked employee-employer database which draws on various data sources, including the Quarterly Census of Employment and Wages and UI benefits databases.[4] For the disaggregated analysis, I construct equivalent measures of EPOP among males and females ages 19–44 and 45–64.

In addition to these measures, I construct the following variables using the SEER population data to account for changes in the demographic composition of the working-age adult population at the county level: share female, share ages 19–24, 24–34, 35–44, 45–54, and 55–64, and share Hispanic, non-Hispanic Black, and other non-Hispanic non-White. Finally, my analytic data set also includes county-by-year shares of adults ages 25 years or older with some college education or an associate's degree.[5] Based on evidence that local home values may drive changes in opioid overdoses more than do employment conditions (Brown and Wehby, 2019), I include a county-level index of annual housing prices developed by the Federal Housing Finance Agency (Bogin et al., 2019). However, because this measure is missing for nearly one quarter of all county-year observations during the 2003–2017 period, I include this measure for sensitivity analyses only.[6]

[4]For this analysis, I use NAICS industries defined at the two-digit level. Collapsing industries into two-digit SIC industries does not meaningfully change my results (results not shown). Following Currie, Jin, and Schnell (2019), I drop all county-quarter-industry employment counts that are flagged as missing, distorted, or suppressed.

[5]Measures of county educational attainment for 2003–2008 come from the 2000 Census and from the 5-year American Community Survey (downloaded from the AHRQ Social Determinants of Health Database) for 2009–2017. This educational attainment measure is imperfect as each county-year observation before 2009 takes on its 2000 Decennial Census value and is therefore time invariant during this period. When estimating the model for the 2010–2017 period separately (when county educational attainment is time varying for all years), results are unchanged when excluding county-level educational attainment as a control. This provides some reassurance that the lack of variation in educational attainment measure in the early years of my main sample may have a negligible impact on my estimates.

[6]Some analyses of local employment conditions on deaths of despair include controls for additional economic characteristics that may mediate the relationship between employment rates and mortality (e.g., share uninsured, share living in poverty, median household income). I do not control for these county-level economic covariates to avoid potential overadjustment bias. However, I do include these variables in regressions examining the correlates of initial industry shares, as described in Section 2.3.3.

2.3.2 Empirical approach

My analysis follows a generalized difference-in-differences framework to estimate the effect of contemporaneous and lagged changes in employment rates on the causes of death described above. I follow a common approach in the literature by estimating a series of two-way fixed effects models that leverage within-county variation in employment rates over the 2003–2017 period while controlling for various sets of geographic controls.

Contemporaneous model

The baseline estimating equation for this analysis is Equation 1, where Y_{ct} is the cause-specific mortality rate per 100,000 population in county c in year t, $EPOP_{ct}$ is the employment-population ratio in county c in year t, θ_t are year fixed effects, and θ_c are county fixed effects:

$$Y_{ct} = \theta_c + \theta_t + \beta_1 EPOP_{ct} + \gamma \mathbf{X}_{ct} + \varepsilon_{ct} \tag{1}$$

I compare estimates across two main model specifications with different sets of geographic controls in addition to the set of county-level demographic covariates mentioned above ($\mathbf{X}_{ct}$): a model with county and year fixed effects only, and a model with county, year, and state-by-year fixed effects. I prefer this latter specification in order to account for time-varying, state-level policy changes that may be associated with mortality and correlated with labor market conditions. For example, recent research by Dow et al. (2020) shows that state-level changes in the Earned Income Tax Credit and the minimum wage, both labor market policies that directly affect employment, also reduce mortality due to non-drug suicides. By including state-by-year fixed effects, these models also control for other state-level policies shown to affect opioid use, such as prescription drug monitoring programs with mandated reporting and medical or recreational marijuana legalization (Bradford et al., 2018; Buchmueller and Carey, 2018) which may be correlated with the unobservable error term in Equation 1.

In addition to these two models, I follow Hollingsworth et al. (2017) and estimate a set of models with county linear time trends, although I do not view these as my preferred specifications because certain counties (particularly those with very small populations) demonstrate highly non-linear mortality trends. Finally, to allow for the possibility that cause-specific mortality rate is not immediately responsive to the current year's employment rate, I follow a common approach in the literature and estimate the identical series of models by replacing the current year's employment rate ($EPOP_{ct}$) with that from the previous year ($EPOP_{(ct-1)}$). These differ from the distributed lag models with one lag year (described in more detail below) in that they do not control for the employment rate in the contemporaneous year. As described below, I find the contemporaneous and one-year lag models to be nearly identical and refer to the former throughout the text as the primary model.

Partial and fully disaggregated models

I estimate the model above with pooled data aggregated at the county-year level (hereafter referred to as the aggregated model) as well as with data disaggregated by four age-by-sex subgroups: males, ages 19–44; females, ages 19–44; males, ages 45–64; and females, 44–64. In these models, the unit of analysis is the county-year-demographic group cell. Specifically, I estimate two disaggregated models, one in which the employment rate does not vary across demographic groups within county-years but is theorized to have heterogeneous effects on mortality across different age-by-sex subgroups. This "partially disaggregated" model (Equation 1a) is presented below, where the subscript i denotes one of four age-by-sex demographic groups. The second model (Equation 1b) replaces the aggregate independent variable $EPOP_{ct}$ with the group-specific employment rate for group i (to estimate own-group effects) or the group-specific employment rate for group j ($j \neq i$), where j denotes one of the other three demographic groups' employment rates. In the instrumental variables analysis described in the next section, I replace each of these aggregate or disaggregated employment variables with its corresponding aggregate or disaggregated predicted employment rate constructed using the shift-share instrument.

$$Y_{ict} = \theta_c + \theta_t + \beta_1 EPOP_{ct} + \gamma \mathbf{X}_{ct} + \varepsilon_{ict} \tag{1a}$$

$$Y_{ict} = \theta_c + \theta_t + \beta_1 EPOP_{ict} + \gamma \mathbf{X}_{ct} + \varepsilon_{ict} \tag{1b}$$

Under the assumption of strict exogeneity in the equations above, the coefficient β_1 can be interpreted as the effect of a one percentage point increase in EPOP (often referred to throughout the chapter as "employment" or "employment rate") on the contemporaneous mortality rate.

However, if health outcomes are not immediately responsive to changes in the employment rate and develop over prolonged exposure to poor economic conditions, this model is unable to distinguish between this lagged response and an instantaneous effect. This may be a particular concern in the context of some mortality outcomes that develop over relatively long periods of time, such as alcoholic liver disease. For example, previous studies show that acute employment-related shocks have large effects on suicide and other mental health outcomes within one to five years following acute employment shocks, while the risk of mortality due to alcohol-related diseases increases less dramatically in the short term but remains elevated 11–15 years after job loss occurs (Browning and Heinesen, 2012).[7] Moreover, because employment rates are highly correlated across years, the contemporaneous effect may reflect changes in employment conditions from prior years, rather than in the year in which the death occurred (Ruhm, 2000). I turn now to describe two additional analytic strategies that will allow for this adjustment period and distinguish between potential instantaneous and lagged effects.

[7] Classen and Dunn (2012) make an important distinction between suicide risk as a function of job loss itself versus as a function of unemployment duration. Using mortality data at monthly intervals, the authors find that prolonged periods of unemployment (15–26 weeks for men and >5 weeks for women) are more predictive of suicide risk than in the period immediately following job loss.

Long difference model

I begin by estimating a series of stacked first difference models over various time horizons, beginning with a one-year difference model and increasing the duration up to six years. Given the evidence presented above and the suggestion that DSA mortality may reflect more "a long-term process of decline" (Case and Deaton, 2017, p. 429) as opposed to short-term shocks, it is reasonable to expect that differencing over longer time horizons will lead to larger magnitude effects. This overall approach is similar to that taken in several existing papers examining the effects of increased import competition from China on premature mortality (Adda and Fawaz, 2020; Autor et al., 2019), although these studies examine changes over substantially longer periods than the present analysis. Equation 2 presents the main estimating equation for these long difference models, where Δ_r denotes the differencing operator across periods of duration r (e.g., $\Delta_3 Y_c = Y_{c(2006)} - Y_{c(2003)}$) for $r = 3$, and θ_p denotes period fixed effects to control for unobserved period-specific shocks.[8]

$$\Delta_r Y_c = \theta_p + \beta_1 \Delta_r EPOP_{ct} + \gamma \Delta_r \mathbf{X}_{ct} + \Delta_r \varepsilon_{ct} \tag{2}$$

Estimating the model in differences introduces a number of important analytic decisions driven by both econometric and theoretical considerations (Baker et al., 1999; Meer and West, 2015). First, the long difference estimator requires the effect of an employment change on mortality to be uniform across the duration of an interval—an assumption that may or may not (as in the case of suicide risk, described above) be reasonable. Moreover, in periods of high-frequency variation such as the time around the Great Recession, taking longer differences will result in less variation to identify causal effects (e.g., the 2012–2009 difference in $EPOP$ is minimal despite substantial variation in the intervening years). To address these concerns, I estimate models across various time horizons ranging from one to six years and with intervals beginning at different start and end points in order to capture different periods of variation during the 2003–2017 period. However, because I require the differenced intervals to be of equivalent length, the number of periods and years included in the sample are a function of the period duration, which results in some years being mechanically dropped from the sample. As I discuss more in Section 2.3.4 and show empirically in Section 2.5, omitting years at the beginning or end of the sample period has noticeable effects on the estimated effects.

Distributed lag model

While the model above provides a crude assessment of changes in mortality as a function of changes in employment rates, it is unable to distinguish instantaneous effects from those that unfold dynamically or accumulate within the differenced interval. To explicitly model this

[8] This equation differs slightly from Equation 4 in Autor, Dorn and Hanson (2019) with respect to the controls included in the model (e.g., I exclude area time trends and include time-varying county-level characteristics in $\mathbf{X}$ rather than start-of-period values) and I estimate models with periods of equal duration only.

adjustment period while addressing the drawbacks of the contemporaneous model, I next estimate a series of distributed lag models, which control for employment conditions from prior periods. The distributed lag equations take the following general form, where $r < 0$ and $r > 0$ denote the number of yearly lead and lag terms relative to the year t, respectively:

$$Y_{ct} = \theta_c + \theta_t + \sum_{r=-1}^{r=4} \beta_r EPOP_{c(t-r)} + \gamma \mathbf{X}_{ct} + \varepsilon_{ct} \tag{3}$$

Due to the relatively short panel, I limit the number of lags in my analysis to four years, although as noted above, it is possible that longer adjustment periods may be required to observe the full adjustment period for certain mortality outcomes in response to economic shocks. However, a previous analysis by Ruhm (2000) using annual mortality data and four lag terms suggests that increases in state unemployment rates result in more substantial contemporaneous or short-term changes in cause-specific mortality rates than in the long term. Specifically, his analysis finds that suicide mortality increases significantly in the contemporaneous year but varies little in subsequent years, while liver disease mortality is not associated at conventional levels of significance with contemporaneous or lagged changes in the state unemployment rate.[9]

On the other hand, in areas highly affected by import competition with China, Adda and Fawaz (2020) estimate a 6% increase in all-cause mortality seven years after an import shock and a seemingly monotonic increase in hospital admissions for opioid use disorder for up to five years. One key tradeoff to including more lag years is a mechanical decrease in the overall sample size (thereby reducing precision); doing so also places more weight on data from later years in the panel. In Section 2.5.3 I show that the two-way fixed effect estimates are particularly sensitive to changes in the start and end years of the panel, suggesting that a shorter number of lag terms is preferable.

2.3.3 Instrumental variables approach

Estimation of β in each of the equations above using ordinary least squares (OLS) would likely suffer from bias due to omitted variables. Because $EPOP_{ct}$ reflects an equilibrium employment level that is determined simultaneously by the supply and demand of labor, the estimate of β above will not capture the intended employment effect if labor supply is correlated with location-specific factors that might drive mortality outcomes. For example, several recent studies point out that areas with high rates of prescription opioid per capita

[9]This lag period is also appropriate given the elevated risk of suicide for up to four years following job displacement in Denmark, as shown by Bowning and Heinesen (2012). Interestingly, follow-up work by Ruhm (2003; 2005) examining the dynamics of non-fatal health conditions and self-reported health behaviors (e.g., smoking, physical exercise) in response to lagged changes in the employment rate does show evidence of differential immediate versus longer-term effects. Using quarterly data on drinking behavior, Ruhm and Black (2002) also show that the unemployment rate is associated with a short-term decrease in alcohol consumption that is insignificant after one year, suggesting that yearly data may conceal within-year dynamics.

or illicit opioid use experience lower labor force participation and employment rates (Harris et al., 2019; Park and Powell, 2021).[10] To address this potential endogeneity issue, I re-estimate each of the equations above using a shift-share instrumental variables (IV) approach intended to isolate the plausibly exogenous demand-side variation in local-level employment. This IV strategy is becoming increasingly common in studies that seek to estimate causal effects of changes in macroeconomic conditions on various health outcomes, including deaths of despair (Autor et al., 2019; Betz and Jones, 2018; Charles et al., 2019; Currie et al., 2019; Pierce and Schott, 2020).

To implement this approach, I most closely follow the strategy of Currie, Jin and Schnell (2019) by instrumenting actual employment rates at the county level with a predicted level of employment based on a weighted average of industry-specific growth rates at the national level.[11]. Specifically, I construct my instrument by interacting the share of each county's initial employment in the base year (2002) with national industry-specific growth rates relative to the base year, and sum over all two-digit NAICS industries j.[12] I define the instrumental variable Z_{ct} as follows, where the subscripts c, t, and j index county, calendar year, and two-digit NAICS industry codes, respectively:

$$Z_{ct} = \sum_{j} \left(emp_{jc(2002)} \times \frac{\sum_{c' \in \{C \setminus c\}} emp_{jct}}{\sum_{c' \in \{C \setminus c\}} emp_{jc(2002)}} \right) \tag{4}$$

In the formula above, emp_{jct} is the county-level employment count for industry j in time t. The right-hand side of the product can be interpreted as the national growth rate in employment in industry j in year t relative to that in 2002, which I calculate by excluding county c's own employment. This quantity is weighted by the employment count in industry j in county c in 2002.[13] Because Equation 4 results in an employment count, I rescale Z_{ct} in the first stage of the two-stage estimating system by dividing the instrument by the working-

[10]Chapter 3 includes a more detailed summary of the recent literature linking prescription opioids to employment.

[11]Lindo et al. (2018) construct a similar measure of predicted employment in their study of labor market conditions on child maltreatment rates across California by interacting the base-year share of each industry's total employment attributable to each county with the state-level employment in each industry. This differs somewhat from the current construction described below in Equation 4 but is similar in that the instrument is an employment rate in *levels* (which is later converted into a ratio), rather than an index of year-on-year or multiple year employment growth as is most commonly used in the literature (Broxterman and Larson, 2020)

[12]My preferred shift-share construction uses employment data from 2002 as the base year. However, county-level QWI data are not available for Arizona until 2004(Q2), Mississippi and New Hampshire until 2003(Q1), and Massachusetts until 2010(Q2). I use the first quarter of available data to define the base year for each of these states. Estimates are robust to dropping these states from my analysis entirely (not shown). Due to large numbers of missing and suppressed employment counts in Washington, DC, I do not include Washington, DC in any sample.

[13]Redefining the instrument as $Z_{ct} = \sum_j \left(\frac{emp_{jc(2002)}}{emp_{c(2002)}} \times \frac{\sum_{c' \in \{C \setminus c\}} emp_{jct}}{\sum_{c' \in \{C \setminus c\}} emp_{jc(2002)}} \right)$, which is the construction used in Musse (2020), does not meaningful change the main results (results not shown).

age population in each county in 2010 and multiply by 100 for ease of interpretation.[14] I take the natural log of this quantity due to a long right-tail in the distribution of predicted employment rates (see Section 4.1, below). Thus, the two-stage system for estimating Equation 1 is as follows:

$$EPOP_{ct} = \alpha_1 + \beta_1 log\Big(\frac{Z_{ct}}{pop_{c(2010)}}\Big) + \theta_c + \theta_t + \gamma_1 \mathbf{X}_{ct} + \omega_{ct} \tag{5a}$$

$$Y_{ct} = \alpha_2 + \beta_2 \widehat{EPOP}_{ct} + \theta_c + \theta_t + \gamma_2 \mathbf{X}_{ct} + \nu_{ct} \tag{5b}$$

I use the user-written Stata (StataCorp., 2021) commands *reghdfe* and *ivreghdfe* for the ordinary least squares and two-stage least squares estimation procedures, respectively (Baum et al., 2010; Correia, 2014). The parameter of interest is β_2, which, conditional on Z_{ct} being a valid instrument, will yield a causal estimate of the effect of a one percentage point increase in EPOP on the rate of cause-specific mortality. I similarly estimate instrumental variables versions for the long difference (Equation 2) and distributed lag (Equation 3) models by taking the period difference in the log of the predicted employment rates and yearly lags of the log of the predicted employment rates, respectively.

Instrument validity

The instrument described above generates a prediction of county-level employment as if the change in county-level, industry-specific employment in a given year were the same as the nation-wide, industry-specific employment in that same year (Currie et al., 2019). In this sense it is more natural to consider a "differential exposure design" where the identifying variation in the instrument comes from heterogeneity in the industry mix across counties in the base year (Goldsmith-Pinkham et al., 2020) rather than from heterogeneous growth rates (Borusyak et al., 2020). Following this approach, I argue that the validity of the instrument Z_{ct} relies on exogeneity of the initial industry shares conditional on the model controls, where the composition of initial industry shares must be orthogonal to unobservable factors driving county-level mortality.

While the strict exogeneity of the industry shares is ultimately an untestable assumption, the plausibility of this exclusion restriction can be explored empirically in several ways (Goldsmith-Pinkham et al., 2020). First, I use variation in the instrument (predicted EPOP) in the first year of the panel (2003) and the top five industry shares at baseline (2002) to explore correlations with changes in county-level demographic and socioeconomic variables. To the extent that there exist unmeasured correlates of county-specific, time-varying characteristics that may be associated with mortality, the presence of a statistically significant relationship between changes in these characteristics and the instrument or baseline shares

[14] I get virtually identical results using the average of the county population over the entire study period as the denominator rather than the 2010 county population. However, using the yearly county population to define the instrument results in noisier estimates and a much weaker first stage, and as such I opt to use the 2010 county population as the denominator for consistency with the instrument used in Currie et al. (2019).

may be suggestive of a violation of the exclusion restriction.[15] In addition to using changes in the demographic compositional variables I include in my main regressions, I also include changes in the share of adults living in poverty and median household income downloaded from the US Census Bureau's Small Area Income and Poverty Estimates Program (SAIPE).

Results from this exercise are presented in Appendix Table A.1 and indicate that the shift-share instrument is highly correlated with nearly all changes in county-level characteristics included in the model (Column 1). The only exception is the share of the working-age population aged 45–54. The overall R^2 for the regression is presented in the final row of the table and indicates that baseline characteristics explain only 7% of the variation in the predicted employment in the base year. Columns 2–6 show that the changes in observable characteristics are also highly correlated with each of the top five industry shares at baseline. Results from this table highlight the importance of controlling for these time-varying demographic characteristics in my regressions. At the same time, the high degree of correlation between the instrument and initial employment shares with changes in baseline characteristics raises the possibility that there are likely unobserved location-specific factors that may be correlated with mortality changes. I return to this possibility and its implications for my IV results in Section 2.6.

A second test examines the extent to which the instrument may be correlated with unobservable factors that would undermine the parallel pretrends assumption implicit in this research design. Visualizing the degree to which this assumption is met using event study plots has become common in studies of the effects of discrete exposures or policy changes, but the fact that the shift-share instrument takes on a new value in each year for each unit means there is no unexposed "pre-period" during which to visualize the presence (or lack of) parallel pretrends. Nonetheless, several techniques have been proposed to probe the validity of this assumption (Borusyak et al., 2020; Goldsmith-Pinkham et al., 2020), including a simple regression-based approach to examine whether future values of the instrument predict previous values of the outcome.

In Appendix Table A.2, I present results from adjusted and unadjusted regressions of the 1999–2002 "pre-period" difference in each cause-specific mortality rate on the value of the instrument in 2003. There is evidence in the unadjusted model that increases in drug non-suicide between 1999 and 2002 predict lower values of the shift-share instrument, but this relationship disappears with the inclusion of 2002 county-level covariates. There is no evidence of a relationship between the instrument and 1999–2002 mortality trends for any

[15]Given that the identifying variation in the shift-share instrument comes from variation in the industry mix at baseline, Goldsmith et al. (2020) recommend identifying the most influential industries based on Rotemberg weights and regressing these shares on changes in baseline characteristics in addition to separately examining the correlates of the instrument. In the present analysis with only 20 2-digit industries (as opposed to 228 3-digit industries used in that paper), it is feasible to examine all industries rather than focusing on those that drive the most variation in the instrument. In doing so (results not shown), I find that nearly all 2-digit NAICS industry shares are highly correlated with changes in the baseline characteristics presented in Appendix Table A.1. For brevity, I only present the regression output from the models with predicted employment and the largest 5 industries based on the overall share of all jobs in 2002.

other cause of death. While interpretation of this test is confounded by the possibility that values of the instrument itself may be endogenous to mortality changes from previous periods, this exercise does provide some assurance that values of the instrument are not strongly correlated with existing trends in mortality conditional on the controls in the model. Such a relationship would further violate the exclusion restriction and undermine the use of the shift-share instrument to uncover unbiased estimates of the effect of employment changes on mortality.

2.3.4 Time periods of analysis

The choice to begin my analysis period in 2003 was driven by availability of the QWI data, which do not exist or is missing for a relatively large share of states until 2002. To maximize the duration of the panel, I included data until 2017—the final year in which restricted-access mortality data were available. Previous econometric studies examining macro-level determinants of mortality over time—particularly those focusing on drug-related causes—report that conclusions are often sensitive to the beginning and end points of the sample period (Ruhm, 2015, 2019; Shover et al., 2019). A recent analysis by Peng et al. (2021) examines this phenomenon in the context of alcohol consumption, where the authors find a strong procyclical variation in self-reported binge drinking from 2004–2010 that attenuates substantially when the sample period is extended to 2014 and 2017.

While I am unable to extend the period of analysis due to data limitations, I conduct a similar series of exercises by estimating the primary contemporaneous model (the IV version of Equation 1) across different windows within the 2003–2017 period. Following the approach implemented by Ruhm (2015), I sequentially re-estimate my preferred model over shorter panel periods by increasing the start or end year of the panel, where the shortest panel period analyzed consists of five years (i.e., 2013–2017 and 2003–2007, respectively). In addition, I estimate yearly fixed effects models over six models of equivalent 10-year-long intervals beginning with the 2003–2012 period and ending with the 2008–2017 period. A change in the point estimates across different period intervals could reflect changes in the underlying relationship between employment conditions and cause-specific mortality (Ruhm, 2015) or temporal shifts in confounding factors that are not captured by these models (Ruhm, 2019; Shover et al., 2019). I describe the results from this series of sensitivity exercises and its implications in more detail in Section 2.5.3.

2.3.5 Adjusting for multiple hypothesis testing in contemporaneous models

Because I examine four related mortality outcomes (alcohol, drug non-suicide, non-drug suicide, and drug suicide) across pooled, partially, and fully disaggregated samples, it is necessary to consider the possibility of over-rejecting the null hypothesis due to multiple testing. Following related work by Peng et al. (2021), I estimate adjusted p-values for all models after controlling for the familywise error rate, defined as the probability of falsely

rejecting the null hypothesis when it is true, or making a Type I error (Anderson, 2008). Specifically, I perform a free step-down resampling procedure that calculates Westfall-Young adjusted p-values for each hypothesis test using the Stata command *wyoung* with 10,000 (or 1,000 in the case of the fully disaggregated model) replications (Jones et al., 2019). For each series of tests, I define the "family" as the set of all four outcomes, which represents the most conservative approach in this setting.

2.4 Results

2.4.1 Descriptive statistics

Table 2.1 presents mortality rates for a combined measure of all DSA mortality as well as the four primary causes of death for the entire study population and stratified by age (19–44 and 45–64) and sex. Consistent with existing research, overall rates of DSA are higher among males than females, and rates for most causes of death are higher among older adults. Older males have over four times the overall rate of DSA (85.5 per 100,000) relative to 19–44-year-old females, who have the lowest rates (19.9 per 100,000). Among the three major categories of DSA mortality, accidental drug overdoses (drug non-suicide) constitute the largest share of deaths among women and younger men, while alcohol makes up the largest share of deaths among older men (alcohol-related mortality rates are also high among older females). Alcohol-related mortality displays the largest age differential, with rates among older women and men over six and seven times higher than their younger peers of the same sex, respectively. Suicide rates are over three times higher among men than women across both age categories, and non-drug suicides constitute the majority of overall suicides across all demographic groups, especially among men.

Level differences in cause-specific mortality between men and women are immediately evident in Figure 2.1, which displays trends in cause-specific mortality rates (left vertical axis) over time across these four key demographic subgroups. All demographic subgroups experienced substantial growth in drug non-suicides throughout the 2003–2017 period, with especially steep increases beginning for men and younger women around 2011. In contrast, 45–64-year-old women experienced a relatively constant increase in the rate of drug non-suicides along with mortality due to alcohol-related causes. Rates of non-drug suicides increased for all subgroups (albeit only slightly for women), while rates of drug suicides remained relatively constant throughout the period for all groups.

Turning to employment conditions during 2003–2017, the mean aggregate employment-population ratio (EPOP) among the primary sample of county-years was 0.59 with a standard deviation of 0.22. As shown on the left-hand side of Figure 2.2, the distribution of aggregate employment rates has a long right tail, which is the result of a small number of counties in the QWI with implausibly large employment rates for all or most data years (e.g., New York County, or Manhattan). As a sensitivity test to these potential outliers, I winsorize the largest and smallest 0.5% of observations in the employment distribution (shown on

the right-hand side of Figure 2.2) and re-estimate my main models. Because these counties have relatively small populations and I weight all regressions by cell-level population due to the large variation in county population size, I find this does not affect my main regression estimates although it does improve the strength of the first stage in the instrumental variables models (see Appendix Table A.12, Panel C). As shown in Figure 2.3, the distribution of the predicted employment rates is similarly right-skewed.

Figure 2.1 also presents population-weighted trends in county-level EPOP by subgroup over the 2003–2017 period (right vertical axis). Mirroring the well-documented differences in the Great Recession's impact on employment across demographic groups (Cunningham, 2018), it is immediately evident that younger males experienced the largest absolute decrease in EPOP while older women experienced the least dramatic decrease from peak to trough. Moreover, the EPOP for both groups of females and older males returned or surpassed their pre-Recession levels by the end of the study period while EPOP among younger men continued to lag. In Section 2.4.3 I return to these group-specific employment trends to explore how these differences may contribute to differential trends in DSA mortality across demographic groups.

To highlight the importance of examining effects at a sub-state level, Figure 2.4 presents a map of US counties shaded by quartiles of each county's percentage point change in county-level EPOP between 2010 and 2017. I select this post-Recession period for this exercise because it was marked by substantial heterogeneity in economic recovery at the local level (Yagan, 2019), and it is plausible that areas with less resilient labor markets may be associated with increased despair and possibly higher rates of mortality (Monnat, 2019). The map displays noticeable within-state variation in economic recovery, with most states containing some counties with strong economic growth and others experiencing stagnant or even decreasing employment rates during this period.

Finally, as motivation for the subsequent regression analysis, I present two additional sets of descriptive figures relating employment conditions to DSA mortality. Figure 2.5 overlays trends in population-weighted averages of county-specific employment with average rates of all DSA (per 100,000) during the study period. The purpose of this exercise is simply to demonstrate that there is no obvious increase in DSA mortality coinciding with the dramatic decrease in the employment rate during the 2007–2009 Great Recession, nor is there evidence of any deceleration in the mortality rate increase as economic conditions improve at the national level in the post-Recession period. In their 2020 book on the origins of the "deaths of despair" crisis, Case and Deaton construct a similar figure and conclude that "it is not possible to explain deaths of despair in terms of America's exceptional poverty or the Great Recession" (Case and Deaton, 2020a, p. 135).

However, these aggregate trends may conceal important underlying causal relationships between employment conditions at the local level and specific mortality outcomes, as suggested by the set of binned scatter plots in Figure 2.6. These figures present the crude "long difference" relationship between changes in county-level EPOP and changes in four cause-specific mortality rates of interest over the same 2010–2017 period described above. While these figures should not be interpreted as evidence of causal effects, the strong negative cor-

relation between alcohol-related mortality and both non-drug and drug suicides points to the possibility that employment growth may be protective against rising mortality rates due to these causes while having a little to slightly positive impact on drug-related causes.[16]

2.4.2 Contemporaneous effects of employment on mortality: aggregate employment shocks

Table 2.2 presents the ordinary least squares (OLS) and instrumental variables (IV) estimates from the baseline set of regressions of cause-specific mortality rates on the contemporaneous employment rate.[17] With the exception of a small negative effect of EPOP on non-drug and drug-related suicide in the OLS model with county-level demographic controls only (column 1a), there is no evidence that changes in county-level EPOP affects the mortality rate for any cause-specific outcome across either of the other two OLS specifications (columns 2a and 3a). In contrast, estimates from the three IV models (columns 1b, 2b, and 3b) indicate consistent statistically significant reductions in non-drug suicide in response to employment increases, with substantially larger effects in the fixed effects models (columns 2b and 3b).

Coefficient estimates from the preferred model with county, year, and state-by-year fixed effects (rightmost column) suggest that a one percentage point increase in EPOP decreases the rate of suicides not involving drugs by 0.16 per 100,000 (95% CI: -0.27, -0.05) on average, or a 1.15% reduction from the mean rate of 14 per 100,000. This model, which will be the main specification presented in subsequent tables unless noted otherwise, estimates these effects to be significant at the 1% level and yields a strong first stage relationship as indicated by a large Kleibergen-Paap F statistic (169.7).[18] Appendix Table A.5, which presents estimates from the identical set of regressions using the one-year lag of EPOP (and predicted EPOP, in the IV models) yields a slightly larger effect of -0.179 per 100,000 (95% CI: -0.29, -0.07).

Meanwhile, the opposite pattern emerges with respect to drug non-suicide mortality, with the fixed effects models yielding large and statistically significant positive coefficients that are consistent with a procyclical relationship. My preferred estimates suggest that drug non-suicide increases by 0.28 per 100,000 (95% CI: 0.08, 0.48) in response to a one percentage point increase in EPOP, or approximately a 1.5% increase relative to the mean of 18.6 per

[16] I perform a similar descriptive analysis using 2007–2010 changes in EPOP and cause-specific mortality, which I present in Appendix Figure A.1. Unlike the 2010–2017 period, mortality rates demonstrate a slight procyclical relationship with EPOP (with the exception of drug suicide, which is slightly countercyclical), pointing to potentially heterogeneous effects at different parts of the business cycle.

[17] For convenience in interpretation, I multiply the EPOP and shift-share instrument by 100 in all regressions. The estimated coefficients in the model can therefore be interpreted as the marginal effect of a one percentage point increase in the share employed, holding all else constant.

[18] Appendix Figure A.2 presents a graphical depiction of the first stage relationship between actual employment rate and the log of the predicted employment rate generated from the shift-share instrument. The figure presents the residuals and line of best fit from the partial regression of the instrument on the actual employment rate, controlling for county, year, and state-by-year fixed effects. Appendix Tables A.3 and A.4 present estimated coefficients from the preferred first stage and reduced form regressions, respectively.

100,000.[19] I find no evidence of an effect of employment changes on drug suicide or alcohol-related outcomes in this preferred model. Estimates from the model using the one-year lag of employment conditions (Appendix Table A.5) are similar to those using current-year employment, although the estimated effect on drug non-suicide is slightly attenuated.

To examine potential heterogeneity in the effects of aggregate employment conditions on mortality, Table 2.3 presents point estimates from the preferred OLS and IV models of the effect of aggregate EPOP on each of the four demographic group-specific mortality rates (the partially disaggregated model). As is the case in the pooled mortality sample, there are no statistically significant effects of employment on any mortality outcome in the OLS models. In contrast, the IV models highlight several demographic group-specific effects that were obscured in the pooled analysis described above. In particular, the negative effect of employment increases on non-drug suicide appears to be entirely driven by decreases among older males (ages 45–64), with the group-specific point estimate suggesting that a one percentage point increase in EPOP leads to a 0.36 per 100,000 (95% CI: -0.66, -0.05) or 1.4% reduction in this outcome relative to the subgroup mean. In contrast, non-drug suicide rates among younger males (ages 19–44) or among females in both age groups do not appear to be responsive to changes in the aggregate employment rate. This stratified analysis further suggests that employment increases may also be protective against drug-related suicides among this older group of males: a percentage point increase in employment is associated with a 0.09 per 100,000 decrease in drug suicides (95% CI: -0.16, -0.02), or an approximate 3.2% decrease relative to the subgroup mean.[20]

Estimates from Table 2.3 also point to heterogeneous effects of aggregate employment shocks on drug non-suicides—an outcome that was positively correlated with increases in the aggregate employment rate. Subgroup analyses suggest that this effect is driven by a large relative increase among younger females (but not older females) and among older males (with only a marginally significant effect among younger males). Specifically, a one percentage point increase in aggregate EPOP increases drug non-suicide among women ages 19–44 by a statistically significant 0.28 per 100,000 (95% CI: 0.08, 0.48), or nearly 2.5% relative to the subgroup mean. The corresponding effect among older men is substantially larger in magnitude but smaller in relative terms, representing a 2.0% increase relative to the sample mean (0.458; 95% CI: 0.10, 0.81).

[19]Because causes of death are reported by coroners or medical professionals, it is possible that a drug overdose was misclassified as a drug suicide when in fact it was an accidental overdose (or vice-versa). Following (2019), I explore sensitivity of these estimates to classifying suicides involving drugs in a larger category of all drug overdoses. The estimated effect of EPOP on all drug-related mortality (0.29; 95% CI: 0.06, 047) is very similar to the measure that excludes intentional drug overdoses (results not shown).

[20]This estimated effect in relative terms is over twice that of non-drug suicides, likely due to the much lower underlying rate of drug suicides during this period (the mean drug-suicide mortality rate for males ages 44–65 is 2.8 per 100,000 compared to a non-drug suicide rate of 24.9). Combining both measures of suicide, the stratified analysis suggests that a one percentage point increase in aggregate EPOP reduces all suicide by 0.45 per 100,000 (95% CI: -0.77, -0.14), or 1.61% reduction relative to the sample mean (full results not shown).

2.4.3 Contemporaneous effects of employment on mortality: fully disaggregated model

As demonstrated above, the changes in the employment rate during the study period were not uniform across demographic groups, raising the possibility that the use of an aggregate employment measure may mask important subgroup effects resulting from differential exposure. I turn now to discuss estimates from the fully disaggregated IV models, where I regress each demographic group-specific mortality rate on their own group's employment rate (denoted "own") as well as models that estimate cross-group effects. Tables 2.4 and 2.5 present these own- and cross-group effects among 19–44- and 45–64-year-olds, respectively.

Unlike in the partially disaggregated model (Table 2.3), where there was no significant effect of aggregate employment fluctuations on non-drug suicide among males ages 19–44, Table 2.4 (Panel A) reveals that non-drug suicide among this demographic group decreases in response to employment increases among their own group. Specifically, a one percentage point increase in EPOP among males 19–44 decreases their own non-drug suicide rates by 0.25 per 100,000 (95% CI: -0.45, -0.05), or by 1.2% relative to the subgroup mean rate. Changes in employment rates across other groups do not appear to affect non-drug suicide among these younger males. However, while rates of mortality due to substances (alcohol and drug non-suicide) among this group were similarly unaffected by changes in the aggregate employment rate, the fully disaggregated model presents evidence of significant cross-group effects. Deaths due to drug non-suicide increase by 0.40 per 100,000 (95% CI: 0.06, 0.74) in response to a one percentage point increase in EPOP among similarly aged females and by 0.21 per 100,000 (95% CI: 0.04, 0.38) among older males. Finally, improvements in employment conditions among younger females are also associated with a 0.11 per 100,000 (95% CI: 0.02, 0.21) increase in alcohol-related mortality among younger males, an effect size that corresponds to a nearly 2.4% increase relative to the sample mean.

Turning to panel B of Table 2.4, drug non-suicide rates among 19–44-year-old women appear to increase in response to employment increases among their own group as well as among their similarly aged male counterparts. Specifically, drug non-suicide increases by 0.21 (95% CI: 0.01, 0.41) and by 0.24 per 100,000 (95% CI: 0.03, 0.44) in response to a percentage point increase in employment among their own group and their similarly aged male counterparts, respectively. Table 2.4 also presents evidence that alcohol mortality increases among younger females by 0.05 per 100,000 (95% CI: 0.00, 0.09) in response to increases in EPOP among older females. This effect, which reflects an increase in alcohol-related mortality of approximately 2% in relative terms, is unique to changes in employment among this subgroup only.

Table 2.5 presents further evidence of cross-group effects on mortality among 45–64-year-old males and females. Recalling that males ages 45–64 were the only demographic group for whom non-drug suicide decreased as a function of aggregate employment, Table 2.5 (Panel A) indicates that this effect was exclusively driven by increases in employment among younger males, rather than among older males themselves. Estimates suggest that a percentage point increase in employment among the younger male population decreases

non-drug suicide among older males by 0.37 per 100,000 (95% CI: -0.60, -0.14), or by approximately 1.61%. This effect is statistically significant at the 1% level. Meanwhile, employment increases among younger females again appear to increase drug non-suicide by 0.50 per 100,000 (95% CI: 0.19, 0.81), or 2.16% relative to the sample mean.

Panel D in Table 2.5 presents estimated own- and cross-group effects for mortality among 45–64-year-old females. Consistent with the lack of significant effects among this group in the partially disaggregated model, there is no evidence that changes in employment among their own group or other groups affect any mortality outcome among this older group of females.

2.4.4 Contextualizing estimated effects relative to mortality trends

Before turning to examine the dynamic effects of employment changes on mortality outcomes, it is important to take stock of the key findings this far in the context of the larger trends in overall and cause-specific DSA mortality during the study period. To better contextualize the contemporaneous effect estimates presented above, I implement a simple series of simulations to visualize the magnitude and timing of the changes in cause-specific mortality predicted by my models against the backdrop of actual mortality trends. For each year in the series, I multiply the estimate from the primary aggregate or disaggregated models by the absolute change in employment (overall or for a given subgroup) relative to 2002. I plot the resulting trend in cause-specific mortality rates, which I then compare to the observed mortality rate during this time. Doing so provides a crude visualization of the "employment-attributable" mortality rates relative to the underlying trends under the assumption that the *only* determinant of mortality in each year is the difference in EPOP between that year and the year 2002. I focus on the two outcomes that appear to be most responsive to contemporaneous increases in employment based on the regression results presented above: non-drug suicide and drug non-suicide.

Both plots in Figure 2.7 demonstrate that while the estimated effects of employment on cause-specific mortality are statistically significant, the employment-attributable mortality rates (dashed lines) constitute a relatively small share of the underlying cause-specific mortality rate (solid lines) during this period. The employment-attributable mortality rates for non-drug suicide (Figure 2.7, panel A) and drug non-suicide (Figure 2.7, panel B) demonstrate the predicted countercyclical and procyclical trends, respectively, but neither closely tracks the actual mortality rates in terms of magnitude or timing. This is especially true for drug non-suicide, where the employment-attributable rate makes up only a small share of the actual cause-specific mortality throughout the entire period. It is clear in Figure 2.7 (panel A) that rates of non-drug suicide began increasing several years *prior* to the Great Recession, a period during which the employment-attributable rate would suggest a sudden increase in suicide mortality; rather, the aggregate rate increases steadily throughout the period with no evidence of a Great Recession-related inflection. Moreover, rates of non-drug

suicide accelerate throughout the final years of the panel—the opposite of what would be expected if this countercyclical relationship lasted throughout the post-Recession economic recovery.

Taken together, these figures suggest that the estimated effects of aggregate employment on non-drug suicide and drug non-suicide do not adequately explain the magnitude or timing of the increases in observed mortality rates. I perform a similar simulation in Figures 2.8 and 2.9, which compare actual to employment-attributable drug non-suicide and non-drug suicide rates, respectively, among selected demographic groups. In Figure 2.8 (panel A), I explore the aggregate procyclical variation in drug non-suicide among younger females, which the results from the series of disaggregated models suggest may be driven by own-group effects and by changes in employment among similarly aged males. Figure 2.8 (panel B) presents drug non-suicide rates among 45–64-year-old males as well as the employment-attributable rates calculated as a function of employment rate changes among younger females only. As was the case above, both figures present little evidence that employment fluctuations—whether measured as the effect of aggregate employment changes (solid line) or group-specific changes (dashed lines)—are qualitatively meaningful drivers of the increase in drug non-suicide mortality during this period.

Finally, Figure 2.9 plots the trends in actual and employment-attributable rates of non-drug suicide among males ages 19–44 (panel A) and 45–64 (panel B), two groups for whom the regression results above suggest that aggregate, own-group, and cross-group employment conditions may play a significant role. Specifically, regression estimates above indicate that non-drug suicide rates for both groups are affected by contemporaneous employment changes among 19–44-year-old males only.[21] An interesting distinction emerges between the two groups when contrasting the actual versus employment-attributable non-drug suicide rates. Unlike the non-drug suicide rates among the aggregate working-age population (Figure 2.7, panel A), where the increase in non-drug suicide predated the Great Recession, rates among younger males (Figure 2.9, panel A) do not begin increasing until 2009—an inflection that occurs one year *following* an increase in the employment-attributable rate induced by the Great Recession. On the other hand, the relationship between actual and employment-attributable rates among older males (Figure 2.9, panel B) more closely resembles the aggregate figure, with increases in mortality clearly predating the Great Recession. For both groups, however, the actual and employment-attributable non-drug suicide rates diverge throughout the recovery period, casting doubt on the hypothesis that employment conditions are the *primary* determinant of the increase in non-drug suicide among males during this period.

[21] Figure 2.9, panel B, also plots the employment-attributable non-drug suicide rate defined using aggregate employment changes, as this measure was significantly associated with decreases in non-drug suicide among older males (see Table 2.3). The aggregate employment-attributable mortality rate is similar to the cross-group rate, although the latter is larger in magnitude.

2.4.5 Estimates from instrumented long difference models

The estimates presented thus far implicitly assume that the effect of an employment shock on mortality materialize within the same data year (or in the following year, in the case of the one-year lag model). I turn now to the estimation of a series of long differences models, an approach better equipped to capture the effect of longer-term trends of employment conditions on health outcomes. Table 2.6 presents estimates from the primary fixed effects model (leftmost column) and series of stacked long difference models with period durations ranging from one-year to six years. Because each of these models begins with 2003 as the first year of the first period, and I require all periods to be of equivalent length, the models vary in the number of periods and therefore overall sample size (i.e., I do not apply "period-equivalent" weights to up-weight shorter periods but rather discard the data years that do not fall within these intervals).[22]

All models in Table 2.6 yield smaller first stage F statistics than the fixed effects model, although they are all comfortably above the threshold of 10. In general, the estimates from the series of long differences models are consistently negative for alcohol, non-drug suicide, and drug suicide, but they are highly volatile, and the models yield few statistically significant findings. The one exception is in the three-year difference model, which suggests that a one percentage point increase in EPOP over the three-year period decreases rates of non-drug suicide by an estimated 0.30 per 100,000 (95% CI: -0.56, -0.03). Meanwhile, the estimates are consistently positive for drug non-suicide, again with the notable exception of the three-year difference model, which yields a statistically significant negative point estimate (-0.28; 95% CI: -0.53, -0.04). That this point estimate suddenly becomes negative and statistically significant in the three-year model (and again in the six-year model) points to the volatility of these estimates. A similar pattern emerges with non-drug suicide, which substantially increases in magnitude between the five-year and six-year models.

2.4.6 Estimates from instrumented distributed lag models

One drawback of the long difference models presented above is that by stacking differences between two years, these models effectively estimate average effects over the various time horizons and ignore potentially important year-on-year changes within periods. To more flexibly estimate the effect of employment conditions over time, I turn next to results from the instrumented distributed lag models, where the lead and lag terms are simply the lead and lag values of the predicted employment generated by the instrument. Appendix Tables A.7–A.10 present the point estimates and standard errors from the distributed lag models for each outcome beginning with one lead term only (column 1) and sequentially adding a contemporaneous term (column 2), and up to four lag terms (columns 3–6). Figure 2.10 presents the distribution of estimated coefficients (along with 95% confidence intervals) for

[22]It turns out that the requirement that all models begin with 2003 as the start of the first period (which requires years near the end of the sample to be dropped in some models) does not substantially alter the point estimates from the long difference models (Appendix Table A.6).

the regression of each cause-specific outcome on the one-year lead employment rate (denoted $r = -1$ on the horizontal axis), the contemporaneous employment rate (denoted $r = 0$), and four lag terms ($r = 1$ to $r = 4$).

In these figures, the coefficient plotted at $r = 0$ along the horizontal axis reflects the estimated contemporaneous effect of a one percentage point increase in employment, controlling for EPOP from previous years and the next year; meanwhile, the effect at $r = 1$ reflects the dynamic marginal effect from a one percentage point increase in EPOP in the previous year, controlling for EPOP from two years prior, in the contemporaneous year, and in all subsequent years.[23] It is important to interpret these estimated effects as a short-term increase in EPOP in a given year that does not persist over time, as that effect would be represented by the sum of these distributed lag coefficients (Greene, 2008; Ruhm and Black, 2002). Given that no lag coefficients are indistinguishable from zero at the 5% level, I do not present plots of these cumulative effects in this analysis.

The lag coefficients for all four mortality outcomes in Figure 2.10 are imprecisely estimated, and I am therefore unable to distinguish between instantaneous versus lagged effects of short-term employment changes with any high degree of confidence. Despite the imprecision of these results, there are some suggestive patterns that emerge from the distribution of lag coefficients for alcohol and non-drug suicide (Appendix Tables A.7 and A.9, respectively). The top-left panel in Figure 2.10 displays the lead and lag distribution for alcohol, corresponding to the point estimates in column 6 of Table A.7. Although it is not indistinguishable from zero at the 5% level, the negative point estimate on the contemporaneous term ($r = 0$) is suggestive of an instantaneous and protective effect of employment rate increases on alcohol-related mortality. However, there is no evidence of any lagged effects of this short-term shock on alcohol mortality as shown by the fact that the point estimates on all subsequent lag terms cluster around zero. This instantaneous effect is also reflected in the coefficients from the less saturated models (i.e., with one, two, or three lags) presented in Appendix Table A.7, which show a relatively large negative estimated effect on the contemporaneous term only.

This pattern stands in contrast with the distributed lag estimates for non-drug suicide, which show slight indications that the previously identified protective effect of employment may be attributable to employment conditions from the previous year, rather than a contemporaneous effect. Specifically, the bottom-left plot in Figure 2.10 shows a discontinuous negative shift on the first lag term but not on the contemporaneous term (although this is

[23] A specific example helps clarify the point estimates presented in Figure 2.10 and Appendix Tables A.7–A.10. Consider the cause-specific mortality rate in year 2010 and the distributed lab model with one lead term and four lag terms. The coefficient presented along the horizontal axis at $r = 0$ in Figure 2.10 is the estimated effect on mortality in 2010 attributable to a one percentage point increase in that year (2010), controlling for the lagged effects of EPOP from 2009, 2008, 2007, and 2006 and the potential future effect of EPOP in 2011. Meanwhile, the coefficient at $r = 1$ represents the estimated effect of a one percentage point increase in EPOP in 2009 on 2010 mortality, controlling for the lagged effects of EPOP from 2008, 2007, and 2006, in addition to the contemporaneous effect (EPOP in 2010) and the future effect of EPOP from 2011. This continues to $r = 4$, which isolates the effect of EPOP from 2006 on 2010 mortality, controlling for all other years.

not statistically significant at the 5% level), which is followed by coefficients that are indistinguishable from zero for lags 2 through 4. This pattern is supported by the models presented in Appendix Table A.9, where the magnitude of the point estimate of the contemporaneous term decreases substantially and is no longer statistically significant after the inclusion of the lag terms (e.g., from -0.21 to -0.089 after a single lag is added between columns 2 and 3). Moreover, the negative coefficient on first lag is the largest in magnitude across all models in columns 3–6, although again it is not statistically significant at conventional levels.

In order to better visualize potential pretrends, Appendix Figure A.3 presents the lag distribution for the model estimated with two lead terms and four lag terms. The pattern of lag coefficients for alcohol and non-drug suicide in these plots is qualitatively similar to those described above, although the estimates are highly volatile particularly at more distal lags. I emphasize again that the patterns described above for alcohol and non-drug suicide should be interpreted with extreme caution given the imprecision of the distributed lag coefficients. Future research leveraging a longer panel should replicate this analysis to see if these suggestive patterns hold with more years of data.

2.5 Model sensitivity

I return now to the results from the baseline set of contemporaneous fixed effect models, which broadly suggest that improvements in county-level employment conditions have countervailing effects on deaths of despair by decreasing rates of non-drug suicide while increasing mortality from unintentional drug-related causes. In this section, I consider several alternative model specifications and variable constructions to probe the robustness of these findings. I also present adjusted p-values for the main point estimates after considering the possibility of over-rejecting the null hypothesis by chance due to the large number of hypotheses being tested using the same model. I also modify two design-based features of the study to further examine the consistency of these results across two key dimensions: the level of geographic aggregation and the duration of the study period. Results from this section call into question some of the key findings from the contemporaneous model. In particular, I find that my results are highly sensitive to the years included in the study sample and, with the exception of the aggregate model, are not robust to corrections for multiple hypothesis testing.

2.5.1 Robustness of contemporaneous model

I begin by discussing the Westfall-Young adjusted p-values for all contemporaneous regression results, which are presented along with the naïve p-values in Table 2.7 (for the aggregate and partially disaggregated models) and Appendix Table A.11 (for the fully disaggregated model). Recall that these p-values adjust for dependence across outcomes within families (defined as the full set of all four mortality outcomes), a procedure that controls for the likelihood of spuriously rejecting the null hypothesis due to the number of hypotheses being tested simultaneously. As shown in the leftmost column in Table 2.7, the Westfall-Young ad-

justed p-values for the effect of aggregate county-level employment on aggregate rates of drug non-suicide and non-drug suicide are significant at the 5% level (p = 0.021 and p = 0.018, respectively). However, after adjusting the p-values in the partially disaggregated model to account for the 16 simultaneous hypotheses, no subgroup effects are statistically significant at conventional levels. Only one relationship—the estimated effect of aggregate employment on drug non-suicide among 19–44-year-old females—is even marginally significant at the 10% level (p-value = 0.080). The significant effects in the fully disaggregated model are similarly sensitive: when adjusting for the 64 simultaneous hypothesis tests, the estimated positive effect of employment increases among younger females on drug non-suicide among older males is the only marginally significant finding (p-value = 0.090, see Appendix Table A.11).

I next consider the possibility that the true causal relationship I uncover in the contemporaneous model is one in which cause-specific mortality rates determine aggregate levels of employment, rather than the other way around. Appendix Table A.12 (panel A) presents results from the distributed lag model for each outcome with a contemporaneous and one-year lead term, which allows for a crude examination of reverse causality bias.[24] Because employment rates are correlated over time, controlling for the contemporaneous term is necessary to isolate any marginal effects of an increase in the next year's EPOP on the current mortality rate—an effect that would point to potential biases due to reverse causality or other sources of endogeneity. For all outcomes, the estimated coefficient for the one-year lead term is relatively small in magnitude and indistinguishable from zero. However, it is noteworthy that the estimated coefficient on the contemporaneous term in the drug non-suicide model is no longer statistically significant when controlling for the previous year's EPOP. In contrast, the effect of employment on non-drug suicide remains negative and statistically significant at the 5% level even after controlling for the one-year lead term.

I next re-estimate the preferred fixed effects model with the addition of a variable that controls for fluctuations in local housing prices—a key characteristic of local economic conditions found in previous research to confound the relationship between employment conditions and drug mortality (Brown and Wehby, 2019). Because this variable may be time-varying at the local level, the current set of county, year, and state-by-year fixed effects will not adequately control for this possibility, and my preferred model may suffer from omitted variables bias. Panel B of Appendix Table A.12 presents estimates from the aggregate model after controlling for a county-level index of annual housing prices developed by the Federal Housing Finance Agency (Bogin et al., 2019). I find no substantial differences between these estimates and those presented in Table 2.2, although the estimated effect of a percentage point increase in EPOP on drug non-suicide is slightly attenuated.

Unlike household- or employer-based sources of employment, the unit of analysis in the Quarterly Workforce Indicators is the job, which introduces the possibility that my employment measure double-counts individuals who hold multiple jobs (Currie et al., 2019). This may result in actual and predicted employment-population ratios substantially greater than

[24]The estimates presented in panel A of Appendix Table A.12 are identical to those in column 2 of Appendix Tables A.7, A.8, A.9, and A.10 for alcohol, drug non-suicide, non-drug suicide, and drug suicide.

1, as shown in Figures 2.2 and 2.3. Panel C in Appendix Table A.12 presents a modification of the preferred model that replaces the full distribution of EPOP and predicted EPOP with measures that are winsorized at the 0.5 and 99.5 percentiles. Despite the long right tail in the full distribution, the coefficient estimates are virtually unchanged in this model although the strength of the F-statistic improves somewhat from 169.7 to 204.2.

Panel D in Appendix Table A.12 presents estimates from a model that replaces the dependent variable with the inverse hyperbolic sine (IHS) transformation of the count of deaths in each county-year cell. The benefit of this transformation is that the estimated coefficients can be interpreted directly as elasticities as in a log-log model but is defined when the count of deaths in a given cell are zero. One drawback, however, is that the performance of this model is unknown when the share of zeros in the dataset is relatively large (see footnote 3), which is often the case for rare outcomes such as mortality. It is therefore noteworthy that the IHS model generates somewhat different results than my preferred rate models. I find that the estimated positive effect of employment on drug non-suicide is attenuated by approximately one-half and no longer statistically significant, while the estimated protective effect on non-drug suicide remains statistically significant and is slightly larger in magnitude.

2.5.2 Sensitivity to level of geographic aggregation

Despite the breadth of literature examining the relationship between macroeconomic conditions and health outcomes using aggregate-level datasets, there is little consensus regarding the most appropriate level of geographical aggregation to use. Common units of analysis in the US-based literature range from states, to commuting zones (CZs), to counties; and some studies leverage variation across even smaller units such as zip codes and census tracts (Currie et al., 2015). On one hand, smaller levels of geographic aggregation may improve the precision of estimates by incorporating variation in economic conditions that would be subsumed in more aggregate-level analyses. On the other hand, larger levels of aggregation are better able to capture the many ways in which "economic conditions both near and far may affect an individual's health"—not only through one's own job loss but also mechanisms ranging from perceptions of job loss, changes in government expenditures, migration, social network effects, and more (Lindo, 2015, p. 84). In light of these statistical and conceptual considerations, it is unclear *a priori* what the most appropriate level of geographic aggregation may be for capturing the effects of economic despair on mortality outcomes.

While the present study uses the county level as a starting point, it may be the case that using census-defined county borders does a poor job of measuring economic activity at the local level relative to alternative measures such as CZs (Autor and Dorn, 2013). To the extent that the present analysis does not capture the potential effects of cross-county spillover effects of economic conditions on mortality, it is likely that my estimates would be smaller in magnitude relative to estimates at larger levels of geographic aggregation (Lindo, 2015).[25] To test this hypothesis, I replicate the models from Table 2.2 at the CZ level using

[25]In a comparison of the effects of employment rates on all-cause mortality at various levels of aggregation,

a county-to-commuting zone crosswalk provided by Autor and Dorn (2013).

The results, presented in panel E of Appendix Table A.12, are qualitatively similar to my preferred estimates, although the coefficient estimates are less precisely estimated and substantially larger in magnitude. Specifically, a one percentage point increase in EPOP at the CZ level is estimated to decrease non-drug suicide by -0.31 per 100,000 (95% CI: -0.59, -0.03), or by approximately 2.23% relative to the CZ-level mean, while increasing drug non-suicide mortality by 1.27 per 100,000 (95% CI: 0.22, 2.31), or 6.85% relative to the CZ-level mean. These substantially larger relative effects (approximately two and four times larger than those estimated at the county level for non-drug suicide and drug non-suicide, respectively) point to potentially meaningful differences in the effect of employment conditions on mortality by the level of geographic aggregation, possibly due to spillover effects not captured at the county level.

2.5.3 Sensitivity to analysis period

The primary analytic sample used in this analysis includes county-years from 2003–2017, a 15-year period during which working-age adults in the United States experienced substantial increases in drug, suicide, and alcohol-related mortality overall and by demographic group. While longer panel periods are undoubtedly preferable, it is important to note that this is not an abnormally short panel in the context of the literature examining macroeconomic changes on health outcomes. A review of studies through 2015 on the effect of unemployment rates on self-reported health show that most studies included in the survey contain fewer than 15 years, with some leveraging substantially shorter panels (Currie et al., 2015). Nonetheless, given the sensitivity to the analysis period identified in previous studies (see section 3.4) and the volatility across the long difference models described above, examining the effect estimates from the contemporaneous model across different data windows within the 2003–2017 period is a useful endeavor.

Figure 2.11 presents the result from this exercise, with each point representing the point estimate (and 95% confidence interval) from the IV regression of contemporaneous EPOP on cause-specific mortality over a sample period ending in 2017 and beginning in the year denoted by the horizontal axis (i.e., each figure contains 11 points corresponding to panel periods ranging from 2003–2017 to 2013–2017). Figure 2.12 presents results from the equivalent exercise with the end year of the panel, where I estimate the model over sample periods defined by sequentially increasing the final year of the panel, beginning in 2007 (i.e., each figure contains 11 points corresponding to panel periods ranging from 2003–2007 to 2003–2017). Finally, Appendix Figure A.4 shows the results from estimating the same model on six decade-long panel windows from 2003–2012 to 2008–2017.

The first two sets of figures demonstrate that the estimated coefficients are sensitive to the years contained in the estimation sample as well as the duration of the period. As

Lindo finds that a one percentage point increase in county-level EPOP is associated with a 1.75 per 100,000 increase in mortality while a one percentage point increase in state-level EPOP is associated with a 3.25 per 100,000 increase (Lindo, 2015).

expected, the point estimates are more volatile (and the confidence intervals are wider) on the right-hand side of the plots in Figure 2.11 and the left-hand side of the plots in Figure 2.12, where the shortest panel is only five years long. It is unsurprising that I find no evidence of a statistically significant relationship between employment and any cause of death with this short of a panel, although five years represents an appropriate lower bound given findings from recent research on prescription opioid availability and labor market outcomes using a panel dataset of this duration (Harris et al., 2019).[26] More interesting, however, is the fact that even at relatively longer panel durations, the addition or removal of just one data year might be the difference between concluding that an estimated effect is (or is not) statistically indistinguishable from zero at the 5% level.

For example, the top-right panel in Figure 2.12 shows that for 10 of the 11 sample periods (the exception being the full 2003–2017 period), the preferred model provides no evidence of a statistically significant effect of employment on drug non-suicide at conventional levels. A similar pattern emerges with non-drug suicide mortality, an outcome that demonstrates significant countercyclical variation in only 3 of the 11 sample periods. Figure 2.11 shows that these two outcomes are sensitive to changes in the start of the panel period as well. While it could be argued that this sensitivity is due to the improved precision of the estimates from longer periods, Appendix Figure A.4, which plots point estimates from a shifting 10-year window, suggests that this is not the case. The top-right panel in Appendix Figure A.4 similarly shows that drug non-suicide is procyclical only when the period covers the most recent data years (2016 and 2017), and non-drug suicide is statistically significant at conventional levels in only one of the six decade-long periods (2005–2014).

Qualitatively, trends in the point estimates are also of interest. The top-left panels in Figure 2.11 and Appendix Figure A.4 show a monotonic decrease in the estimated effect of employment on alcohol mortality, suggesting that alcohol-related mortality may be becoming more counter-cyclical over time. In contrast, drug non-suicide appears to be trending in a more procyclical direction in more recent years (top-right panels of Figures 2.12 and Appendix Figure A.4). This latter finding is in contrast with Ruhm's conclusion that most external causes of death, including suicides and poisonings, are becoming more countercyclical and less procyclical in recent decades (Ruhm, 2015). While the 15- to 10-year periods in the current study are not long enough to draw strong conclusions regarding the changing cyclicality of cause-specific mortality, this exercise highlights how point estimates are sensitive to different panel periods and may point to the changing nature of these relationships over time.

[26] Ruhm also examines the cyclicality of mortality outcomes over 5-year windows, although he argues that such "analysis periods are too short to provide reliable estimates" and suggests a 15-year minimum period to adequately examine mortality outcomes (Ruhm, 2021a, p. 21).

2.6 Discussion

The present study examines whether short- and medium-term fluctuations in county-level employment conditions affect mortality rates from drugs, suicide, and alcohol-related causes, and whether these effects change over time. There are several competing narratives that emerge from the analysis presented above. The first relates to the substantive findings on the effects of the contemporaneous impact of employment changes on DSA mortality—a set of findings that, if taken at face-value—have important implications for the population health impact of local-level employment conditions. These findings are especially relevant in the wake of the COVID-19 recession, which has severely disrupted local economies and led to unprecedented levels of job loss and financial hardship. The second through-line concerns the sensitivity of the estimated effect of county-level employment conditions on mortality, which provides a cautionary tale for researchers and consumers of the evidence linking macroeconomic conditions to deaths of despair. I begin by discussing the key findings from the aggregate and demographic group-specific models and then turn to the limitations of this analysis and implications for future research on this topic.

2.6.1 Review of key findings in the context of the existing literature

In the present analysis of the effects of contemporaneous employment conditions on cause-specific mortality, I find statistically significant and robust evidence of a protective effect of employment increases on non-drug suicide. I present suggestive evidence that this relationship may be unique to males, although the results from the multiple hypothesis testing calls into question the robustness of this finding. This countercyclical variation aligns with a large body of prior studies examining the effects of economic decline, unemployment, and job loss on adverse mental health outcomes. The majority of research on this topic—from both individual and aggregate studies—converge in finding that economic recessions and elevated levels of economic distress are deleterious to mental health and have a particularly strong association with suicide among men (Goldman-Mellor et al., 2010; Modrek et al., 2013).[27] In a systematic review of mental health outcomes following the Great Recession, Modrek et al. (2013) identify 21 studies that document increasing rates of suicide or suicide attempts as a result of economic decline. These findings echo those from Ruhm and others on business-cycle fluctuations and mortality throughout the 20th century, suggesting that this particular relationship is not unique to the Great Recession and may reflect a more general pattern of population-level psychological distress as a result of weakening economies (Granados, 2005; Ogburn and Thomas, 1922; Ruhm, 2000, 2015).

With the caveat that these results are imprecisely estimated in the current study, I find suggestive evidence that the effects of employment fluctuations on suicide are transitory and

[27] One notable exception to this is Pierce and Schott (2020), who find no evidence of increased suicide rates in response to trade-induced economic shocks.

do not persist beyond one year following an employment shock. This pattern is similar to findings from Ruhm (2000), who finds larger immediate-term effects of unemployment on suicide. On the other hand, this is somewhat inconsistent with recent findings by Adda and Fawaz (2020), who demonstrate significant deleterious effects of trade shocks on aggregate mental health outcomes for up to 4 years. The fact that I identify no effect after only one year may be due to the nature of an economic recession being less persistent than a trade-induced shock—the latter of which may fundamentally change the labor market in ways above and beyond the loss of employment (e.g., the high cost of moving across sectors), which may contribute to more lasting adverse health effects (Adda and Fawaz, 2020).

However, these short-term (albeit suggestive) effects of employment changes on suicide are largely consistent with findings from individual-level studies of job loss on mental health outcomes. Leveraging individual-level data following plant closures in Denmark, Browning and Heinssen (2012) show that the risks of death due to mental illness and suicide are 4.5 and 3.1 times higher, respectively, in the year of a plant closure. The authors find that this effect dissipates, although it is still significantly elevated for up to three years beyond the year of displacement. Schaller and Stevens (2015) similarly find evidence for increased self-reported anxiety or depression following a job loss that is concentrated in the period immediately following displacement. Short-term effects are also consistent with recent literature documenting decreases in suicide following the implementation of income support policies such as the minimum wage and Earned Income Tax Credit (Dow et al., 2020). Together, these findings suggest that the mechanism through which employment affects suicide may be relatively rapid and point to the potential for policies that increase labor market opportunities to have immediate health benefits.

While many area-level analyses—including the present study—are unable disentangle the many potential mechanisms linking changes in employment conditions to suicide, the overall convergence between aggregate (i.e., ecological) and individual-level studies of this relationship suggests that the effect of employment on suicide is not limited to those who experience changes in employment status themselves. Rather, these findings are suggestive of high levels of population-wide distress in response to economic decline that may result from changes in one's own employment status as well as via indirect pathways such as perceptions of job precarity, changes in employment among friends or family members, and fiscal austerity measures that decrease access to the social safety net or medical care (Modrek et al., 2013).

The findings from the disaggregated analysis in the current study, while also not robust to multiple hypothesis testing, are also suggestive of potential widespread effects of employment conditions on mortality. The decrease in non-drug suicide among older and younger males that appears to respond only to increases in employment among younger males, points to potential cross-generational effects of changes in employment conditions that should be thoroughly examined in future research. Despite the well-established literature examining the psychological effects on children of a parental job loss (Brand, 2015), to my knowledge there are no studies considering effect of a younger generation's economic prospects on the health and well-being of their parents.

However, this relationship is theoretically plausible in the context of a multi-generational

household in which employment among the younger generation is the primary source of income or support for older family members. The decreasing likelihood that one's offspring will have a better life than oneself, as is shown to be the case in extensive work on intergenerational mobility in the US (Chetty et al., 2017) and alluded to by Case and Deaton (2017), may also contribute to a sense of hopelessness and increased psychological distress that would be consistent with these findings. The well-documented trend in "doubling up" (moving into shared living arrangements) during economic downturns may increase the likelihood that this mechanism is at play (Mykyta and Macartney, 2012; Wiemers, 2014). Due to the ecological nature of the data in the present study, and the sensitivity of these findings when correcting for multiple hypothesis testing, I emphasize that such a relationship can only be speculated based on the current analysis.

The finding in the present study that county-level employment fluctuations significantly increase drug overdoses is a surprising result that largely contrasts the "deaths of despair" hypothesis as well as much of the existing empirical evidence on countercyclical variation in drug-related mortality. This finding most notably diverges with the previous literature that has taken area-based approaches and identified significant increases in opioid overdose fatality, emergency room visits, and self-reported opioid use disorder in response to high unemployment rates (Carpenter et al., 2017; Hollingsworth et al., 2017) or in areas recently experiencing mass layoff events (Venkataramani et al., 2020). Much of the trade shock literature has similarly found evidence of increased opioid and drug mortality in response to economic shocks (Charles et al., 2019; Pierce and Schott, 2020), as have other studies using similar shift-share instruments (Betz and Jones, 2018; Musse, 2020). While Carpenter and colleagues do find evidence that the use of some illicit substances are procyclical (e.g., crack and LSD), the relative contribution of overdose fatalities from these substances is minor in comparison with those attributable to opioids.[28]

Despite the consistency in these countercyclical findings with respect to opioids, the relative role of macroeconomic factors as determinants in the overall opioid addiction epidemic in the US is far from settled (Currie and Schwandt, 2020; Maclean et al., 2020). Currie et al. (2019) find limited evidence of a relationship between local employment conditions and demand for prescription opioids, and other studies find negligible effects of unemployment or other macroeconomic characteristics on opioid overdose after accounting for potentially confounding factors and controlling for other macroeconomic indicators (Brown and Wehby, 2019; Ruhm, 2019). Recent findings by Ruhm (2019) suggest that changes in county-level macroeconomic factors between 1999 and 2015, proxied by a composite measure that includes the unemployment rate, explain less than 10% of changes in all drug mortality during that period after controlling for county-level characteristics. While suggestive of an opposite relationship between economic conditions and fatal drug overdose, the current findings ap-

[28]However, it is also noteworthy that despite finding an overall shift from weakly procyclical over the 1976–1995 interval to strongly countercyclical over the 1991–2010 period, Ruhm documents that poisoning deaths do begin to trend in a more procyclical direction as later sample years include the period of the Great Recession (Ruhm, 2015). A continuation of these trends in the years following the Great Recession would be consistent with the procyclical findings in the current analysis.

pear to be consistent with the magnitudes estimated by Ruhm and overall conclusion that macroeconomic factors are not primary drivers of the current drug crisis.

To my knowledge, only three studies find evidence of a significant positive effect of employment on opioid-related outcomes (Betz and Jones, 2018; Currie et al., 2019; Metcalf and Wang, 2019). Currie et al. (2019) estimate IV models with age-by-sex specific employment shocks and find significant increases in prescription opioids per capita among women. Because opioid prescriptions per capita is the dependent variable, one plausible explanation for this positive relationship is one in which women may increase consumption of prescription opioids in order to remain or return to the labor force. This suggestion is consistent with their simultaneous finding that areas with high levels of prescription opioids per capita experience increases in female employment. However, I find this explanation unlikely in the current analysis of mortality and theorize that the positive effect I find is more consistent with a strong income effect or a mechanism in which economic expansion drives higher rates of overdose via increases in work-related injury and consumption of highly addictive opioids. This increase in the demand for opioids, which I examine in the next chapter, could lead to misuse, addiction, and ultimately higher rates of fatal overdose.

In their study of opioid overdose fatalities in response to changes in employment in the mining industry, Metcalf and Wang (2019) find evidence that increases in the mining employment share increase opioid overdose fatalities at the county level—a procyclical pattern they suggest is likely due to job-related injuries. Despite constituting less than 5% of the employment in the industry, the authors find significant increases in female overdose rates, which the authors speculate are likely due to community-level increases in opioid use. The results from the cross-group models in the current study, which point to increases in drug non-suicide mortality as a result of employment increases in other demographic groups, are similarly suggestive of increased access to prescription opioids within households or in the broader community.

Finally, changes in the industry composition of the economy during expansions or contractions may also explain this procyclical finding. For example, if employment increases are disproportionately concentrated in high-skill industries, this may decrease employment rates or depress wages among lower-skilled individuals and lead to increases in substance use consistent with the procyclical stress mechanisms described above. This is the explanation suggested by Betz and Jones (2018), who find that opioid overdose fatalities increase in response to employment growth in industries characterized by high median wages, while increases in employment in low-paying industries are found to be protective against opioid overdose. A formal test of these hypotheses—along with the mechanisms underlying the other findings described above—is beyond the scope of this analysis and should be prioritized in future research.

Finally, I find no consistent evidence that employment fluctuations at the county level significantly affect alcohol-related causes of mortality, a finding that is consistent with the null effects found in several recent studies examining the effects of trade-induced economic shocks. However, the literature on the effects of aggregate economic conditions on alcohol consumption and related outcomes remains mixed (de Goeij et al., 2015), and previous

studies have shown recessions to increase heavy drinking and binge drinking but decrease overall consumption (Bor et al., 2013; Dávalos et al., 2012). To the extent that changes in alcohol-related mortality would primarily reflect changes in consumption among heavy drinkers, it is possible that the contemporaneous models do not adequately allow for this process to take place. However, in an examination of these potential lagged effects of alcohol consumption on mortality, Ye and Kerr (2011) find little difference between contemporaneous and distributed lag fixed effect models, suggesting that an increase in alcohol consumption is likely to occur over short time horizons. One additional consideration is the fact that alcohol is often involved in deaths due to suicide and drug overdoses, and the current models consider these to be independent causes of death. Between 1999 and 2017, the share of opioid overdoses that involved alcohol as a contributing cause hovered around 15%, and studies show that co-occurring alcohol use significantly increases the risk of death from opioid overdose (Tori et al., 2020; Witkiewitz and Vowles, 2018). Previous studies looking at alcohol involvement in suicides show that suicides involving alcohol are among the only alcohol-related causes of death to increase consistently during economic recessions, and that the share of alcohol-involved suicides increased during the Great Recession (Kaplan et al., 2015; Kerr et al., 2017). In light of these findings, it may be the case that by using information from only the underlying cause of death rather than all contributing causes, the current analysis underestimates the effect of employment on alcohol-related mortality.

2.6.2 Limitations of the current study

Findings from this paper rest on the assumption that the shift-share instrument I employ identifies purely exogenous variation in labor demand, conditional on the controls in my models. As described above, the key challenge in ensuring the exogeneity in this instrument is that the initial industry mix is uncorrelated with unobserved drivers of year-on-year changes in the mortality outcomes of interest (Goldsmith-Pinkham et al., 2020). Existing research suggests that local labor markets with disproportionate exposure to structural changes over the past several decades (e.g., increased automation and trade) may have experienced elevated rates of deaths of despair (Autor et al., 2019; Charles et al., 2019; O'Brien et al., 2021; Seltzer, 2020). To the extent that the initial industry shares are directly correlated with existing mortality trends (or location-specific correlates of these trends), estimates from the shift-share analysis are likely to still be biased if these factors are unaccounted for. While I am able to control for many of the time-varying factors that appear to be correlated with initial industry shares, I am unable to fully rule out potential unobserved factors correlated with both changes in mortality and changes in the predicted employment rate. The fact that the addition of these time-varying controls substantially alters the point estimates raises concern that there may be unobserved time-varying factors that would bias the estimated effects (see Appendix Table A.13).

Moreover, the implicit assumption in this generalized difference-in-differences design is that the trends in mortality outcomes in counties experiencing an employment shock in a given year do not fundamentally differ from those not experiencing a change in employ-

ment—the equivalent of the "parallel trends" assumption in a setting with continuous treatment. Recent econometric studies demonstrate that the two-way fixed effect estimator may be biased in the presence of variation in treatment timing and heterogenous treatment effects over time (Callaway and Sant'Anna, 2021; de Chaisemartin and D'Haultfœuille, 2020; Goodman-Bacon, 2021). It is likely that in the present scenario, where the independent variable of interest is continuous and there exist no "non-treated" units, similar biases may arise (Callaway and Sant'Anna, 2021). To my knowledge, however, no alternative estimator has been proposed to overcome these challenges.

Relatedly, I take as my preferred empirical specification a model that includes state-by-year fixed effects in addition to year and county fixed effects. While these models adequately control for time-varying state-level factors that may be correlated with employment conditions and mortality trends, it is possible that I do not adequately control for the possibility that county mortality trends changed over time in ways that are systematically related to changes in employment conditions. One solution to this problem is to include county-level time trends, which I present in Appendix Table A.14. These models yield coefficient estimates that are much smaller in magnitude and not statistically significant, although the direction of the effects is similar to the preferred models. I opt for the model with state-by-year fixed effects primarily because small counties demonstrate highly non-linear mortality trends and due to the possibility that models with miss-specified lag structures may be biased in the presence of dynamic effects (Meer and West, 2015). Nonetheless, that my primary estimates are sensitive to the omission of these trends is an important caveat.

There are also several measurement concerns that warrant mention. While considered to be among the most reliable health outcome measures, mortality data from death certificates is not without its own limitations (Burgard et al., 2013). To a certain extent, the underlying cause of death (as well as the contributing causes) listed on a death certificate is still a subjective decision, as it is recorded by a medical examiner, coroner, or other medical professional. Therefore, any systematic biases in this decision-making or recording process, if correlated with the shift-share instrument, may bias the main results from this analysis. Extensive research has shown that there exist substantial geographic heterogeneities in cause-of-death coding, at least in part attributable to differences in the training and resources available for medical certifiers (National Academies of Sciences, Engineering, and Medicine, 2021).

This concern has been most thoroughly considered in the context of opioid-involved overdose mortality due to the large number of drug overdoses classified as overdoses due to "unspecified narcotics." This is a new ICD-10 classification thought to greatly distort the true nature of the opioid epidemic in the United States (Buchanich et al., 2018; Milam et al., 2021; Ruhm, 2018). While the current study largely circumvents this specific issue by not distinguishing between opioids and other drug-related overdoses, similar misclassification issues include distinguishing between intentional and unintentional drug overdoses (i.e., classifying drug non-suicide and drug suicide) and the frequency with which alcohol and drugs co-occur. Acknowledging that these outcomes are not mutually exclusive by considering the full set of contributing causes listed on the death certificate may be an important extension of this work (Boslett et al., 2020).

As discussed at length above, the relatively short panel period used in this study (2003–2017) is an important limitation, especially for the estimation of dynamic effects. The long difference periods in this study, which range from one to six years, are unable to capture the multiple-decade "deterioration in opportunities" hypothesized to be at the root of Case and Deaton's despair hypothesis. However, as alluded to above and supported by empirical evidence from individual-level studies, the behavioral and lifestyle changes that plausibly underlie the relationship between changes in employment conditions and mortality may indeed develop over the relatively short time horizons examined in this study. The short panel period has implications for the distributed lag models as well, where the most saturated model—which includes a contemporaneous term and four lag terms—omits the first four years of the panel by construction, leaving only 11 years of data for estimation. If the results from Section 2.5.3 are any indication, the omission of years early in the panel has the potential to substantially alter the point estimates and overall conclusions from this analysis.

Finally, there are several important dimensions of the "midlife mortality crisis" in the United States that are not considered in the present analysis. It is almost certainly the case that there exist heterogeneous effects of employment conditions on mortality across a variety of population characteristics beyond age and sex, the two dimensions studied here. In addition to key demographic factors such as educational attainment and race (as I discuss in more detail in Chapter 4), disaggregating overall employment trends into industry- or occupation-specific measures would likely illuminate key differences in the degree to which changes in employment affect mortality outcomes across different segments of the labor market. Many recent studies examining labor market changes and deaths of despair have taken this approach, finding evidence of heterogeneity in the relationship between aggregate employment conditions and mortality across industries (Charles et al., 2019; Metcalf and Wang, 2019; Monnat et al., 2019; Musse, 2020; Seltzer, 2020) and labor markets characterized by higher shares of lower-skilled and lower-wage workers (Adda and Fawaz, 2020; Betz and Jones, 2018).

2.6.3 Implications for policy and future research

The findings in this study have direct implications for health policy and point to important areas for future research. The responsiveness of suicide mortality to short-term fluctuations in employment highlights the potential for policies that connect those seeking employment to jobs as a way to significantly improve population health. For example, "active labor market programs" in European countries, which are designed to train and retain or reintegrate workers into the labor force, were shown to decrease suicide more than any other public expenditure during previous economic downturns (Stuckler et al., 2009). Such policies may not only protect the health of individuals who lose jobs during economic downturns but also provide an important sense of job security and reassurance to individuals, families, and communities that may experience the harmful "spillover effects" of macroeconomic declines. In the wake of unprecedented job loss following the COVID-19 pandemic, investing in such

programs could be incredibly beneficial. In Chapter 4, discuss these programs and additional policy levers that could mitigate an acceleration of DSA mortality.

Important gaps in our understanding of the association between macroeconomic factors and deaths of despair remain. As Case and Deaton remark, the key to understanding the rise in midlife mortality through the lens of despair is to examine "a long-term and slowly unfolding loss of a way of life for the white, less educated, working class" (Case and Deaton, 2017, p. 146)—a nebulous construct that is difficult to quantify and points to a process that may unfold slowly across decades. As such, more cohort-based studies that follow individual health over long periods of time—particularly among individuals who enter the labor market during economic decline—can help shed light on the extent to which prolonged periods of economic change may accumulate and adversely affect health throughout the life course. Several researchers have taken this life course perspective to understand the relationship between socioeconomic status, macroeconomic conditions, and health outcomes (Coile et al., 2014; Lleras-Muney et al., 2021; Schwandt and von Wachter, 2020) but much more work remains to be done on this topic.

To date, the "deaths of despair" literature has primarily focused on mortality rather than non-fatal (often intermediary) outcomes such as alcohol use disorder and addiction, substance use disorder, and serious mental illness, although there are some key exceptions to this (Shanahan et al., 2019). For example, Carpenter and colleagues (2017) use survey data from the National Survey on Drug Use and Health and find strong counterclyical patterns for substance use disorder involving alcohol and illicit substances. Hollingsworth and colleagues (2017) find a similar relationship between macroeconomic conditions and emergency department visits for opioid overdoses. Aggregate and individual-level studies have similarly documented adverse effects of macroeconomic shocks and rising unemployment rates on overall mental well-being, psychological distress, and suicide attempts (Avdic et al., 2020; Charles and DeCicca, 2008; Elbogen et al., 2020; Eliason and Storrie, 2009).

Nonetheless, expanding the scope of the "deaths of despair" research beyond mortality to include related but non-fatal outcomes is necessary given the enormous population health burden due to serious mental illness and susbtance use disorder in the United States. In 2018, an estimated 11.5 million adults in the United States experienced a major depressive episode with severe impairment, representing a 24% increase since 2008 among all adults and a 70% increase among young adults (ages 18–25). Furthermore, 10.7 million adults reported suicidal ideation, and it is estimated that in 2018 alone, 1.4 million adults made a non-fatal suicide attempt (Substance Abuse and Mental Health Services Administration, 2019). Moreover, approximately 19 million adults had a past-year substance use disorder, nearly a quarter of whom also suffered from severe mental illness. Given this widespread prevalence and the evidence presented in this study, there is reason to believe that the health effects of improving employment conditions—and potential effects of targeted policies—could substantially improve population health, productivity, and quality of life in the United States.

Chapter 2: Tables and Figures

Table 2.1: Summary statistics

		Age 19–44		Age 45–64	
	Overall	**Male**	**Female**	**Male**	**Female**
All DSA	47.09	53.07	19.94	85.45	37.64
	(21.35)	(31.90)	(16.58)	(40.00)	(23.62)
Alcohol	12.25	4.77	2.38	34.60	14.43
	(7.43)	(6.58)	(4.93)	(22.13)	(12.65)
Drug non-suicide	18.55	24.93	11.43	23.17	14.51
	(13.70)	(23.04)	(12.61)	(20.97)	(14.05)
Non-drug suicide	14.00	21.71	4.41	24.85	5.37
	(7.80)	(15.99)	(6.16)	(17.20)	(7.09)
Drug suicide	2.28	1.65	1.72	2.83	3.34
	(2.23)	(3.31)	(3.49)	(4.63)	(5.14)
EPOP	0.59	0.62	0.60	0.58	0.55
	(0.22)	(0.23)	(0.21)	(0.24)	(0.21)
Observations	46834	46773	46773	46773	46773

Notes: Table presents overall and subgroup-specific mean mortality rates (per 100,000) and standard deviations in parentheses for the main 2003–2017 estimation sample. Observations are weighted by the estimated working-age population in each county-year or county-year-demographic group cell.

Table 2.2: Contemporaneous effect of aggregate employment rates on mortality, aggregate sample (2003–2017)

	(1a) **OLS**	(1b) **IV**	(2a) **OLS**	(2b) **IV**	(3a) **OLS**	(3b) **IV**
Alcohol	-0.0116	-0.0129	-0.0123	-0.0725	-0.00429	-0.0226
(mean = 12.25)	(0.00858)	(0.00970)	(0.0138)	(0.0525)	(0.0114)	(0.0513)
Drug non-suicide	-0.0125	0.0329	-0.0326	0.369***	-0.000454	0.280**
(mean = 18.55)	(0.0251)	(0.0193)	(0.0313)	(0.111)	(0.0368)	(0.103)
Non-drug suicide	-0.0310***	-0.0436***	0.00432	-0.210***	0.0113	-0.161**
(mean = 14.00)	(0.00842)	(0.0100)	(0.0116)	(0.0548)	(0.0102)	(0.0562)
Drug suicide	0.00534***	0.00529***	-0.00239	-0.0136	-0.00319	-0.0166
(mean = 2.28)	(0.00119)	(0.00133)	(0.00424)	(0.0171)	(0.00423)	(0.0179)
County + year FE	N	N	Y	Y	Y	Y
State-by-year FE	N	N	N	N	Y	Y
First stage F-stat.		991.7		167.4		169.7
Observations	46646	46596	46646	46595	46646	46595

Notes: Each cell presents an estimate from a separate regression of the cause-specific mortality rate (per 100,000) on the employment-population ratio (EPOP). The unit of analysis is the county-year. The sample mean of the independent variable (EPOP) is 59.56. In the IV models, EPOP is instrumented using the log of the shift-share instrument constructed analogous to Currie et al. (2019) described in Section 3.3. All models control for the following county-level characteristics: share of the total working-age population that is female; share ages 25–34, 35–44, 45–54, and 55–65; share Hispanic, non-Hispanic Black, and other non-Hispanic non-White; and share with some college education. Each regression is weighted by total working age population (ages 19–64) at the county level. Robust standard errors clustered at the county level are in parentheses. Significance: * p<0.05, ** p<0.01, *** p<0.001.

Table 2.3: Contemporaneous effect of aggregate employment rates on mortality, by age-sex subgroup (2003–2017)

	Males, 19–44		**Females, 19–44**		**Males, 45–64**		**Females, 45–64**	
	OLS	**IV**	**OLS**	**IV**	**OLS**	**IV**	**OLS**	**IV**
Alcohol	0.0116	0.0378	0.00979	0.0681	-0.0519	-0.128	-0.0175	-0.0626
	(0.0105)	(0.0546)	(0.00695)	(0.0422)	(0.0352)	(0.168)	(0.0235)	(0.116)
DNS	0.00118	0.304	0.00877	0.280**	-0.0153	0.458*	-0.0194	0.0409
	(0.0670)	(0.181)	(0.0342)	(0.101)	(0.0516)	(0.182)	(0.0271)	(0.120)
NDS	0.00412	-0.200	0.0130	-0.0504	0.0120	-0.355*	0.0153	-0.00473
	(0.0217)	(0.123)	(0.00918)	(0.0590)	(0.0264)	(0.155)	(0.0133)	(0.0606)
DS	-0.00592	0.000106	-0.00420	-0.00693	-0.00859	-0.0905*	0.00386	0.0225
	(0.00593)	(0.0297)	(0.00563)	(0.0319)	(0.00873)	(0.0370)	(0.00896)	(0.0424)
First stage F		143.9		172.1		172.9		192.0
Observations	46844	46773	46844	46773	46844	46773	46844	46773

Notes: Table presents OLS and IV estimates from the regression of mortality rate per 100,000 on the employment-population ratio (EPOP). The unit of analysis is the county-year-demographic group cell. The sample mean of EPOP is 59.56. Mean mortality rates for each gender-age subgroup are presented in Table 1. In the IV models, EPOP is instrumented using the log of the shift-share instrument constructed analogous to Currie et al. (2019) described in Section 3.3. All regressions include county, year, and state-by-year fixed effects and county-level controls described in the footnote to Table 2. Each regression is weighted by the estimated population size in each cell. Robust standard errors clustered at the county level are in parentheses. Significance: * $p<0.05$, ** $p<0.01$, *** $p<0.001$. Abbreviations: DNS (drug non-suicide), NDS (non-drug suicide), DS (drug suicide).

Table 2.4: Contemporaneous effect of group-specific employment rates on mortality among 19–44-year-olds, by sex

	Panel A: Males, 19–44				Panel B: Females, 19–44			
	Own	**F1**	**M2**	**F2**	**M1**	**Own**	**M2**	**F2**
Alcohol	0.0498	0.114*	0.0252	0.0368	0.0540	0.0313	-0.0180	0.0478*
	(0.0447)	(0.0493)	(0.0276)	(0.0295)	(0.0328)	(0.0456)	(0.0214)	(0.0232)
Drug non-suicide	0.301	0.403*	0.211*	0.160	0.235*	0.214*	0.0548	0.0230
	(0.164)	(0.174)	(0.0854)	(0.0856)	(0.103)	(0.102)	(0.0497)	(0.0540)
Non-drug suicide	-0.251*	0.0300	-0.0783	-0.0441	-0.0297	0.0483	-0.0412	0.0152
	(0.102)	(0.117)	(0.0609)	(0.0702)	(0.0486)	(0.0536)	(0.0270)	(0.0287)
Drug suicide	0.0418	0.00906	0.00616	0.000332	-0.0137	-0.00332	-0.000302	-0.0306
	(0.0262)	(0.0260)	(0.0155)	(0.0155)	(0.0278)	(0.0270)	(0.0149)	(0.0177)
First stage F-statistic	125.9	93.35	298.2	257.9	129.8	83.58	306.8	237.5
Observations	46624	46561	46551	46535	46624	46561	46551	46535

Notes: Table presents IV estimates from the regression of demographic group-specific mortality rates on the employment-population ratio (EPOP) of the demographic group listed in each column, where EPOP is instrumented using the log of the group-specific shift-share instrument. The columns denoted "own" present the estimates from the regression of the group's own EPOP. The unit of analysis is the county-year-demographic group cell. All regressions include county, year, and state-by-year fixed effects and county-level controls described in the footnote to Table 2. Mean mortality and employment rates for each age-sex subgroup are presented in Table 1. Each regression is weighted by the estimated population size in each cell. Robust standard errors clustered at the county level are in parentheses. Significance: * $p<0.05$, ** $p<0.01$, *** $p<0.001$.

Abbreviations: M1 (Male, 19–44), F1 (Female, 19–44), M2 (Male, 45–64), F2 (Female, 45–64)

Table 2.5: Contemporaneous effect of group-specific employment rates on mortality among 45–64-year-olds, by sex

	Panel A: Males, 45–64				Panel B: Females, 45–64			
	M1	**F1**	**Own**	**F2**	**M1**	**F2**	**M2**	**Own**
Alcohol	-0.146	0.00446	-0.0356	-0.172	0.000818	0.107	0.00340	0.0486
	(0.152)	(0.156)	(0.0816)	(0.104)	(0.0968)	(0.0900)	(0.0510)	(0.0571)
Drug non-suicide	0.219	0.501**	-0.00506	0.144	0.0830	0.129	-0.0169	0.0823
	(0.179)	(0.158)	(0.0826)	(0.0785)	(0.104)	(0.108)	(0.0638)	(0.0551)
Non-drug suicide	-0.374**	-0.198	-0.0810	-0.0376	0.00298	0.0377	-0.0530	0.0393
	(0.120)	(0.139)	(0.0662)	(0.0831)	(0.0489)	(0.0559)	(0.0323)	(0.0350)
Drug suicide	-0.0417	-0.0380	-0.0129	-0.00735	0.0221	0.0251	0.0228	0.0435
	(0.0327)	(0.0375)	(0.0206)	(0.0214)	(0.0320)	(0.0377)	(0.0209)	(0.0241)
First stage F-statistic	142.2	127.6	342.9	304.6	141.8	121.8	344.4	285.7
Observations	46624	46561	46551	46535	46624	46561	46551	46535

Notes: Table presents IV estimates from the regression of demographic group-specific mortality rates on the employment-population ratio (EPOP) of the demographic group listed in each column, where EPOP is instrumented using the log of the group-specific shift-share instrument. The columns denoted "own" present the estimates from the regression of the group's own EPOP. The unit of analysis is the county-year-demographic group cell. All regressions include county, year, and state-by-year fixed effects and county-level controls described in the footnote to Table 2. Mean mortality and employment rates for each age-sex subgroup are presented in Table 1. Each regression is weighted by the estimated population size in each cell. Robust standard errors clustered at the county level are in parentheses. Significance: * p<0.05, ** p<0.01, *** p<0.001.

Abbreviations: M1 (Male, 19–44), F1 (Female, 19–44), M2 (Male, 45–64), F2 (Female, 45–64)

Table 2.6: Estimates from contemporaneous fixed effects (FE) and stacked long difference IV models (2003 start year)

		Stacked long difference models					
	Year FE Model	**1-year**	**2-year**	**3-year**	**4-year**	**5-year**	**6-year**
Alcohol	-0.0226	-0.160	-0.172	-0.0603	-0.183+	-0.0981	-0.0894
	(0.0513)	(0.143)	(0.112)	(0.112)	(0.108)	(0.113)	(0.119)
Drug non-suicide	0.280**	0.126	0.147	-0.282*	0.196	0.0807	-0.328*
	(0.103)	(0.163)	(0.155)	(0.125)	(0.153)	(0.146)	(0.143)
Non-drug suicide	-0.161**	-0.140	-0.133	-0.297*	-0.0214	-0.0897	-0.337**
	(0.0562)	(0.139)	(0.132)	(0.136)	(0.126)	(0.150)	(0.115)
Drug suicide	-0.0166	-0.0237	-0.00711	-0.0169	-0.0130	-0.00343	-0.0377
	(0.0179)	(0.0449)	(0.0443)	(0.0384)	(0.0527)	(0.0503)	(0.0408)
First stage F stat.	169.7	97.40	143.5	146.9	114.0	116.9	131.5
Observations	46595	43632	21789	(0.0384)	9334	6207	6206

Notes: Each cell presents an estimate from a separate IV regression of the period difference in cause-specific mortality rates (per 100,000) on the period difference in the employment-population ratio (EPOP). The independent variable is instrumented using the period difference in the log of the predicted EPOP generated by the shift-share instrument. All long difference models control for the period difference in the county-level characteristics described in the footnote to Table 2 and include period fixed effects. The yearly fixed effect (FE) model in the leftmost column is identical to column 3b in Table 2 and is included for comparison. Each regression is weighted by total working-age population (ages 19–64) at the county level. Robust standard errors clustered at the county level are in parentheses. Significance: * p<0.05, ** p<0.01, *** p<0.001

Table 2.7: Estimated contemporaneous coefficients and Westfall-Young adjusted p-values

		Female		Male	
	Aggregate	**19–44**	**45–64**	**19–44**	**45–64**
Alcohol	**-0.0226**	**0.0681**	**-0.0626**	**0.0378**	**-0.128**
Naive p-value	0.659	0.107	0.588	0.488	0.446
Adjusted p-value	*0.785*	*0.688*	*0.995*	*0.991*	*0.991*
Drug non-suicide	**0.280**	**0.280**	**0.0409**	**0.304**	**0.458**
Naive p-value	0.006	0.005	0.733	0.093	0.012
Adjusted p-value	*0.0206*	*0.080*	*0.996*	*0.688*	*0.162*
Non-drug suicide	**-0.161**	**-0.0504**	**-0.00473**	**-0.200**	**-0.355**
Naive p-value	0.004	0.393	0.938	0.103	0.023
Adjusted p-value	*0.0182*	*0.990*	*0.997*	*0.688*	*0.254*
Drug suicide	**-0.0166**	**-0.00693**	**0.0225**	**0.0001**	**-0.0905**
Naive p-value	0.354	0.828	0.596	0.997	0.015
Adjusted p-value	*0.713*	*0.996*	*0.995*	*0.997*	*0.183*

Notes: Table presents estimated coefficients (bold text), naive p-values, and Westfall-Young adjusted p-values (italics) for the preferred contemporaneous aggregate and partially disaggregated models. The Westfall-Young adjusted p-values are calculated using the *rwyoung* Stata command (Jones et al. 2019) with 10,000 replications. All models control for the county, year, and state-by-year fixed effects and county-level characteristics described in Table 2. Each regression is weighted by total working-age population in each cell.

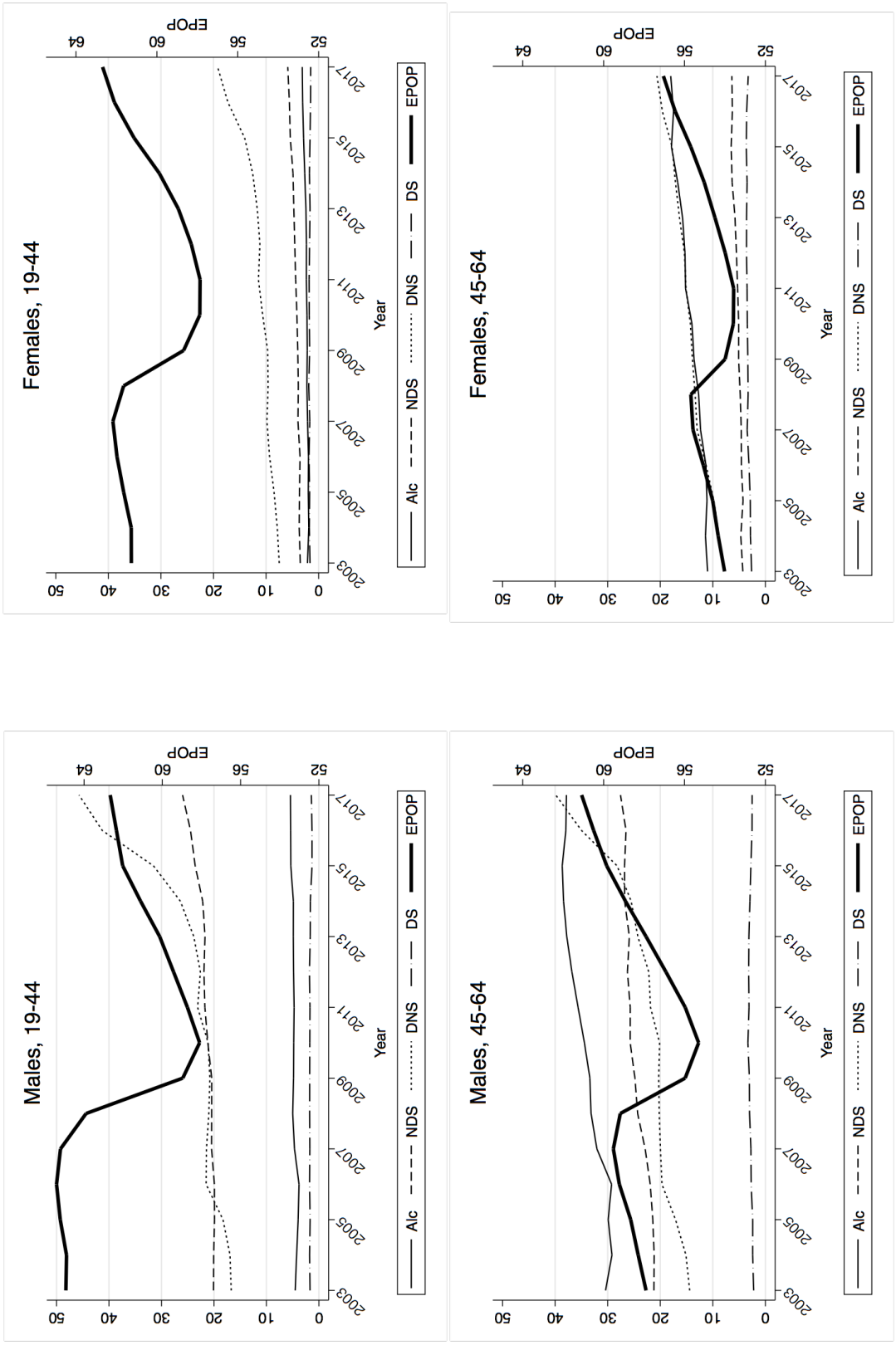
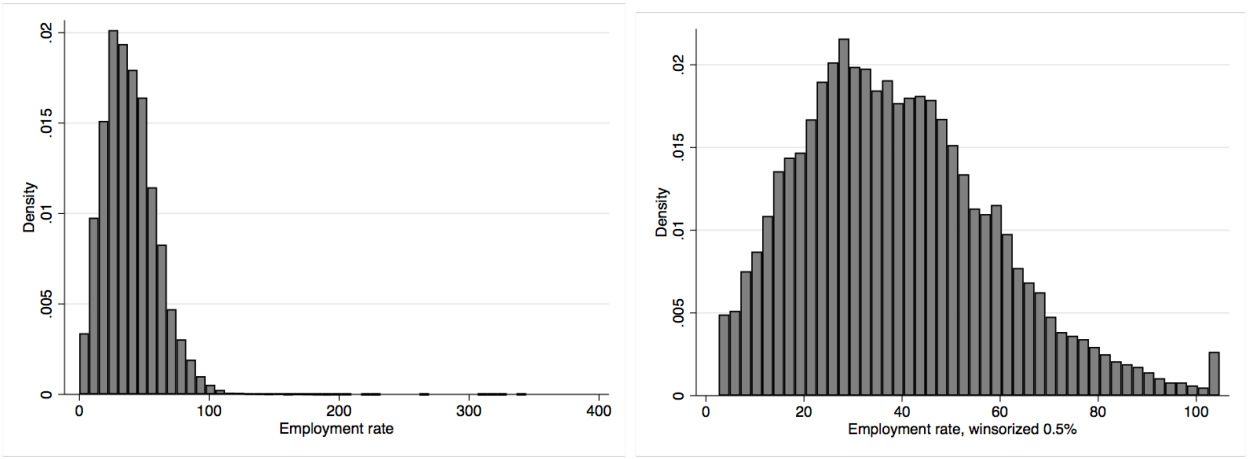


Figure 2.1: Cause-specific mortality rates and employment-population ratios, by demographic group

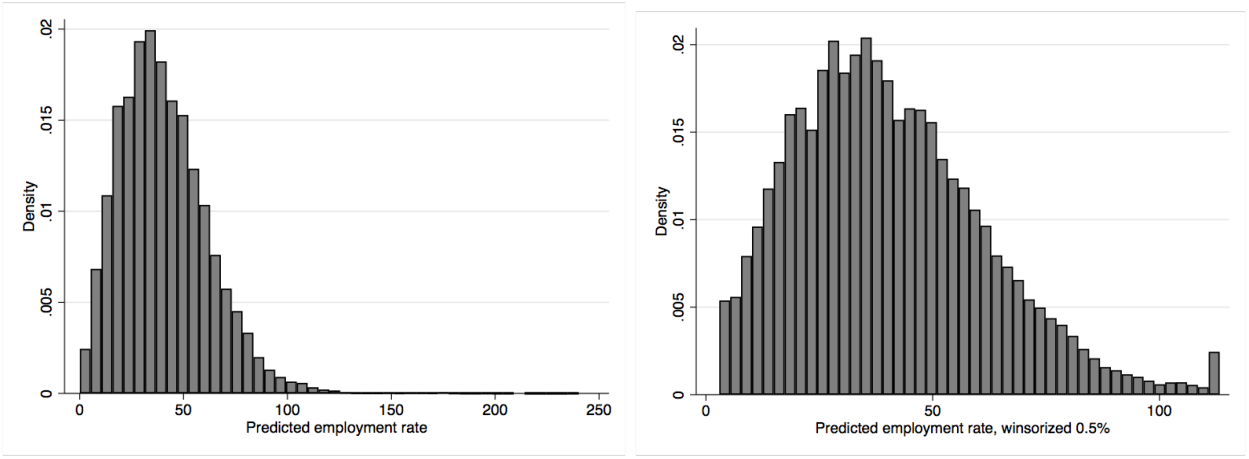
Notes: Figures present county-level population-weighted average mortality rates per 100,000 population (left vertical axis) and employment-population ratios (right vertical axis), by year from 2003–2017. Abbreviations: Alc (alcohol), NDS (non-drug suicide), DNS (drug non-suicide), DS (drug suicide), EPOP (employment-population ratio).

Figure 2.2: Distribution of county-level employment-population ratios, 2003–2017



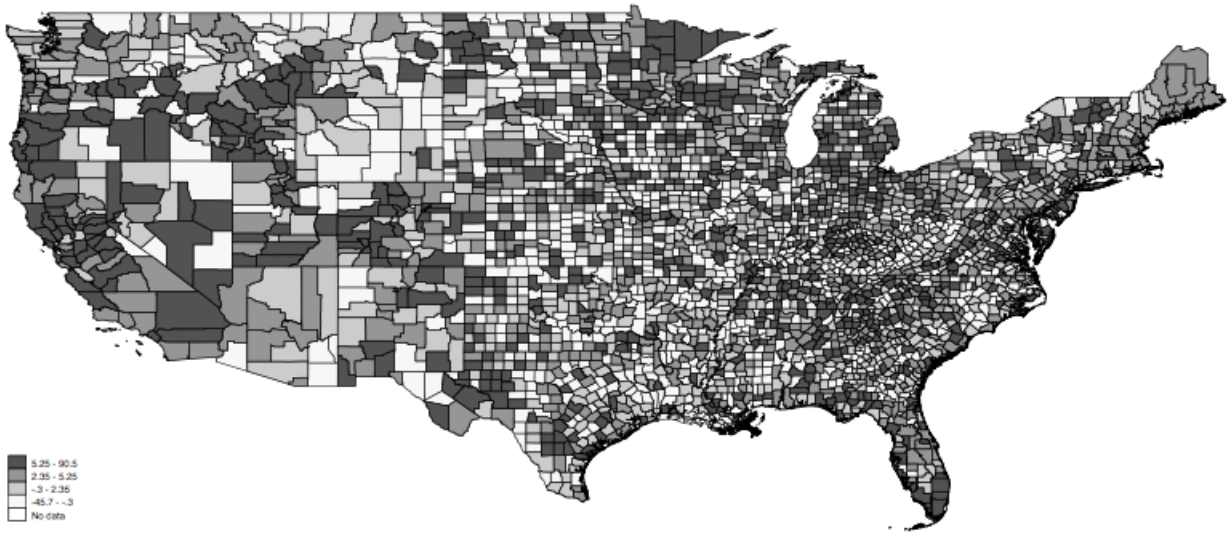
Notes: The figure on the left presents the (unweighted) distribution of the entire sample of county-year employment-population ratios while the figure on the left presents the winsorized distribution of these values (0.5% on the top and bottom of the distribution).

Figure 2.3: Distribution of predicted county-level employment-population ratios, 2003–2017



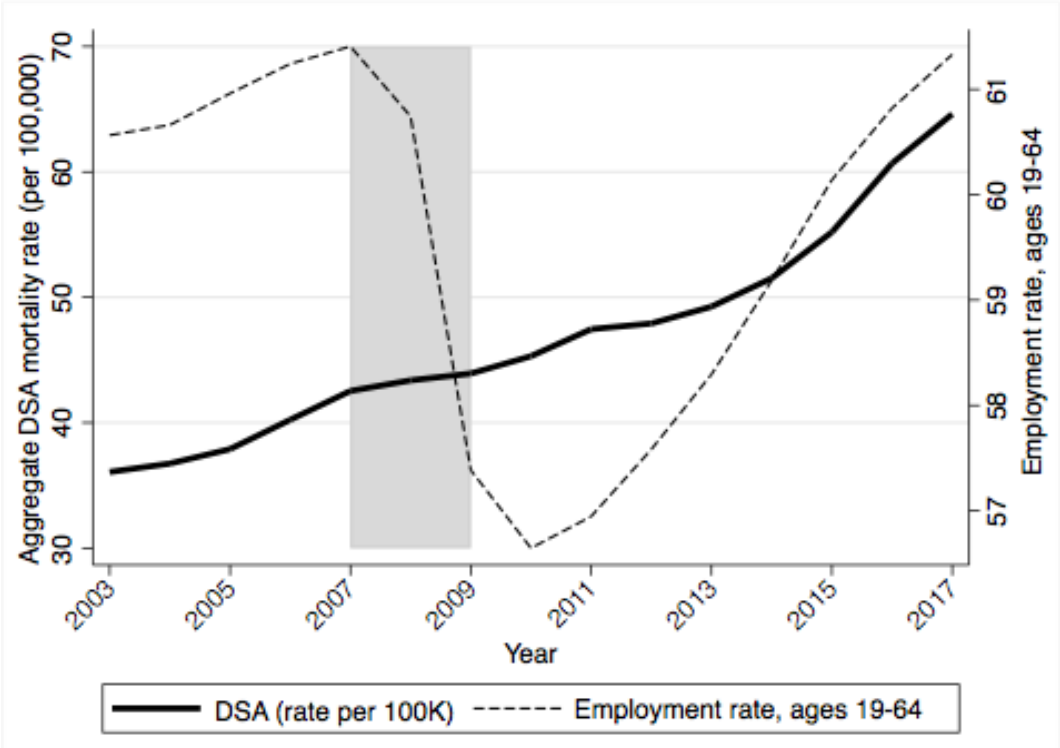
Notes: The figure on the left presents the (unweighted) distribution of the entire sample of predicted county-year employment-population ratios, while the figure on the left presents the winsorized distribution of these values (0.5% on the top and bottom of the distribution).

Figure 2.4: Geographic distribution of changes in employment-population ratios, 2010–2017



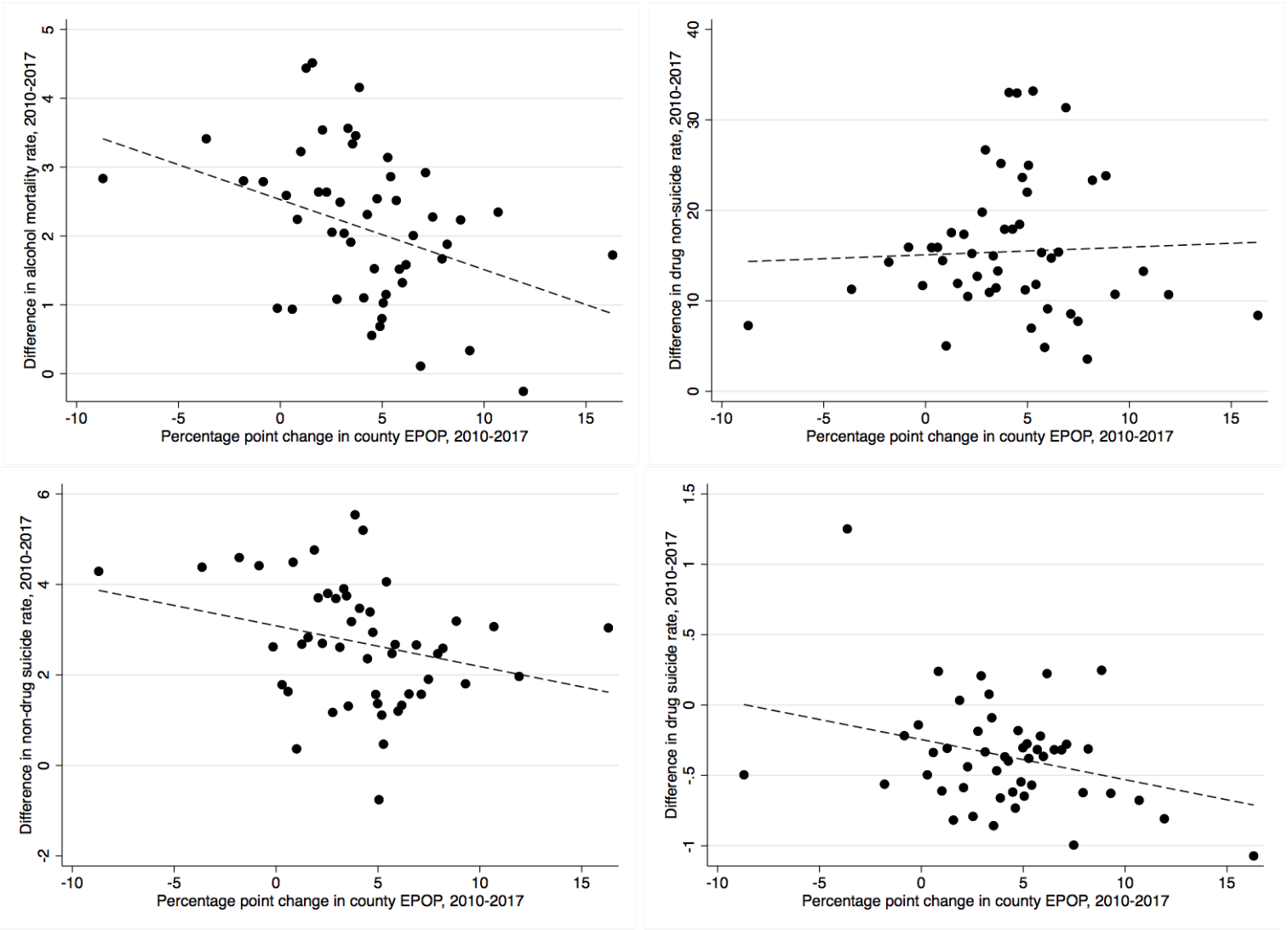
Notes: Map shows quartiles of percentage point changes in county-level employment-population ratios between 2010 and 2017. Darker shades denote counties with larger increases in employment during the 7-year period. Data from Alaska and Hawaii are included in the analytic dataset but are not presented here.

Figure 2.5: Trends in employment rate and despair-related mortality rates, 2003–2017



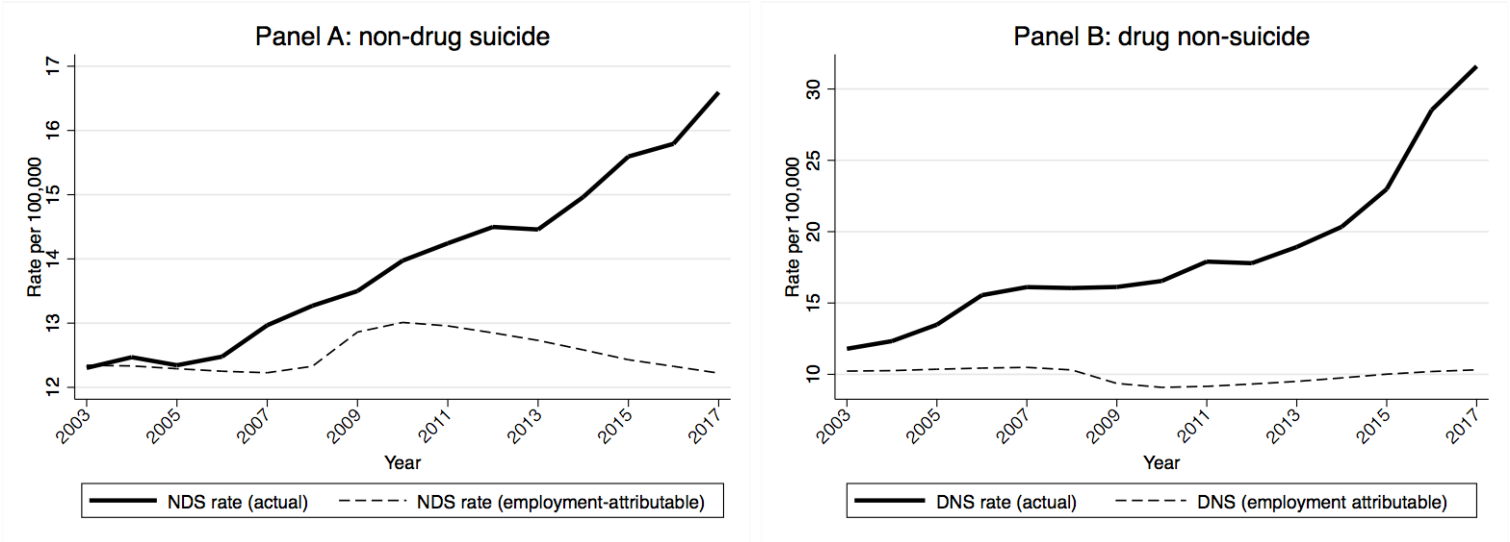
Notes: Figure displays trends in the aggregate employment rate (dashed line, right vertical axis) and overall DSA mortality rate (solid line, left vertical axis) over the 2003–2017 period. Rates are population-weighted averages of county-specific rates in each year. The shaded area denotes the period of the Great Recession.

Figure 2.6: Relationship between changes in cause-specific mortality and employment-population ratios, 2010–2017



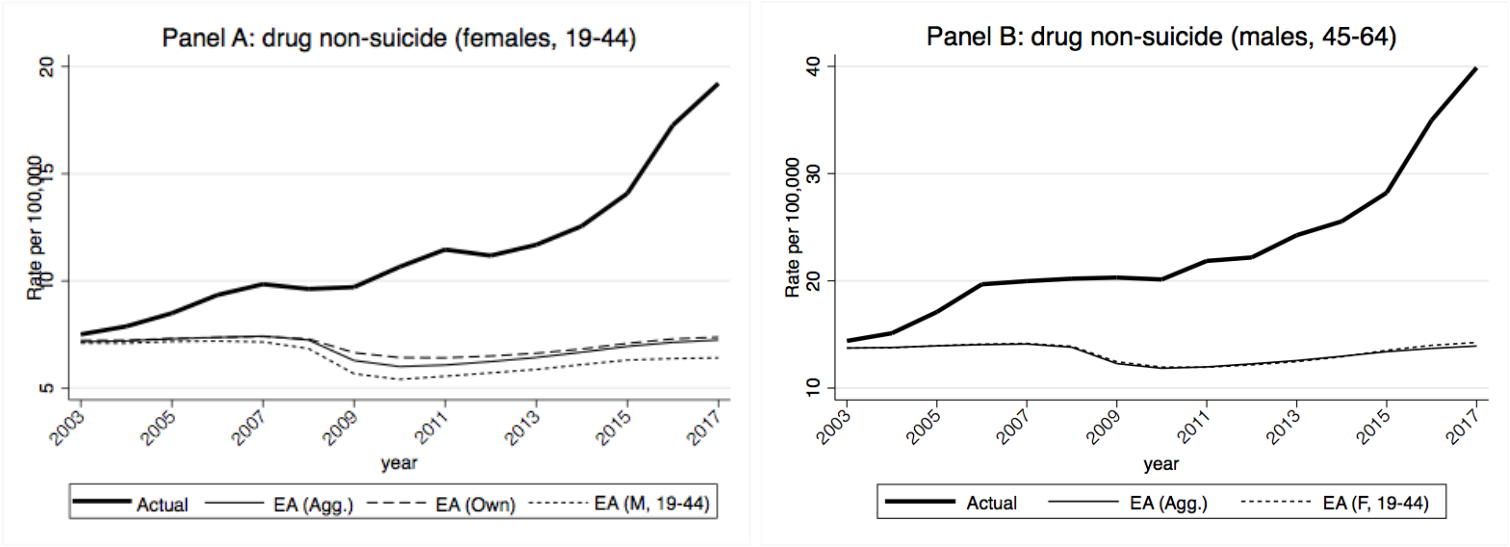
Notes: Figures show the non-parametric relationship between changes in county-level employment-population ratios and cause-specific mortality rates from 2010 to 2017. Points represent mean values of the outcome within 50 evenly sized bins of changes in EPOP. The dashed line depicts a linear fit line weighted by the estimated 2010 population in each county-year observation.

Figure 2.7: Magnitude of estimated aggregate employment effects on non-drug suicide and drug non-suicide relative to actual trends



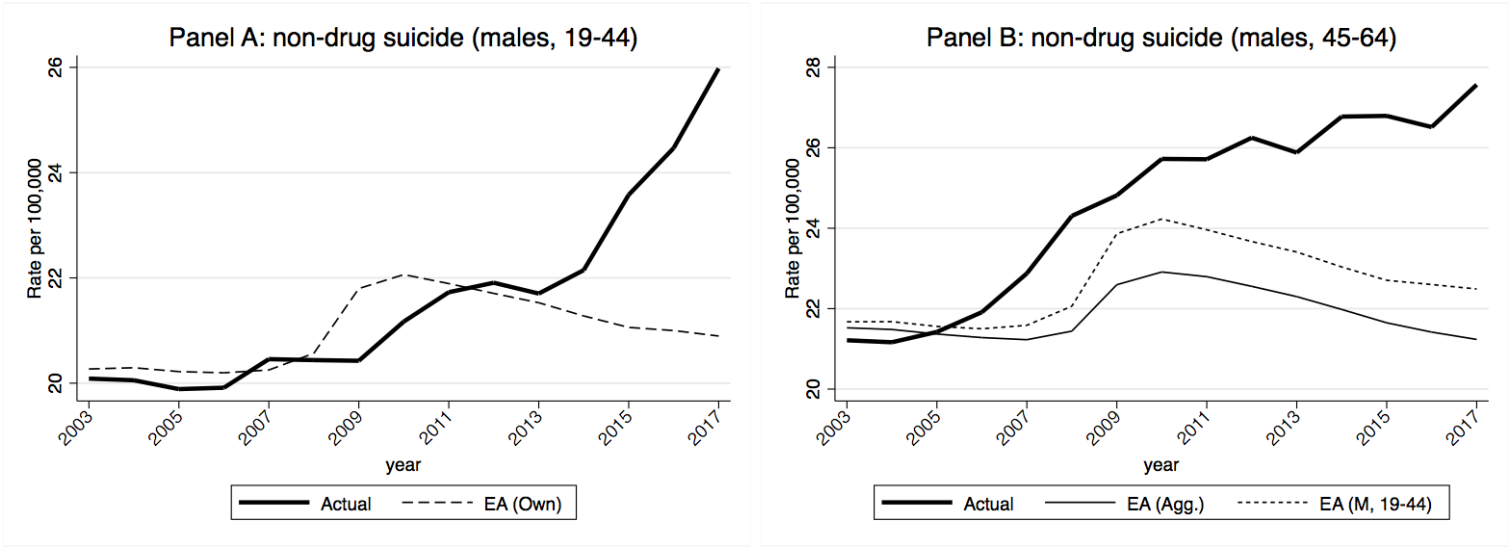
Notes: Figure presents actual (solid) and "employment attributable" (dashed) mortality rates (per 100,000) from non-drug suicide (left) and drug non-suicide (right) over the 2003–2017 period. Employment-attributable rates for each year are calculated by multiplying the difference in the aggregate employment-population ratio in each year relative to 2002 by the preferred IV effect estimate from Table 2. Abbreviations: NDS (non-drug suicide) and DNS (drug non-suicide).

Figure 2.8: Magnitude of estimated aggregate, own- and cross-group employment effects on drug non-suicide among females (19–44) and males (45–64)



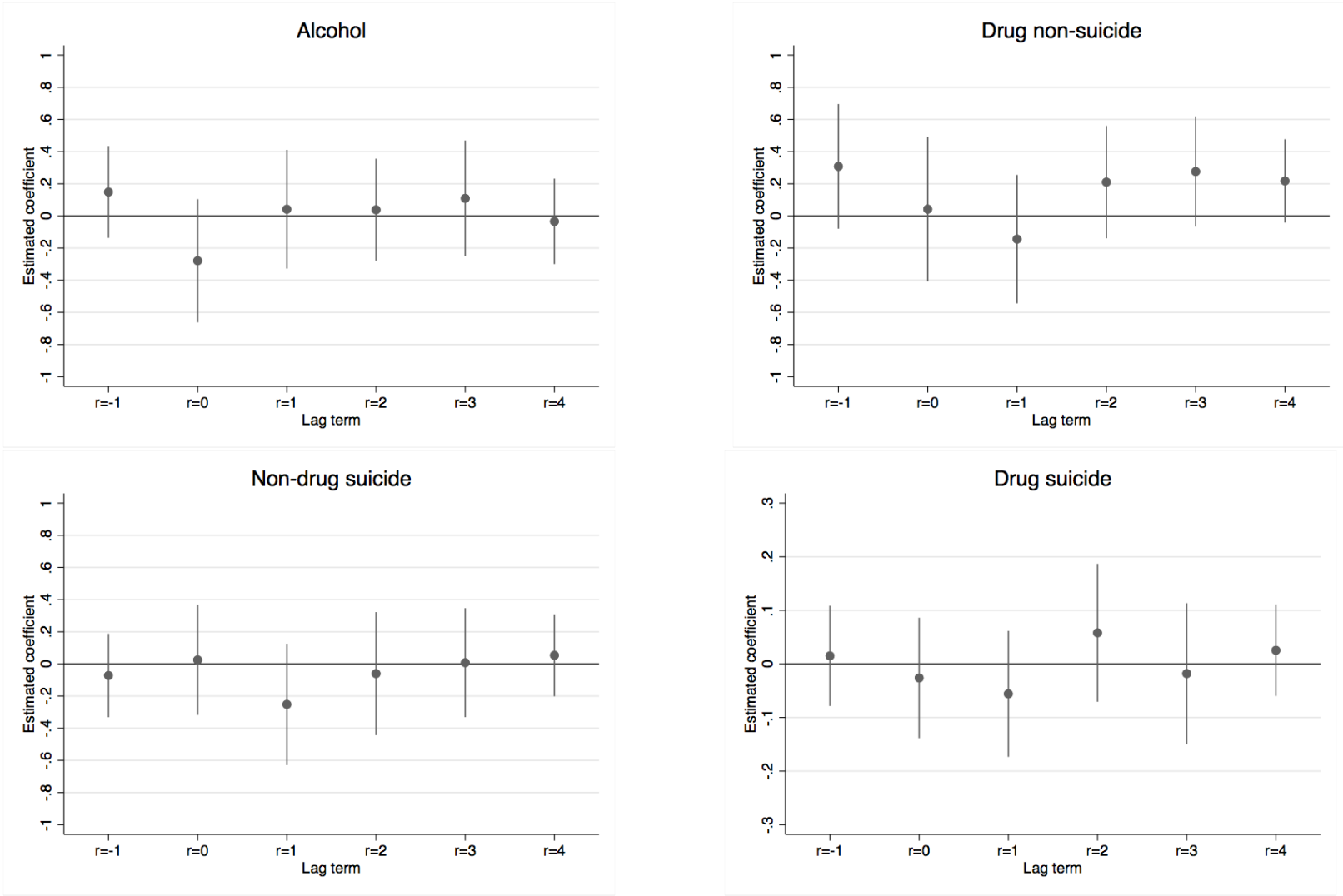
Notes: Figure presents actual and employment attributable mortality rates (per 100,000) from drug non-suicide among 19–44-year-old females (left) and 45–64-year-old males (right). Thick solid lines represent actual mortality rates. Thin solid lines represent employment-attributable rates calculated by multiplying the difference in the aggregate employment-population ratio in each year relative to 2002 by the preferred IV effect estimate from Table 3. Dashed lines represent own- and cross-group employment-attributable mortality rates calculated by multiplying the difference in each group's employment-population ratio in each year relative to 2002 by the corresponding effect estimate from Tables 4 and 5. Aggregate and own/cross-group shocks are included in figures only if found to be statistically significant in Tables 3 and Tables 4/5, respectively. Abbreviations: Agg. (Aggregate); EA (employment attributable); M, 19–44 (males, ages 19–44); and F, 19–44 (females, ages 19–44).

Figure 2.9: Magnitude of estimated aggregate, own-, and cross-group employment effects on non-drug suicide among males



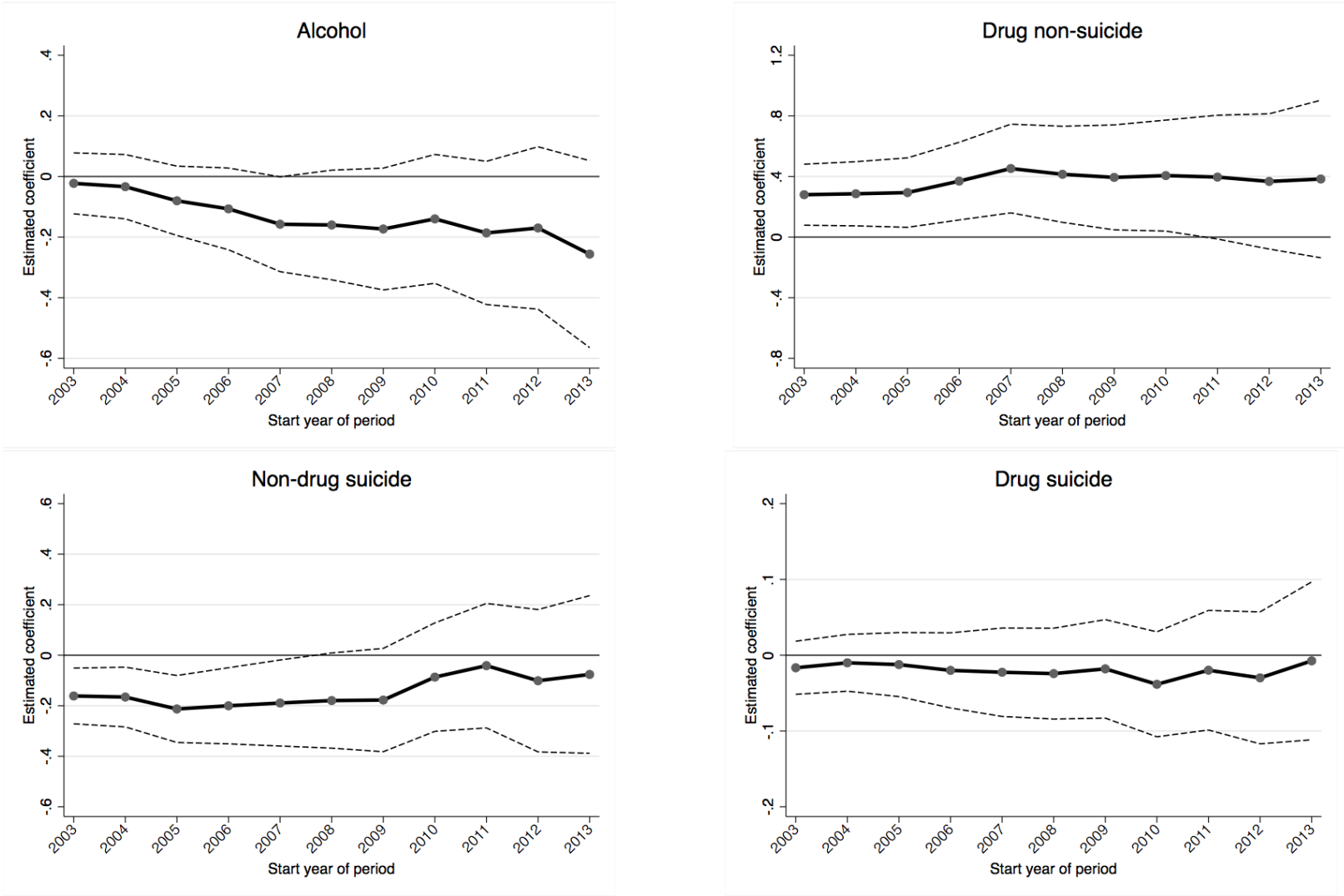
Notes: Figure presents actual and employment-attributable mortality rates (per 100,000) from non-drug suicide for 19–44-year-old males (left) and 45–64-year-old males (right). Thick solid lines represent actual mortality rates. Thin solid lines represent employment-attributable rates calculated by multiplying the difference in the aggregate employment-population ratio in each year relative to 2002 by the preferred IV effect estimate from Table 3. Dashed lines represent own- and cross-group employment-attributable mortality rates calculated by multiplying the difference in each group's employment-population ratio in each year relative to 2002 by the corresponding effect estimate from Tables 4 and 5. Aggregate and own/cross-group shocks are included in figures only if found to be statistically significant in Tables 3 and Tables 4/5, respectively. Abbreviations: Agg. (Aggregate); EA (employment attributable); M, 19–44 (males, ages 19–44)

Figure 2.10: Estimated dynamic effect of short-term employment increases on cause-specific mortality



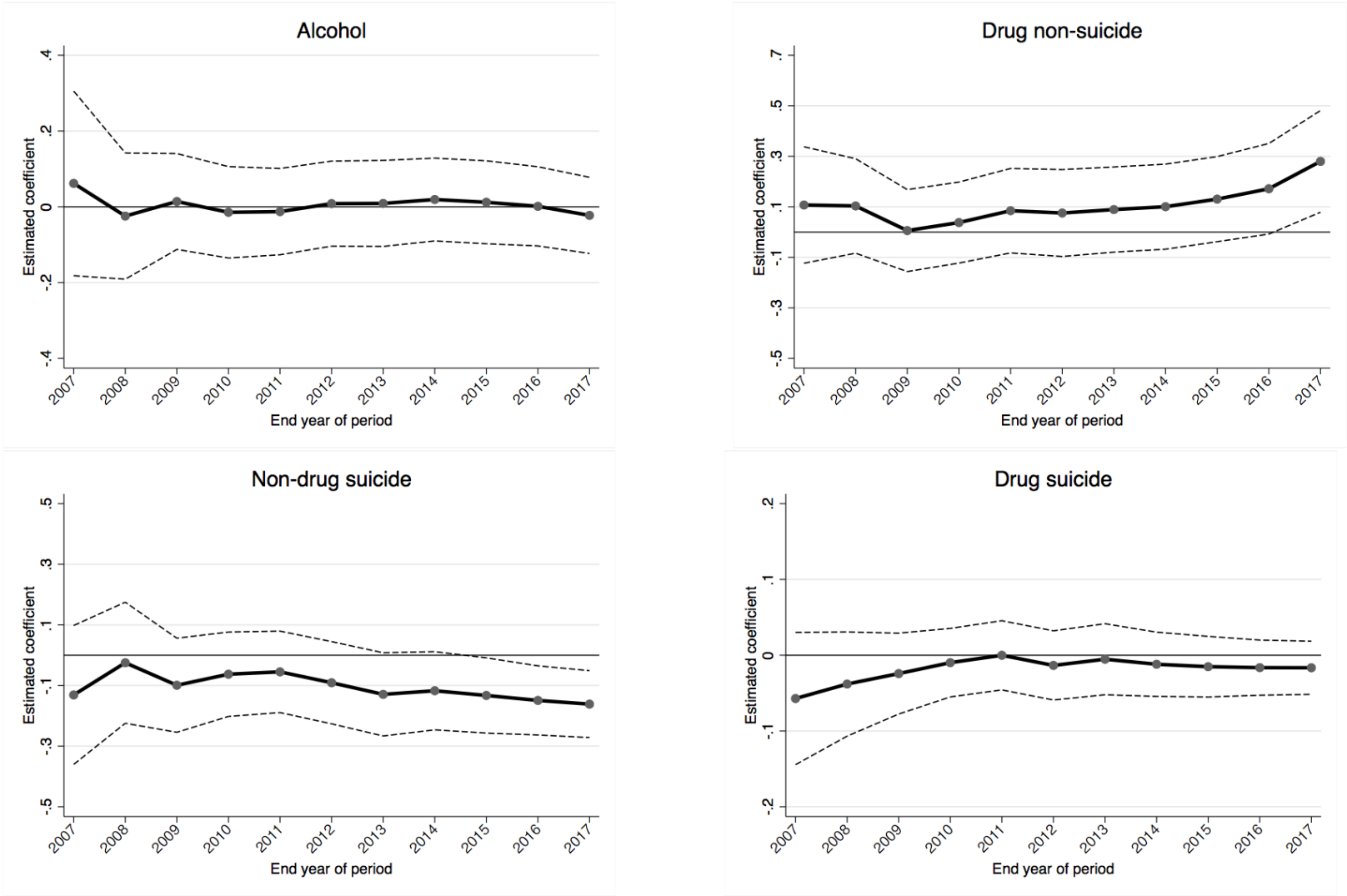
Notes: Figures display the estimated contemporaneous effect (at $r = 0$) and distribution of lead and lag coefficients (with 95% confidence intervals) from the IV model that includes a one-year lead, a contemporaneous term, and four lag terms of the aggregate shift-share instrument. The dependent variable is the cause-specific mortality rate (per 100,000) at the county-year level. Models include county, year, and state-by-year fixed effects and the county-year controls listed in Table 2. Data years include 2003–2017.

Figure 2.11: Estimated effect of aggregate employment rates on cause-specific mortality across panel start years



Notes: Figures present point estimates and 95% confidence interval bands from the IV regression of each cause-specific mortality rate (per 100,000) on the aggregate county-level employment-population ratio. All periods of analysis end in 2017, and years on the horizontal axis denote the beginning year of the period. All models control for county, year, and state-by-year fixed effects and the county-year controls listed in Table 2.

Figure 2.12: Estimated effect of aggregate employment rates on cause-specific mortality across panel end years



Notes: Figures present point estimates and 95% confidence interval bands from the IV regression of each cause-specific mortality rate (per 100,000) on the aggregate county-level employment-population ratio. All periods of analysis begin in 2003, and years on the horizontal axis denote the end year of the period. All models control for county, year, and state-by-year fixed effects and the county-year controls listed in Table 2.

Chapter 3

Do county-level employment changes affect the demand for prescription opioids? Evidence from a sample of commercially insured adults

ABSTRACT

The analysis presented in this chapter examines the relationship between county-level employment conditions and the demand for prescription opioid medication among a population of commercially insured adults in the United States. We draw on deidentified, individual-level pharmacy and medical claims from 2003–2017 aggregated to the county level to test the hypothesis that the demand for prescription opioids that present high- versus low-risk for subsequent abuse responds differentially to county-level employment fluctuations. We compare our findings to two recent studies examining the effect of county employment conditions on the demand for prescription opioids. Unlike the existing studies, we find no statistically significant evidence that county employment rates affect the overall demand for prescription opioids in our sample, and our confidence intervals rule out effect sizes as large as the point estimates from existing literature. Our smaller point estimates may be the result of estimating these effects among the commercially insured, a population that may be disproportionately insulated from the impacts of county-level fluctuations in employment conditions.[1]

[1]Note: This chapter presents results from an analysis conducted in collaboration with Anna Godøy of the Norwegian Institute of Public Health and the University of Oslo (previously at UC Berkeley), William H. Dow and Michael Reich of UC Berkeley, Pamela Morin of OptumLabs, and Henry (Joe) Henk of UnitedHealthcare (previously at OptumLabs). This project was supported by a Robert Wood Johnson Foundation Health Data for Action Grant.

3.1 Introduction

While the rapid proliferation of heroin and synthetic opioids such as fentanyl now contributes to more overdose deaths than prescription opioids (Rudd et al., 2016; Seth et al., 2018), the misuse and abuse of prescription pain relievers remains a key driver of opioid-related mortality, particularly in non-urban communities (Peters et al., 2020). According to provisional data from the Centers for Disease Control and Prevention, approximately 17,000 deaths were attributable to prescription opioids between March 2020 and March 2021, or nearly one-fifth of all drug overdose fatalities during the one-year period.[2] As discussed in detail in Chapter 2, researchers and policymakers continue to debate the degree to which the rapid acceleration of fatal drug overdose—the majority of which involve opioids—is the primary result of supply- versus demand-side factors. To the extent that these mortality trends are driven by widespread social, economic, and psychological "despair" as hypothesized by Case and Deaton (2017; 2020a) one would expect that some of these same forces that drive overdose similarly affect the demand for prescription opioids. This analysis aims to explicitly examine this "demand-side" mechanism using prescription opioid data from a large sample of commercially insured adults and proxying for economic despair with measures of local employment rates.

This analysis leverages deidentified pharmacy and medical claims data from the OptumLabs® Data Warehouse to distinguish between the potential effects of employment conditions on county-level rates of high- versus low-risk opioid prescriptions. By distinguishing between high- and low-risk prescriptions in this way, we seek to contribute to the relatively small literature on the effects of changes in employment conditions on the demand for prescription opioids (Currie et al., 2019; Musse, 2020). Relative to previous analyses using administrative datasets that define exposure to opioid prescriptions as either the number of overall prescriptions per capita (Currie et al., 2019) or milligrams of morphine equivalents (MME) per capita (Krueger, 2017), the distinction between high and low risk allows us to test a hypothesis that may better elucidate key mechanisms linking employment conditions to opioid use and adverse opioid-related outcomes.

Throughout this analysis, we loosely associate "low-risk" with prescriptions for pain medication that serve a targeted, therapeutic purpose and which are less likely to lead to adverse opioid-related outcomes, while "high-risk" encompasses prescriptions with a higher likelihood of abuse and dependence. This latter categorization likely includes many opioids intended for non-pain relief (e.g., coping or recreational) purposes. Specifically, we hypothesize that improved county-level employment conditions will be associated with higher rates of low-risk prescriptions, possibly due to higher rates of workplace injury that typically accompany economic expansions (Asfaw et al., 2011). In contrast, we anticipate that high-risk opioid

[2] Authors' calculations from provisional data provided by the Centers for Disease Control and Prevention on November 4, 2021. All natural and semi-synthetic opioids, including methadone (drug type codes T40.2 and T40.3) are included in the calculation of "prescription" opioids. All drug overdose deaths include the following cause of death codes: X40-X44, X60-X64, X85, Y10-Y14 (Centers for Disease Control and Prevention, 2021a).

prescriptions will decrease in response to improvements in the employment rate. This countercyclical effect may be driven by improved economic and social conditions, a mechanism consistent with the "deaths of despair" hypothesis.

3.2 Related literature

3.2.1 Prescription opioids and the "first wave" of the opioid addiction epidemic

The role of prescription opioids in fueling the opioid addiction epidemic has been extensively documented in academic literature (Alpert et al., 2019; Madras, 2017; Peters et al., 2020) as well as in the popular press (Macy, 2018; Quinones, 2015). This period—often referred to as the "first wave" of the opioid crisis (Seth et al., 2018)—began in the mid-1990s with the emergence and aggressive marketing of OxyContin as an alternative to existing products for pain relief, along with an increased focus among clinicians to aggressively treat chronic pain (Alpert et al., 2019; Van Zee, 2009). Between 1999 and 2010, sales of opioid pain relievers quadrupled and prescription pain relievers were involved in nearly three-quarters of all prescription drug-related deaths (Paulozzi et al., 2011). Estimates suggest that in 2003, the full economic burden of prescription opioid-related mortality and abuse in the United States totaled upwards of $78 billion (Florence et al., 2016). This amount is thought to be a vast underestimate in that it did not consider costs due to lost quality of life resulting from opioid use disorder and overdose (Florence et al., 2021).[3]

Extensive efforts to curb the over-prescription of opioids through a combination of increased physician and patient education, modified prescribing guidelines, and increased government oversight have been largely successful (Dowell et al., 2019). The abuse-deterrent reformulation of OxyContin in 2010 also contributed to a decrease in misuse of opioid prescription (Wolff et al., 2020), although some research suggests this change came with the tragic consequences of increased substitution toward heroin (Beheshti, 2019a; Evans et al., 2019). Despite an overall decrease in opioid prescriptions since 2012, high levels of prescription opioid abuse and misuse persist. In 2017, the rate of prescription opioids per capita was approximately 58 per 100, and prescription opioids contributed to over one-quarter of all overdoses involving opioids in 2019 (Centers for Disease Control and Prevention, 2021b,c).

3.2.2 Labor market conditions and prescription opioids

A rapidly growing literature examines the effects of both (1) opioids on labor supply decisions as well as (2) the effect of labor demand on opioid-related outcomes (see Maclean et al. (2020) for a review of this literature). Studies of the former have burgeoned in recent years following suggestions that the high and rising prevalence of pain and pain reliever use among prime-age

[3] A more recent analysis that includes estimated costs due to quality-of-life losses puts the total economic burden of opioids (prescription and illicit) at over $1 trillion in 2017 (Florence et al., 2021).

men may lie at the root of declining rates of labor force participation (LFP) in the United States (Krueger, 2017). For example, in a study examining changes in county LFP rates and per capita opioid prescriptions, Krueger (2017) estimated that increases in prescription pain medication may have contributed to a 20-percent decrease in LFP among prime-age men between 1999 and 2015.

Researchers have since extended this work on the labor supply implications of prescription opioids by leveraging policy-induced variation in opioid supply (Beheshti, 2019b; Deiana and Giua, 2018; Park and Powell, 2021), geographic variation in high-volume prescribers and other place-specific factors (Finkelstein et al., 2021; Harris et al., 2019), and instrumental variables shift-share approaches to isolate plausibly exogenous changes in employment demand (Aliprantis et al., 2019; Currie et al., 2019; Savych et al., 2019). The results from these studies largely converge in finding that areas exposed to decreases in prescription opioid availability tend to experience higher rates of labor force participation and employment, especially among lower-educated men.

The current analysis is primarily concerned with the latter relationship—the effect of labor market conditions on the demand for prescription opioids. In the general population, this association is theoretically ambiguous: on one hand, higher employment rates may increase work-related injuries or access to prescription opioids through employer-sponsored health insurance. In contrast, improvements in economic conditions may decrease use due to workplace policies, fear of addiction and job loss, or less using to cope with economic uncertainty consistent with the "deaths of despair" hypothesis. This paper focuses on a subset of these hypotheses though, by analyzing opioid demand among a predominantly employed population (and their dependents) with health insurance. Thus, the key mechanisms operating in this sample are potential procyclical effects due to higher work-related injury rates found during economic expansions, versus potential countercyclical effects due to generalized stress during an economic downturn.

The hypothesized procyclical effect is motivated by a well-established literature documenting increases in workplace injuries occurring during economic expansions (Asfaw et al., 2011; Davies et al., 2009; Hartwig et al., 1997; Ussif, Al-Amin, 2004) and the use of prescription opioids to treat work-related pain and injury (Kowalski-McGraw et al., 2017; O'Hara et al., 2018). Several studies using workers' compensation claims databases report increases over the past several decades in the share of all claims with opioid prescriptions for treatment of acute and chronic pain among workers (Bernacki et al., 2012). In a recent working paper, Musse (2020) estimates that a one-percent increase in the aggregate county employment rate increases the demand for prescription opioids for pain relief by 0.08 percent, while the demand for all opioids decreases by 0.20%.

The countercyclical stress mechanism, often referred to as the provocation hypothesis (Catalano, 1997; Modrek et al., 2013), posits that individuals may increase substance use in order to cope with stressful life events such job loss or perceived job insecurity. Existing research has documented the misuse of prescription opioids for such non-medical purposes in response to employment loss (Rigg and Ibañez, 2010) and higher-risk opioid prescriptions as treatments for generalized anxiety or depression (Braden et al., 2009; Sullivan et al., 2006).

Moreover, theory and empirical evidence suggest that aggregate changes in the macroeconomic environment may have spillover effects on health among individuals who remain employed during recessions or mass layoff events (Brand, 2015; Elser et al., 2019; Modrek et al., 2015). Together, this set of mechanisms aligns with Case and Deaton's "deaths of despair" hypothesis in the suggestion that Americans may have turned to opioids in part due to social and psychological pain accompanying loss of opportunity and economic decline.

Relative to the literature examining the role of macroeconomic decline or individual-level employment loss on adverse opioid-related health outcomes (e.g., overdose, opioid use disorder), studies linking these same economic factors to the *demand* for prescription opioids are scarce.[4] To our knowledge, only two studies have studied the effect of employment conditions on the demand for prescription pain relievers using prescription-level data from retail pharmacies or medical claims databases. Currie, Jin, and Schnell (2019) examine county-level employment rates on prescription opioids per capita from 2006 to 2014 and find mixed evidence with respect to the cyclicality of demand for prescription opioids. The authors estimate a negative relationship between county-level employment and opioid prescribing rates among 18–44-year-olds in higher educated counties (defined as counties with above-median shares of adults with more than a high school education) and positive effect among 40–64-year-old women in lower-educated counties (see footnote 3). Using a combination of prescription opioid and over-the-counter pain reliever transaction data from 2006 to 2012, Musse (2020) finds evidence of a negative net effect of employment increases on prescription opioids. However, Musse proceeds to decompose this effect into two distinct sources of demand, conceptualized as the "physical pain" and "substance abuse" channels. Unlike the net effect, results suggest that the demand for opioids for physical pain increases when the local employment rate increases, while the demand for opioids for "substance abuse" decreases in response to employment rate increases.

A key implication of both of these novel studies is that the overall association between employment conditions and the demand for prescription opioids may mask important nuances including heterogeneities across the population and different, often countervailing, mechanisms linking employment to opioid use. To this end, the current analysis seeks to further our understanding of the effect of local economic conditions on opioid prescription rates by examining differential effects of employment rates on prescriptions more likely to be used responsibly for therapeutic purposes (low-risk, which we hypothesize to be procyclical) from those that may increase the likelihood of adverse, opioid-related outcomes (high-risk, which we hypothesize to be countercyclical).

[4]See Chapter 2 for a comprehensive review of studies examining the effects of employment conditions on adverse opioid-related outcomes such as overdose and opioid use disorder. There is also limited research on employment conditions and self-reported use of prescription opioids. Two notable exceptions include studies by Carpenter et al. (Carpenter et al., 2017) and Aliprantis et al. (Aliprantis et al., 2019), both which use self-report data from the National Survey on Drug Use and Health. The former finds that state-level unemployment rates were associated with increased propensity of prescription pain reliever use between 2002 and 2015, while the latter finds little evidence of short-term employment shocks on changes in self-reported opioid use in response to the Great Recession.

3.3 Data

3.3.1 Analytic sample

This analysis draws on an unbalanced county-year panel dataset from 2003–2017 to examine the relationship between aggregate labor market conditions and rates of prescription opioid use among a sample of commercially insured adults. This study uses de-identified administrative claims data from the OptumLabs Data Warehouse (OLDW), which includes medical and pharmacy claims, laboratory results, and enrollment records for commercial and Medicare Advantage (MA) enrollees. The database contains longitudinal health information on enrollees and patients, representing a mixture of ages and geographical regions across the United States (OptumLabs, 2020). This dataset includes primary enrollees as well as dependents, although we are unable to distinguish between the two in our data. Previous studies using these data have noted that the study population is largely representative of the adult, commercially insured population in the United States in terms of age, gender, and race/ethnicity and is geographically concentrated in the South and Midwest (Jeffery et al., 2018; Togun et al., 2021).

We define the primary study population as all individuals aged 19–64 enrolled in a commercial health insurance plan, excluding those who meet the following criteria: simultaneously enrolled in Medicare Advantage; have unknown or conflicting gender or year of birth; evidence of malignant cancer, chemotherapy, or radiation during the past year; in hospice or palliative care (defined as any related claim in past year); or those in long-term care, skilled nursing facility, or nursing home facility for 90 days or longer. For our primary analytic sample, we further require individuals to have at least 24 months of continuous insurance coverage (12 months of continuous enrollment in the current year and 12 months in the previous calendar year) in order to restrict our sample to enrollees with no prior exposure to opioids during the prior-year washout period (henceforth referred to as "opioid naïve"). Additional details on criteria used to exclude enrollees, including specific International Classification of Diseases 9 and 10 (ICD-9 and ICD-10) diagnosis or procedure codes used to identify cancer diagnosis and treatment, are included in the appendix to this chapter.

Using deidentified prescription-level data from this sample, we construct three opioid prescription measures for individuals who filled at least one high- or low-risk opioid prescription in the current year based on the criteria described in more detail below. We aggregate observations to the county-year level and express these opioid prescription measures as rates per 100 enrollees. We then merge these data at the county-year level with employment and demographic information drawn from the Quarterly Workforce Indicators (QWI) and the Surveillance, Epidemiology, and End Results (SEER) program. This results in a 15-year county-year panel dataset consisting of approximately 47,000 observations across all 50 US states and the District of Columbia.

3.3.2 Variable construction

Among the population of opioid-exposed patients, our analysis distinguishes between patients who receive high- versus low-risk opioid prescriptions. High-risk prescriptions are defined as those that fail to meet each of the following criteria as outlined in the 2016 CDC guidelines for the safe prescription of opioids for chronic pain (Dowell et al., 2016)[5]: prescribed when the patient is not exposed to benzodiazepines; prescription is not for methadone; prescription is for short-acting formulation; prescription is for $\leq$ 50 MME per day; and prescription is for $\leq$ 7 days' supply. Any prescription for naïve opioid patients that is in accordance with all five guidelines we consider to be a low-risk prescription. Appendix A contains additional information on the criteria used to determine opioid and benzodiazepine exposure from the pharmacy claims data.

We use the employment-population ratio (EPOP) measured at the county-year level as our primary indicator of local labor market conditions. EPOP is constructed as the ratio of the employed population relative to the total working-age population, which we define in this study as all noninstitutionalized adults ages 19–64. We prefer to use EPOP rather than the county-level unemployment rate because it is less prone to measurement error at the county level, and moreover, because it is not sensitive to changes in the labor force participation rate—which changed substantially among certain populations during this time period (Leon, 1981; Lindo, 2015). We construct county-year EPOPs by aggregating county-industry-quarter employment counts from the Quarterly Workforce Indicators (QWI), a linked employee-employer database which draws on various data sources including the Quarterly Census of Employment and Wages and Unemployment Insurance benefits databases. County-level population denominators come from the SEER database's intercensal population estimates for ages 19–64. For ease of interpretation, we multiply EPOP by 100 in all models to interpret estimated regression coefficients as prescription rate changes in response to a one percentage point increase in the employment rate.

3.4 Empirical approach and model specification

Our analytic approach uses a two-way county-year fixed effects estimator, which leverages variation in employment conditions within counties over time. Our preferred fixed effects

[5]Drawing on stakeholder input and the most recent scientific evidence at the time, these recommendations were oriented around three primary considerations: "determining when to initiate or continue opioids for chronic pain; opioid selection, dosage, duration, follow-up, and discontinuation; [and] assessing risk and addressing harms of opioid use" (Dowell et al., 2016, p. 15). Research using OptumLabs data from 2016 found that just over 55% of prescription opioid fills for opioid-naïve patients met these CDC-recommended guidelines with substantial heterogeneity across US counties (Sanghavi et al., 2017). An analysis from 2018 found that high-risk prescribing practices (e.g., the use of long-acting formulas for acute pain, high-dose prescribing, and simultaneous prescribing of opioids and benzodiazepines) were already declining prior to 2016 but appeared to have declined more rapidly in the years immediately following the release of the guidelines (Bohnert et al., 2018).

estimating equation is as follows, where subscript c, s, and t denote county, state, and calendar year, respectively:

$$Y_{ct} = \theta_c + \theta_t + \theta_{st} + \beta EPOP_{ct} + \gamma \mathbf{X}_{ct} + \varepsilon_{ct} \tag{1}$$

In this equation, Y_{ct} refers to the outcome of interest (the rate of opioid prescriptions per 100 enrollees in a given county-year cell), $EPOP_{ct}$ is the employment-to-population ratio (also referred to interchangeably as "employment" or the "employment rate"), X_{ct} is a vector of time-varying controls at the county level, θ_t are year fixed effects, and θ_c are county fixed effects. The vector $\mathbf{X}_{ct}$ includes the age and gender composition of the enrollee population in each county-year as well the following measures of the demographic composition of the full working-age population in each county-year drawn from the SEER database: total share of females, share ages 19–24, share ages 25–44, and share nonwhite. Finally, our preferred model also includes state-by-year fixed effects, θ_{st}, which control for unobserved, time-varying policies at the state level that are likely correlated with both employment and opioid prescription rates or its determinants.

To explore sensitivity to various sets of controls, we estimate four separate model variants ranging from a model with county and year fixed effects only to the most saturated model that includes county, year, state-by-year fixed effects, and enrollee and county-level demographic shares. We view this latter model as our preferred specification for several reasons. First, state-by-year fixed effects control for the possibility of confounding due to the well-documented effects of state-level policies on employment rates and opioid-related outcomes, such as the presence of a must-access prescription drug monitoring programs (Buchmueller and Carey, 2018; Deiana and Giua, 2018) or state-level legalization of medical and recreational marijuana (Abouk et al., 2021; Bradford et al., 2018). Moreover, to the extent that changes in county-level or enrollee demographic characteristics are correlated with unobserved factors affecting both the demand for opioids and county employment conditions, omitting these demographic variables may bias our estimates.

As described in detail above, a well-established literature suggests an association between prescription opioid prevalence and labor supply decisions. This relationship is likely to bias the estimated effect of employment rates on opioid prescriptions (β) when estimating Equation 1 using ordinary least squares (OLS). To mitigate this possibility, we re-estimate Equation 1 using an instrumental variables (IV) shift-share approach intended to isolate demand-side variation in employment conditions. This is the same approach employed by both Currie et al. (2019) and Musse (2020) in their analyses of county-level employment on opioid prescription rates (described in more detail in Chapter 2). Specifically, we construct the instrument Z_{ct} as follows:

$$Z_{ct} = \sum_j \left(emp_{jc(2002)} \times \frac{\sum_{c' \in \{C \setminus c\}} emp_{jct}}{\sum_{c' \in \{C \setminus c\}} emp_{jc(2002)}} \right) \tag{2}$$

In the equation above, the subscripts c, t, and j index county, calendar year, and two-digit NAICS industry codes, respectively. The variable emp_{jct} is interpretable as the county-level

employment count for industry j in time t. We convert the instrument Z_{ct} to a predicted employment rate by scaling Z_{ct} by the county population in 2010 and multiplying by 100. As shown in Figure 2.3 in Chapter 2, the distribution of predicted employment rates is heavily right-skewed, and we therefore take the natural log of this quantity as the independent variable in the first stage regression. We estimate the following two-stage system using the *ivreghdfe* command in Stata 17.0 (Baum et al., 2010; Correia, 2014; StataCorp., 2021):

$$EPOP_{ct} = +\theta_c + \theta_t + \theta_{st} + \beta_1 log\Big(\frac{Z_{ct}}{pop_{c(2010)}}\Big) + \gamma_1 \mathbf{X}_{ct} + \omega_{ct} \tag{1a}$$

$$Y_{ct} = \theta_c + \theta_t + \theta_{st} + \beta_2 \widehat{EPOP}_{ct} + \theta_c + \theta_t + \gamma_2 \mathbf{X}_{ct} + \nu_{ct} \tag{1b}$$

Under the strict assumption that the instrument is orthogonal to all unobserved factors driving prescription opioid rates at the county level, β_2 will yield a causal estimate of the effect of county-level employment on the contemporaneous rate of opioid prescriptions. The feasibility of the exclusion restriction in the context of shift-share instrumental variables designs has received significant attention in the econometrics literature in recent years (Borusyak et al., 2020; Goldsmith-Pinkham et al., 2020) and is discussed in more detail in Chapter 2 (sections 3.3.1 and 6.2) and in the discussion.

3.5 Results

3.5.1 Descriptive statistics

Table 3.1 presents overall and demographic group-specific enrollment statistics for the main sample of commercial enrollees during the 2003–2017 period. In the first row, we present the county-level mean count of enrollees overall and in each demographic group, along with standard deviations in parentheses and ranges in brackets. The mean enrollment per county was around 3,000 working-age adults (ages 19–64), with nearly equal representation between males and females in each of the three age brackets (19–24, 25–44, 45–64). Figure 3.1 displays a histogram of the proportion of the total county population ages 19–64 included in the enrollment sample across all county-years. The distribution has a long right tail: the vast majority of county-year cells consist of less than 5% of the total county population, while a small share of counties have upwards of 10% of the adult working-age population represented in the sample. The second row in Table 3.1 presents the overall mean in each demographic group included in the enrollee sample as a share of the total county population in each group. The overall and group-specific means are all between 3% and 4% of the total population in each group.

Because our analysis draws on data from a sample of continuously insured enrollees in commercial health insurance plans, examining how enrollment rates change over time — and to what extent these trends may be correlated with employment conditions and prescription opioid demand — is an important exercise to gauge potential selection bias. We present descriptive trends of enrollment by three age groups (19–24, 25–44, and 45–64) and gender

in Figure 3.2. These figures compare mean enrollment shares over time across age groups for females (left) and males (right), where the vertical axis denotes the overall share of the working-age population in each age-by-sex demographic group. Enrollment as a share of county population increased throughout the 15-year period for both females (from 2.9% in 2003 to 4.1% in 2017) and males (from 3.0% to 4.4%), although this upward trend was interrupted for all demographic groups around the time of the 2007–2009 Great Recession.[6] The figures present some evidence that the enrollee share became slightly younger over time: among females, the share of enrollees in the 19–24 age group increased by 52% (relative to 38% and 43% increases among the 25–44 and 45–65 age groups, respectively), and by 66% (relative to a 46% increase and 50% increase) among males. Overall, however, there do not appear to be substantial shifts in the age-by-sex composition of the main sample of enrollees over the 15-year period.

Figure 3.3 shows 2003–2017 trends in the percent of all enrollees in the sample who were continuously enrolled in a given year, defined in this study as appearing in the sample for the current and previous year. This percentage varied between approximately 35% and 45% across the 15-year period. We note that to the extent that the enrollment patterns in Figures 3.2 and 3.3 are correlated with changes in the employment rate as well as with enrollees' propensity to demand opioid prescriptions, the regression estimates we present below will reflect a combination of these compositional changes as well as the behavioral effects of interest. We return to discuss the potential bias due to compositional effect in more detail in the discussion below.

Table 3.2 presents summary statistics for the three opioid prescription outcomes of interest, and trends in these outcomes over the study period are presented in Figures 3.4–3.6. The overall rate of any prescription opioid was approximately 14 per 100 enrollees, with rates of high- and low-risk prescriptions of 6.1 and 8.1 per 100 enrollees, respectively. Rates of overall opioid prescriptions were slightly higher among females than males (15.4 and 12.7 per 100 enrollees, respectively) and among those ages 45–64 relative to ages 19–44 (14.4 and 14.0 per 100 enrollees, respectively). For the full sample of enrollees and across all subgroups, rates of low-risk opioid prescriptions were higher than those classified as high-risk. Trends in overall and group-specific rates for all opioid prescriptions followed similar patterns across the 15-year period.

As shown in Figure 3.4, overall and high-risk opioid prescription rates increased during the 2003–2008 period (reaching a maximum of 15.9 and 7.2 per 100 enrollees, respectively) and decreased from 2009 onward. The rate of low-risk prescription opioids hovered around 8 per 100 enrollees throughout the sample period with a noticeable decrease beginning in 2011. Demographic group-specific means of high-risk prescription rates varied over time

[6]Consistent with nationwide statistics on changes in employment-sponsored health insurance following the Great Recession (Holahan, 2011), the dip in enrollment during this period was most pronounced among the youngest age groups for both females and males. Between 2008 and 2010, females aged 19–24 experienced a 12.9% decrease in enrollment relative to a 7.5% and 5.5% decrease among their 25–44-year-old and 45–64-year-old female counterparts, respectively. For males, the corresponding decreases were 13.3%, 11.4%, and 6.1%.

and reached a maximum in 2008 for all subgroups. The relative relationship in high-risk prescriptions between the groups remained relatively constant over time with higher rates among women and older working-age adults, on average, throughout the sample period (Figure 3.5). Rates of low-risk prescriptions were also consistently higher among women and 19–44-year-olds throughout the study period, although the difference between older and younger adults decreased beginning around 2011, and both age groups had similar rates by the end of the study period (Figure 3.6).

3.5.2 Estimated effect of employment conditions on prescription opioids

Table 3.3 presents estimated coefficients from separate OLS and IV regressions of the three outcomes of interest on county-level employment-population ratios. Coefficient estimates in this table are interpretable as changes in the opioid prescription rate per 100 enrollees in response to a one percentage point increase in the county employment rate. Column 1 presents estimates from the least saturated model with county and year fixed effects only, while Column 4 denotes the full model with county and year fixed effects, state-by-year fixed effects, and county and enrollee demographic controls. All four IV models appear to yield strong first stages as demonstrated by relatively large Kleibergen-Paap F statistics ranging from 97 in Model 1 to 146 in Model 4.

Three of the four OLS models (columns 1a, 2a, and 3a) yield positive and statistically significant coefficients on EPOP for all opioid prescriptions and low-risk opioid prescriptions, suggesting that the demand for prescription pain medication increases as a function of the county-level employment rate and is driven by increases in demand for low-risk prescriptions. For example, the OLS estimates across Models 1–3 suggest that a one percentage point increase in EPOP increases the demand for low-risk prescription by approximately 0.02 per 100 enrollees, or by about 0.2% relative to the sample mean of 8.05 low-risk prescriptions per 100. However, when adding state-by-year fixed effects in Model 4, the magnitude of this point estimate decreases by over 50% to 0.007 per 100 enrollees (95% CI: -0.0029, 0.017), and we fail to find any evidence of a significant effect of employment on any prescription rate outcome.[7]

On the other hand, the IV models fail to find any statistically significant effects of EPOP on opioid prescription rates. While point estimates are close to zero in all IV specifications, the models are estimated with low precision, and the associated confidence intervals are

[7]Results from Model 1 (leftmost column in Table 3.3) present the most direct comparison with the OLS estimates from Currie et al. (2019) and Musse (2020) given that the model does not include demographic controls or state-by-year fixed effects. For all opioid prescriptions, we estimate an increase of 0.0197 per 100 enrollees in response to a percentage point increase in EPOP, which corresponds to a demand elasticity of 0.08 (95% CI: 0.04, 0.12). This effect size is smaller than that estimated by Musse (0.11) but within the 95% confidence interval (0.069, 0.16). Relative to estimates by Currie et al., our estimate is noticeably smaller, as their estimates range from 0.21 (95% CI: 0.086, 0.34) for older males to 0.55 (95% CI: 0.37, 0.72) for younger females; our 95% confidence intervals rule out effects as large as those of Currie et al. for these OLS models.

consistent with a wide range of effect sizes. For example, the estimate from our preferred IV model is suggestive of a low-risk opioid prescription demand elasticity of -0.22 with respect to employment, yet we cannot rule out elasticities as small as -0.59 or as large 0.18 at the 5% level.[8] We obtain a smaller but more precise estimate of the effect of employment on all opioid prescriptions (estimated effect = -0.0092 per 100 enrollees, 95% CI: -0.041, 0.022), corresponding to a demand elasticity of -0.04 (95% CI: -0.17, 0.09).

This latter result serves as the best point of comparison between our preferred IV results and those from the existing literature, as both Musse (2020) and Currie and et al. (2019) present estimated elasticities for all opioids without distinguishing between high- and low-risk prescriptions. Notably, the demand elasticity we estimate for all opioid prescriptions (-0.04) is estimated with far less precision than that calculated by Musse (2020), who calculates an overall demand elasticity of -0.20 (95% CI: -0.18, -0.21). While we cannot directly compare our estimates to those from Currie et al., as the authors present all IV estimates stratified by age group, gender, and educational attainment of the population, our estimated demand elasticities are substantially smaller in magnitude for all statistically significant findings presented in their paper. For example, Currie et al. estimate demand elasticities of -1.1 (95% CI: -2.4, -0.09) and -0.80 (95% CI: -1.5, -0.20) for 20–39-year-old females and males (respectively) in counties with high educational attainment. The lower bound of the 95% confidence interval of our estimate is -0.17, which is within the 95% confidence interval for Currie and colleagues' estimate for females but not males, but our point estimate (-0.04) is outside the bounds for both groups.[9] Overall, our confidence intervals rule out effect sizes as large as the point estimates in either Musse or Currie and colleagues.

3.6 Discussion

Unlike findings from previous studies (Currie et al., 2019; Musse, 2020), our instrumental variable shift-share models do not indicate that increases in the county-level employment rate are associated with statistically significant changes in prescription opioid rates. One strength of our approach is that our use of the shift-share instrument should better ensure that our models estimate effects of employment on opioid prescription rates by leveraging variation in demand-side employment changes (i.e., purging the potentially confounding effect of supply-side effects that may be correlated with prescription rates). A second strength of our analysis is that we explore whether this null effect is the result of countervailing behavioral responses

[8]The demand elasticity for low-risk prescription opioids is calculated using the estimated IV point estimate from column 4 as follows: (-0.03/8.10)/(1/59.6), where 8.10 is the mean rate of low-risk opioid prescriptions per 100 enrollees, and 59.6 is the mean employment rate during the sample period. Other elasticities from the point estimates in Table 3.3 are calculated similarly.

[9]The 95% percent confidence intervals for the estimated elasticities in the referenced papers are not included in papers themselves but calculated by the authors based on the point estimates and standard errors from the log-log models presented in Table 2 (Musse, 2020, p/ 31) and Table 5 (Currie et al., 2019, n.p.).

to an exogenous increase in employment demand: increases in low-risk opioids such as for work-related injuries but decreases in high-risk opioids related to despair.

On one hand, work-related injuries may increase with employment, leading to more prescription opioids for therapeutic purposes. This mechanism is consistent with the findings from Musse (2020), who finds that the demand for prescription opioids for pain relief is procyclical, as well as from a recent study showing that employment in the mining industry is associated with increased opioid overdose mortality (Metcalf and Wang, 2019). Extraction occupations, along with occupations in other industries with high rates of physical injury such as construction, accommodation and food services, and healthcare support, tend to have high rates of adverse opioid-related outcomes (Shaw et al., 2020). Given that we do not know the industries or occupations of the employees in this sample, it is unclear the extent to which employment increases may increase demand for opioids through this channel.

However, these procylical effects may be offset by mechanisms that decrease (or increase) the demand for prescription opioids as employment conditions improve (or worsen). The most obvious is through the economic instability, psychological distress, and social isolation that come with unemployment, all of which are elements of "despair" hypothesized to increase the demand for prescription pain medication (Case and Deaton, 2017, 2020a). If decreases in employment demand at the county level are indicative of broader macroeconomic contraction, austerity measures may reduce expenditures for substance abuse treatment services, which are largely funded by state and local governments (Buck, 2011). In turn, this may increase demand for prescription opioids via reduced access to addiction treatment and recovery services (Hodgkin and Karpman, 2010; Modrek et al., 2013). However, while such reductions in treatment access are a hypothesized mechanism underlying many of the studies finding countercyclical variation in adverse opioid-related outcomes (e.g., Carpenter et al. 2017, Hollingsworth et al. 2017), empirical studies of the effect of business cycle fluctuations on treatment utilization for substance use disorder are mixed, possibly reflecting counteracting changes in both the supply and demand of services (Cantor et al., 2017).[10]

The lack of any significant effects of county employment rates on prescription opioids rates in the present analysis for either low-risk or high-risk opioids may also be due to fundamental differences in the underlying study population as compared to other studies. Unlike Musse (2020) and Currie et al. (2019), both of whom use retail data from the general population to construct county-level opioid demand measures, the current analysis is restricted to commercially insured enrollees only. Therefore, it is plausible that a large share of individuals in our sample are unaffected directly by changes in the labor market, and the effect we estimate is not the direct result of within-sample employment changes

[10] A priori, the effect of an economic downturn on the equilibrium quantity of substance use treatment services is ambiguous. If the demand for treatment services increases while the supply of such services decreases, the effect on the quantity of treatment (e.g., treatment admissions) will depend on the relative magnitudes of these changes. However, given that fewer than half of adults report having substance abuse treatment covered by their insurance (Buck, 2011), it is also possible that aggregate demand will decrease if individuals are forced to pay out of pocket. This would result in an unambiguous decrease in the quantity of treatment services, assuming that supply also decreases.

but rather the aggregate effects of changes in the labor market on population-level health outcomes.

It is also noteworthy that the preferred models from both Musse and Currie et al. control for quarter-year and quarter fixed effects (respectively) but do not include state-by-year fixed effects or county-level demographic controls. This may explain some of the discrepancy between our estimates and those in the existing literature, although the estimated demand elasticity in our model with county and year fixed effects only (0.07, 95% CI: -0.08, 0.23) is still well outside the 95% confidence bounds calculated from the preferred estimates in Musse and Currie.

3.6.1 Study limitations

Because this analysis draws on medical and pharmacy claims data from a population enrolled in commercial health insurance programs, and we further restrict the analytic sample to continuous enrollees, it is possible that compositional shifts in our sample over time may introduce selection bias. This bias would occur if local labor market conditions change the probability of appearing in our sample in a way that is correlated with propensities for opioid prescriptions. We are limited in individual-level characteristics that would allow us to examine this potential threat to identification in a rigorous way, although controlling for the age and gender composition of the sample may help mitigate any potential bias introduced by the share of younger enrollees increasing over time (see Figure 3.2).[11]

Meanwhile, there are several well-known risk factors for patients developing opioid use disorder or dependence (conditional on exposure to opioids) that we are unable to control for, such as an individual's family history of substance use disorder, concurrent alcohol use disorder, or mental health comorbidities (Dowell et al., 2016). In order to bias our results, however, employment conditions would have to affect higher-opioid-propensity individuals (i.e., those employees using opioids for reasons unrelated to employment conditions) such that they are disproportionately selected into or out of our sample. Given the difficulty in employers' observing opioid propensity (e.g., when hiring), we suspect that any such bias would be swamped by changes in the employee mix that are part of the causal effect of interest, such as higher injury rates that are well-documented to occur during expansions as a result of adding less experienced employees. Nonetheless, we cannot rule out the possibility that the point estimates we report above may reflect a combination of our hypothesized mechanisms along with some selection on opioid propensity, though we consider the potential bias introduced by the latter to be small.

[11]For example, selection bias due to the age composition of the enrollee pool may occur if employers are more likely to shed younger workers during economic contractions (Rodriguez and Zavodny, 2003). Research suggests that despite being less likely to be prescribed opioids (Schieber et al., 2020), younger adults face a higher risk of opioid misuse or abuse relative to older adults conditional on opioid exposure (Kaye et al., 2017). This relationship may introduce a downward bias in the estimated relationship between employment conditions and the overall demand for opioids while inducing an upward bias in the estimated demand for high-risk opioids.

The present analysis is concerned with only one direction of the likely bidirectional causal relationship between prescription opioids and labor market outcomes. The extensive literature on the labor supply effects of opioids has important implications for the present analysis in that it underscores the need to separate plausibly exogenous sources of variation in labor demand in order to identify causal effects of employment conditions on the demand for prescription opioids. Despite the use of the shift-share instrument to isolate demand-side changes in employment, it is possible that the variation we use to identify causal effects may still be contaminated by unobserved time-varying factors affecting employment and prescription opioid use. Given the set of fixed effects we include to control for potential time-invariant confounders, the identifying assumption in the present analysis contends that the industry shares are exogenous to the time-varying component of the error term in equation (1).

Specifically, the presence of any time-varying factor correlated with the industry mix at baseline and changes in the demand for opioid prescriptions would violate the exclusion restriction (Goldsmith-Pinkham et al., 2020). Appendix Table A.1 in Chapter 2 demonstrates significant correlations between initial industry shares in 2002 and changes in observed county-level characteristics over the study period, although the inclusion of these variables does not substantially alter the point estimates in the IV models (see Table 3.3). Nonetheless, we are unable to rule out the possibility that there may exist additional time-varying factors that we do not account for that could undermine the validity of the shift-share instrument.

3.6.2 Conclusions

In their most recent paper on the topic, Case and Deaton respond to arguments positing that social and economic despair merely exacerbated a pre-existing, supply-driven opioid crisis (an order of events proposed by Maclean et al. 2020, and others) by reiterating their view of a "demand driven epidemic, propelled by despair, in which pharma companies did little more than meet rising demand" (Case and Deaton, 2021). In the analysis presented above, we attempt to empirically test this claim by estimating the effect of county-level employment rates on the demand for prescription opioids. Recognizing the complexities underlying the relationship between economic conditions and prescription opioid demand, we seek to separate the potentially differential effects of employment changes on the demand for high- versus low-risk prescriptions. We find no evidence of an overall or differential effect of county-level employment rates on the demand for prescription opioids, although we acknowledge that our estimates are relatively imprecise and cannot rule out substantial positive or negative effects.

Moreover, we recognize that the degree to which our sample population is composed of the population at the root of Case and Deaton's "despair" argument—Americans without a college degree—is unknown but likely small. For example, it is well known that the education distribution of privately insured adults in the United States is skewed toward the higher-educated. Despite constituting only 30% of the adult population ages 25–64 in the United States, adults with at least a Bachelor's degree make up nearly 40% of all similarly aged adults with private health insurance coverage. In contrast, nearly 40% of all adults ages

25-64 have a high school diploma or less, yet this group represents only 30% of the privately insured population. Among adults with health insurance, over 97% of adults with at least a bachelor's degree have private insurance compared to only 76% of insured adults with no college education.[12]

Future studies examining the effect of employment conditions on the demand for opioids should focus on populations most likely to have experienced declining economic and social capital over the past several decades. Finally, while the aim of this study is to better elucidate potential mechanisms underlying the "deaths of despair" narrative in the context of prescription opioid overdose, this study is unable to directly link changes in opioid prescription prevalence with adverse health outcomes at the population level. Higher rates of opioids for medical purposes increase the risk of adverse outcomes among patients (Monnat, 2019) as well as potential diversion through family and social networks for non-medical, recreational use (Powell et al., 2020). Future research should prioritize examining the degree to which prescription opioid demand acts as an intermediary outcome linking employment conditions to increases in opioid dependence, abuse, and overdose.

[12]Data come from authors' calculations of 2012 American Community Survey 5-year Estimates (US Census Bureau, 2012).

Table 3.1: Characteristics of the primary sample

		Female enrollees			Male enrollees		
	Full sample	**19-24**	**25–44**	**45–64**	**19–24**	**25–44**	**45–64**
# of enrollees per county	2998.18 (11,404) [0; 276,126]	181.92 (699.22) [0; 18,519]	726.57 (2,921.31) [0; 71,219]	589.97 (2,192.00) [0; 55,159]	183.43 (683.13) [0; 17,505]	742.25 (2,901.49) [1; 69,327]	586.36 (2,111.75) [0; 50,534]
Share of total county pop.	3.66 (3.67) [0; 61.33]	3.91 (4.37) [0; 91.34]	3.83 (3.90) [0; 61.68]	3.46 (3.79) [0; 50.34)	3.85 (4.20) [0; 90.00)	3.97 (3.76) [0.03; 62.06]	3.55 (3.58) [0; 55.23]

Notes:Table displays the mean number of enrollees per county-year in the full sample of commercial enrollees (row 1) and the mean share of enrollees as a proportion of the total county-year population (row 2), overall and by demographic group. The primary analytic sample consists of all commercially insured enrollees who meet the initial inclusion criteria described in section 3.1. Standard deviations and ranges are presented in parentheses and brackets, respectively. Data come from 3,152 unique counties over the 2003–2017 period.

Table 3.2: Mean county-level rate of prescription opioids among continuously enrolled, commercially insured sample

	Full sample	**Female**	**Male**	**Age 19–44**	**Age 45–64**
Any opioid Rx	14.16 (2.94)	15.35 (3.41)	12.73 (3.03)	13.95 (3.34)	14.41 (3.03)
Any high-risk Rx	6.10 (1.76)	6.81 (2.15)	5.29 (1.96)	5.58 (1.90)	6.71 (1.98)
Any low-risk Rx	8.10 (1.75)	8.60 (2.10)	7.48 (1.96)	8.41 (2.17)	7.75 (1.86)
No. county-year obs.	46,776	46,673	46,720	46,511	46,487

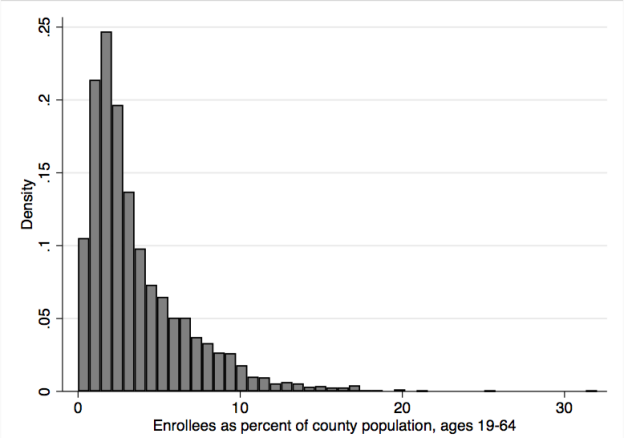
Notes: Table displays overall county-year mean rates per 100 enrollees and standard deviations (in parentheses) of each outcome among all continuously enrolled enrollees. County-level means are averaged across all 2003–2017 years.

Table 3.3: Estimates from OLS and IV regressions of opioid prescription shares on employment-population ratio

	(1a) **OLS**	(1b) **IV**	(2a) **OLS**	(2b) **IV**	(3a) **OLS**	(3b) **IV**	(4a) **OLS**	(4b) **IV**
All opioid Rx	0.0197**	0.0172	0.0202***	0.0125	0.0174***	0.00903	0.00567	-0.00916
(mean = 14.2)	(0.00511)	(0.0196)	(0.00497)	(0.0192)	(0.00491)	(0.0181)	0.00346)	(0.0161)
High-risk Rx	0.00237	-0.00790	0.00234	-0.00702	0.000288	-0.0117	0.00116	-0.0206
(mean = 6.10)	(0.00521)	(0.0212)	(0.00514)	(0.0212)	(0.00562)	(0.0212)	(0.00452)	(0.0201)
Low-risk Rx	0.0219***	0.00714	0.0224***	0.00325	0.0176***	-0.00404	0.00697	-0.0302
(mean = 8.05)	(0.00555)	(0.0275)	(0.00555)	(0.0273)	(0.00558)	(0.0264)	(0.00502)	(0.0260)
Cty+year FE	Y	Y	Y	Y	Y	Y	Y	Y
Cty demog.	N	N	Y	Y	Y	Y	Y	Y
Enrollee demog.	N	N	N	N	Y	Y	Y	Y
State-by-year FE	N	N	N	N	N	N	Y	Y
First stage F-stat.		96.54		102.7		103.5		145.8
Observations	46611	46556	46559	46508	43449	43398	43449	43398

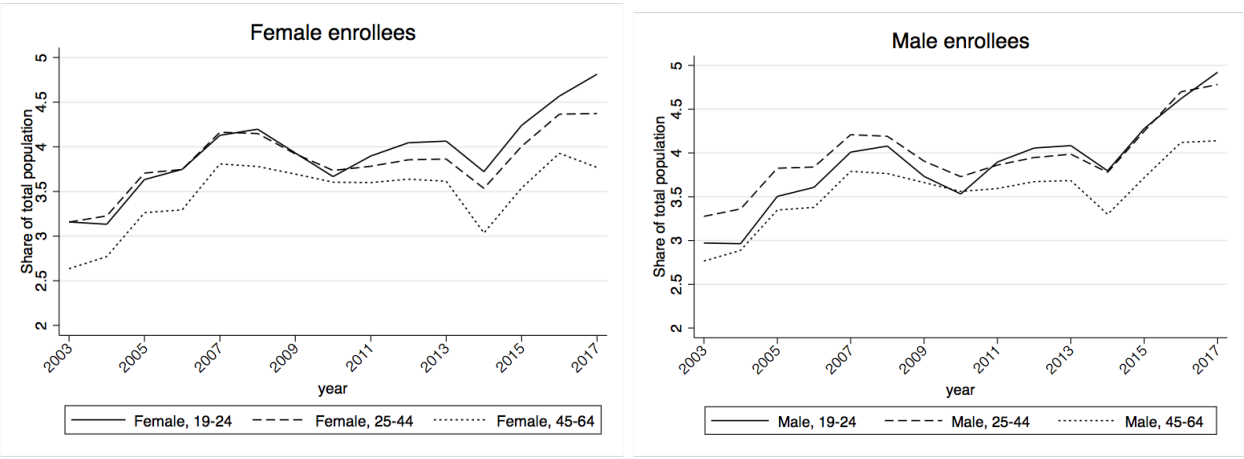
Notes: Table displays estimated coefficients (and standard errors clustered at the county level in parentheses) from regressions of the share of continuously enrolled and commercially insured adults with at least one naïve opioid prescription on the county-level employment-population ratio (EPOP). The independent variable in the OLS models is the employment-population ratio, which is instrumented using the predicted employment-population ratio in the IV models. The dependent variable is the rate of prescription opioids per 100 continuously enrolled adults in each county-year. The mean of the independent variable (EPOP) is 59.6%. County demographic controls include the share of the working-age population in the following demographic groups: female, male, ages 19–44 and ages 45–64. Enrollee demographic controls include the share of enrollees in each of the following demographic groups: female 19–24, female 25–44, female 45–64, male 19–24, male 35–44, male 45–64. Each regression is weighted by the county population ages 19–64. Significance: * p<0.10, ** p<0.05, *** p<0.01

Figure 3.1: Distribution of mean county-level enrollment, as a share of total county population



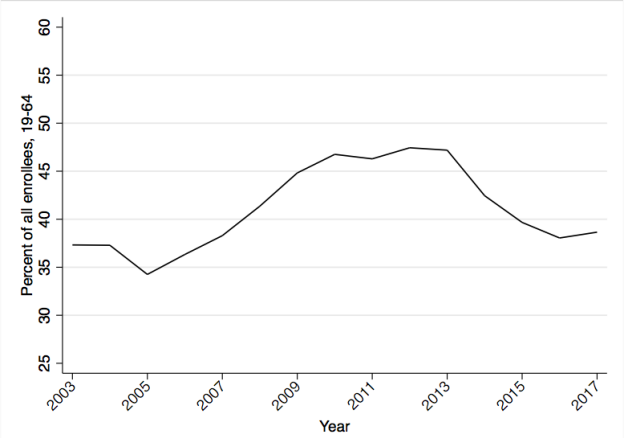
Notes: Figure shows distribution of enrollment as a share of the total working-age population at the county level from 2003–2017. All observations are defined at the county-year level.

Figure 3.2: Mean county-level enrollee share, 2003–2017, by gender and age-group



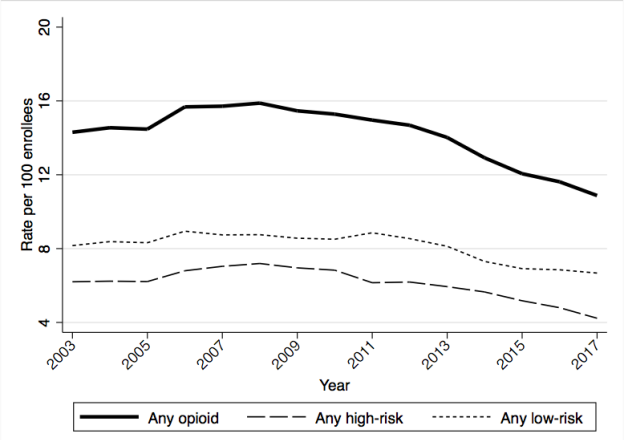
Notes: Figure shows trends in demographic group-specific enrollment as a share of the total working-age population in each subgroup, from 2003–2017. Lines display unweighted yearly means across all county-year cells.

Figure 3.3: Mean county-level continuous enrollee share, 2003–2017



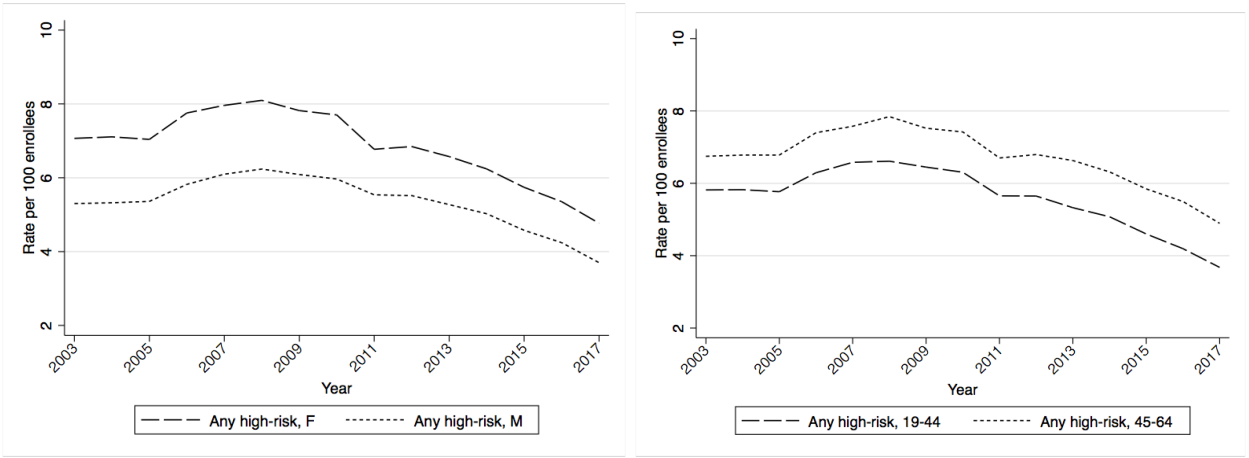
Notes: Figure shows trends in continuous enrollees as a share of the total number of county-level enrollees in each year from 2003–2017. The denominator is restricted to enrollees ages 19-64 who meet the initial inclusion criteria defined in section 3.1. Line displays unweighted yearly means across all county-year cells.

Figure 3.4: Rates of any, high-risk, or low-risk opioid prescription among continuously enrolled sample



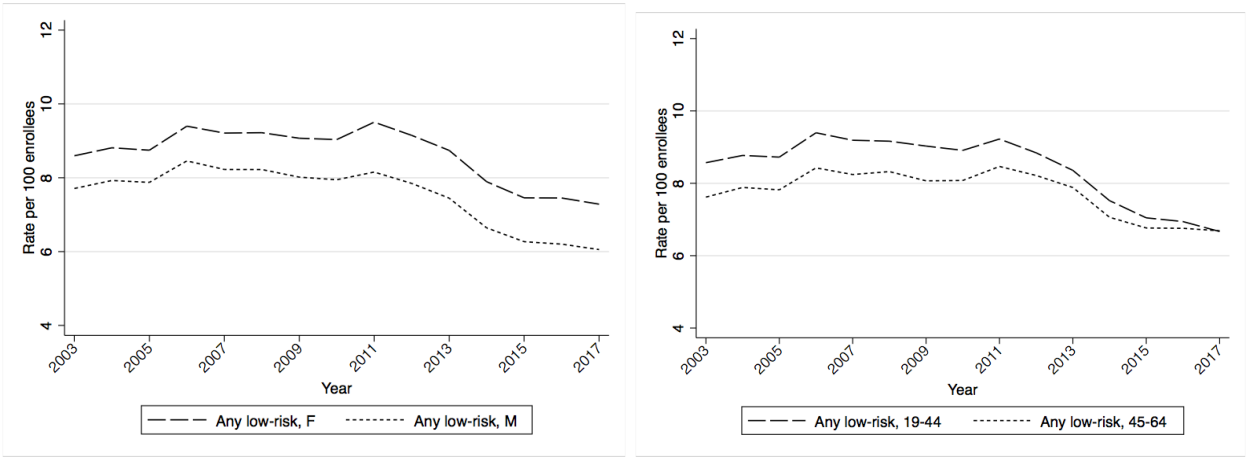
Notes: Figure shows yearly mean rates per 100 enrollees with any opioid prescription, any high-risk opioid prescription, and any low-risk opioid prescription among the analytic sample of all continuously enrolled commercial enrollees. Rates are calculated based on naive opioid prescriptions (i.e., no opioid exposure within 12 months of opioid claim). Lines connect yearly mean rates weighted by the total number of enrollees in each county-year cell.

Figure 3.5: Rates of any high-risk opioid prescription by demographic group among continuously enrolled sample



Notes: Figures show yearly mean rates per 100 enrollees with any high-risk opioid prescription among the sample of all continuously enrolled commercial enrollees ages 19–64. Rates are calculated based on naive opioid prescriptions (i.e., no opioid exposure within 12 months of opioid claim). Lines connect yearly mean rates weighted by the total number of enrollees in each county-year-demographic group cell.

Figure 3.6: Rates of any low-risk opioid prescription by demographic group among continuously enrolled sample



Notes: Figures show yearly mean rates per 100 enrollees with any low-risk opioid prescription among the sample of all continuously enrolled commercial enrollees ages 19–64. Rates are calculated based on naive opioid prescriptions (i.e., no opioid exposure within 12 months of opioid claim). Lines connect yearly mean rates weighted by the total number of enrollees in each county-year-demographic group cell.

Chapter 4

Discussion

Policymakers and researchers across a broad array of disciplines have sought to understand the root causes of and effective policy responses to the recent reversal in life expectancy in the United States—a trend largely driven by increases in mortality from drugs, suicide, and alcohol-related causes. This dissertation presents two sets of analyses to explore the extent to which local labor market conditions may be contributing to these disconcerting trends. This work contributes to a large and well-established literature on economic conditions as a key determinant of health, and more specifically, to a rapidly growing literature exploring the role of employment conditions as potential drivers in the midlife mortality crisis in the United States. In this concluding chapter, I begin by summarizing the key conclusions from the empirical analyses in Chapters 2 and 3. I then discuss the public policy implications of these results and important future directions for research examining labor market policies, employment conditions, and the rise in midlife mortality due to these causes of death. I conclude with a brief discussion of these findings in the context of the COVID-19 economic crisis.

4.1 Summary of key findings

The goal of the empirical analyses in Chapters 2 and 3 is to develop a more complete understanding of the role of local labor market conditions as determinants of rising mortality from drug, suicide, and alcohol-related causes. Chapter 2 presents evidence that a one percentage point increase in the current-year local employment rate (the proxy for local economic conditions used throughout this dissertation) decreases the rate of drug non-suicides among working-age adults by just over one percent according to the preferred model. In contrast, the same increase in employment increases the rate of drug non-suicide rates by a similar magnitude (1.2%). Using these point estimates and the change in employment relative to 2002 to construct "employment attributable" mortality rates for each cause of death, I show that in general (and especially for drug non-suicide), these estimated effects are small relative to the increases in suicide and drug-related mortality that took place over the 2003–2017

period.

Results from Chapter 2 also point to potential age and sex heterogeneity in this contemporaneous effect. Consistent with existing research, I present suggestive evidence that older males are especially vulnerable to the adverse effects of changes in employment conditions on suicide, and this adverse effect may reflect a combination of employment changes among their own demographic group and possibly spillover effects from other younger males. The positive effects of employment on drug non-suicide rates appear to be concentrated among younger adults and also may reflect a spillover effect from employment changes in other segments of the labor market. In general, however, these subgroup-specific estimates are not robust to correcting for multiple hypothesis testing and should be viewed as exploratory results to be probed in future research. Likely due to the short duration of my panel, I am unable to draw conclusions regarding the dynamics of these effects over the short- to medium-term, nor am I able to isolate differential effects on cause-specific mortality resulting from short-term fluctuations versus prolonged periods of economic decline.

Chapter 3 explores potential mechanisms underlying the procyclical pattern in drug non-suicide rates uncovered in Chapter 2 by attempting to distinguish between the effects of employment on the demand for high- versus low-risk prescription opioids. The analysis in Chapter 3 attempts to identify the potentially differential role of employment conditions as a contributing factor in the use of prescription opioids for pain relief versus as a means for coping with economic distress. Unlike in existing studies, we find no statistically significant evidence that county employment rates affect the overall demand for prescription opioids in our sample, nor do we find any evidence that there is a differential effect based on the opioid prescription risk-type. However, we note that these results may be largely a reflection of estimating these effects among a sample of commercially insured adults, who may be less impacted by county-level fluctuations in employment conditions relative to other adults in the labor force.

4.2 Policy implications

The many mechanisms through which employment may improve health provides a road-map for policymakers to leverage work-related policies to improve health and promote health equity. The consistent finding in Chapter 2 that non-drug suicide rates decrease during economic expansions suggests that labor market policies that connect job seekers to employment opportunities could substantially reduce rates of suicide during economic downturns. As the economy becomes increasingly automated and the demand for lower-skilled labor decreases, policies that help workers develop new skills to be competitive in the modern economy will become increasingly important. As described in Chapter 2, active labor market policies – which include programs that offer educational or skills training, job-search assistance, or subsidized employment (Card et al., 2018)—were among the most effective public policy approaches to combating the increase in suicide rates in Europe following the Great Recession (Stuckler et al., 2009). Results from a recent survey administered by the OECD documents

that active labor market programs are being being widely implemented to stimulate labor demand in response to the COVID-19 economic crisis (Organisation for Economic Cooperation and Development, 2021).

However, policies that increase employment may not be unambiguously health enhancing, as evinced by the procyclical variation in drug-related causes of death uncovered in Chapter 2. Balancing the potential benefits and costs of such policies from a health perspective will require more nuanced analyses of the mechanisms through which employment affects health outcomes—an important extension of the reduced form analyses presented in this dissertation. For example, if the active labor market policies described above lead to increases in employment in industries or occupations with high injury risk, substance-related mortality may increase via procylical mechansisms such as increased utilization of highly addictive opioids for pain management. However, these potential adverse health effects could be mitigated by combining labor market policies with enhanced supply-side policy approaches to reduce the over-prescription of addictive opioids and improve access to addiction treatment and rehabilitation.

To the extent that the benefits (or harms) of employment on health operate primarily through the positive (or negative) effects of higher incomes, policies that increase earnings among employed workers may also play an important role. Policies that target low-wage workers may be of particular salience given that the recent increases in drug, suicide, and alcohol-related mortality are highly concentrated among working-age adults with less than a bachelor's degree, and this population is most likely to be employed in low-wage occupations. Recent studies have consistently found reductions in suicide mortality in states with a more generous minimum wage and evidence that this protective effect is driven by reductions in suicide among lower-educated individuals Dow et al. (2020); Kaufman et al. (2020). Relatedly, wage increases have been shown in previous research to be associated with reductions in opioid overdose fatalities among individuals in lower-skilled industries (Betz and Jones, 2018).

Expansions in other income support policies such as the Earned Income Tax Credit (EITC) have also been linked with improvements in adult mental health outcomes and reductions in deaths of despair (Boyd-Swan et al., 2016; Dow et al., 2020; Evans and Garthwaite, 2014; Shields-Zeeman et al., 2021). For example, Dow et al. (2020) find that a 10% increase in state EITCs reduced the rate of non-drug suicide among lower-educated adults by nearly 3%. In addition to the income effect in the form of a tax credit, the EITC is designed to incentivize employment, thereby potentially improving health via the procyclical mechanisms independent of income (e.g., increased sense of purpose, social engagement). However, this positive labor supply effect may be partially offset as employment gains following EITC expansions are concentrated in low-wage industries, where workers may be at the highest risk of experiencing workplace injury, unpredictable scheduling, or increased stress associated with low-wage work.

The emerging evidence connecting the rise of precarious work (e.g., increased job insecurity, schedule instability, lack of workplace supports and paid benefits) to increases in despair-related health outcomes points to another suite of policy tools that policymak-

ers should consider to combat rising midlife morbidity and mortality (Benach et al., 2014; Berkman and Kawachi, 2014). For example, leveraging detailed death certificate data from deceased Massachusetts residents from 2011 to 2015, Hawkins et al. (2019) find that opioid-related overdose rates were significantly higher among individuals in occupations with less access to paid sick leave and higher degrees of job insecurity. Policies that provide paid sick leave and other forms employment protection have become increasingly important tools for protecting the health of workers in the COVID-19 era, where the virus itself as well as increased care-taking responsibilities may disproportionately hurt workers in precarious employment arrangements. Moreover, policies that facilitate the formation of unions may also improve health outcomes among workers (Leigh and Chakalov, 2021), and higher union density has been associated with lower rates of deaths of despair in several recent studies (DeFina and Hannon, 2019; Eisenberg-Guyot et al., 2020).

4.3 Other dimensions of local economic conditions

All analyses presented in this dissertation use the county-level (or commuting zone-level) employment-to-population ratio as the only indicator of local economic conditions. As described in Chapter 2, there are many reasons to expect changes in the employment rate to directly affect mortality through a combination of income and time-use mechanisms, as well as via workplace exposures, policies, and the psychological effects of attachment to work. The nature of employer-sponsored health insurance may also induce a mechanical relationship between employment and health. However, there are additional dimensions to work that may be of equal or greater importance when explaining the complex relationship between employment conditions and mortality from drugs, suicide, and alcohol-related causes. Considering these competing or synergistic aspects of employment will be critically important in designing policies to mitigate the potential harms of macroeconomic decline on health.

Within the domain of employment, changes in wages have been shown in previous research to be associated with opioid overdose fatalities (Betz and Jones, 2018), and as described above, several studies have documented how states with higher minimum wages tend to experience lower rates of suicide (Dow et al., 2020; Gertner et al., 2019; Kaufman et al., 2020). Recent research has also focused on how the nature of work has changed in ways that may have implications for population health (Benach et al., 2014). The manufacturing industry, for example, has become far less labor intensive and more highly skilled in the past several decades, leading to a shift in the composition of the workforce and decreased job security among lower-educated workers (Case and Deaton, 2017; Charles et al., 2019). As described in Chapter 2, perceived job insecurity—even among those who are employed—may have adverse effects on workers' psychological and behavioral health (Burgard et al., 2009; Elser et al., 2019; Modrek et al., 2015). Another important dimension is temporal, characterized by a recent increase in employer flexibility resulting in schedule instability and unpredictability for workers. Recent studies using survey data from US workers in the retail sector finds that exposure to such work-schedule instability is associated with a variety of negative health

outcomes, including increased psychological distress (Schneider and Harknett, 2019).

Considering this broad array of employment and work-related factors that affect health, it is unsurprising that employment is only one of the many measures of local economic conditions that have been linked with deaths of despair. For example, Brown and Wehby (2019) find that declines in local housing prices are stronger predictors of opioid overdose fatalities than the unemployment rate, a relative effect the authors attribute to a "theoretically clearer mechanism for an increase in opioid use and overdosing that likely affects a wider segment of the population" (p. 474). Relatedly, Jou et al. (2020) find that increases in housing wealth significantly decrease the rate of drug-related overdoses at the county level. Knapp et al. (2019) draw on a composite measure of economic security consisting of county-level labor force participation, unemployment, average share of income spent on rent, share with subprime credit ratings, and employment in service occupations, which the authors find to be consistently associated with increases in rates of mortality due to drugs, suicide, and alcohol. Dimensions of economic "despair" have also been proxied using area-level measures such as decreases in economic and geographic mobility (Graham and Pinto, 2021; O'Brien et al., 2017), eviction rates (Bradford and Bradford, 2020), exposure to international trade (Pierce and Schott, 2020), and overall economic disadvantage (Monnat, 2019), all of which have all been found to correlate with increases in one or more despair-related causes of death.

4.4 Heterogeneous effects of employment on deaths of despair

Across much of the literature examining trends and determinants of midlife mortality, educational attainment remains one of the most well-documented determinants of increased risk of death due to DSA, especially among non-Hispanic Whites (Case and Deaton, 2017; National Academies of Sciences, Engineering, and Medicine, 2021). Education is considered a fundamental cause of health (Link and Phelan, 1995) not only because of its implications for labor market opportunities but also as a result of the many ways in which individuals leverage education to improve health through their behavior, relative position in society, social networks, and ability to translate health-related information and resources to enhance health (Cutler and Lleras-Muney, 2006; Grossman, 1972; Leive and Ruhm, 2021). In their most recent working paper, Case and Deaton remind readers that the "deaths of despair" narrative is one "of two Americas, with and without a college degree"—and that analyses examining determinants of the increase in DSA mortality over the past several decades are largely incomplete without attention to this key divide (Case and Deaton, 2021).

In the original paper in their "deaths of despair" series, Case and Deaton note that increased mortality due to DSA, while seen among all non-Hispanic White Americans between 1999 and 2013, was most pronounced among those with a high school degree or less (Case and Deaton, 2015). Specifically, the authors documented that the drug poisoning mortality rate among 45–54-year-old non-Hispanic Whites increased by 44 per 100,000 between 1999

and 2013 relative to a 3.6 per 100,000 increase among those with at least a bachelor's degree. Disparities in the rates of all-cause and cause-specific mortality have continued to widen between those with and without a college degree in more recent years, and these educational gradients have been documented among Black non-Hispanic and Hispanic populations as well (Case and Deaton, 2020a, 2021; Leive and Ruhm, 2021). Between 2003 and 2019, drug mortality doubled among non-Hispanic Black and Hispanic adults with less than a BA, and suicide and alcohol-related causes of death increased by approximately one-third and one-quarter for both groups, respectively (Case and Deaton, 2021).

However, many scholars have extended this cause-specific analysis to highlight important differences in the education-mortality gradients within each demographic group. For example, Geronimus and colleagues (2019) find that while DSA mortality contributed to between one-half and 80 percent of the increase in the all-cause mortality differential between higher- and lower-educated non-Hispanic Whites between 1990 and 2015, there was no detectable widening of the educational gradient in all-cause mortality among Black adults due to these causes of death. This points to the importance of considering both education and race and ethnicity in future analyses of these trends.

Despite being initially framed as an issue affecting declining life expectancy among non-Hispanic White adults (Case and Deaton, 2015), recent research has continued to probe the extent to which premature mortality due to DSA also burdens communities of color. For example, recent findings by Tilstra and colleagues (2021) indicate that there are few differences in the overall trends in opioid-related mortality between Black and White working-age adults over the past several decades, yet the "deaths of despair" narrative continues to center on deteriorating economic and social prospects of middle-aged White Americans. In fact, increases in opioid-related mortality over the past several years has been disproportionately concentrated among Black relative to White populations (Furr-Holden et al., 2021; Lippold et al., 2019; Tilstra et al., 2021), and lower-educated Black populations have been especially hard hit as illicit opioids have proliferated in recent years (Leive and Ruhm, 2021).

Meanwhile, alcohol-related mortality rates have increased for both White and Black Americans, and it is noteworthy that this represents a *reversal* in trend relative to decades of decreasing alcohol-related mortality among Black men and women (National Academies of Sciences, Engineering, and Medicine, 2021; Tilstra et al., 2021). Furthermore, while increases in suicide over the past several decades appear to be a trend primarily experienced by non-Hispanic White adults, there is some evidence that suicide rates are increasing more dramatically among non-White populations in the wake of the COVID-19 pandemic (Bray et al., 2021; Curtin and Hedegaard, 2021).

These trends build on an accumulating body of evidence suggesting that race is a key moderating factor in the relationship between economic exposures and adverse health outcomes, and that this effect may vary across health outcomes. For example, Hollingsworth and colleagues (2017) find that county-level unemployment was linked with significant increases in fatal drug overdoses among non-Hispanic White adults and significant decreases among non-Hispanic Black adults. Despite experiencing substantially higher infection and mortality rates from COVID-19 (likely due to preexisting disparities in healthcare, overrepresentation

in frontline or "essential" work, and other structural causes), survey evidence from the early months of the pandemic indicated that Black respondents were substantially more likely than White respondents to report feeling optimistic and hopeful for the future, and they reported higher levels of mental well-being (Graham et al., 2020)). This latter finding is consistent with previous studies demonstrating that White individuals appear to fare worse in terms of mental health in response to stressful life events relative to racial and ethnic minorities (Assari and Lankarani, 2016).

In contrast, some individual-level studies examining alcohol use in the wake of the Great Recession found that African Americans experienced stronger negative effects of job loss relative to Whites (Jones-Webb et al., 2016; Zemore et al., 2013). Currie and co-authors (2015) find evidence that depression among White women was less negatively impacted by increases in the state-level unemployment rate relative to minority women, yet they had larger relative increases in binge drinking. Relatedly, a series of large studies conducted by Assari and colleagues points to the "diminished return" in health status experienced by Black Americans relative to Whites in response to gains in economic (as well as social and psychological) resources (Assari, 2018). This mixed literature points to the need to further examine the potentially differential effects of positive economic shocks (both aggregate and subgroup-specific) among different demographic subgroups.

4.5 Concluding remarks

Beginning in the early months of the COVID-19 crisis, researchers and policymakers began to speculate that the widespread impacts of the pandemic on all aspects of life—including the extensive economic consequences, the toll of social isolation, grief and trauma following the loss of friends and family, disruptions to healthcare access, and more—would exacerbate the existing mortality crisis due to deaths of despair (Gunnell et al., 2020; Petterson et al., 2020; Reger et al., 2020). In December 2020, the CDC issued an Emergency Preparedness Alert to warn the public about a "concerning acceleration of the increase in drug overdose deaths, with the largest increase recorded from March 2020 to May 2020, coinciding with the implementation of widespread mitigation measures for the COVID-19 pandemic" (Centers for Disease Control and Prevention, 2020). One report published early in the pandemic predicted that the United States could see an additional 27,600 to 150,00 deaths of despair depending on the duration of the economic recovery (Petterson et al., 2020). Some policymakers drew on these predictions to advocate for the reopening of the US economy (Azar, 2020) despite little evidence at the time indicating any substantive change in rates of DSA (Faust et al., 2021; Ranney and Gold, 2020). In their view, Case and Deaton emphasized that the unusual nature of the COVID-19 recession and long-term nature of "despair" as a determinant of DSA mortality suggested that the country was unlikely to see a dramatic increase in deaths of despair, at least in the short term (Case and Deaton, 2020b).

Now nearly two years into the pandemic, there is a rapidly accumulating body of literature examining different dimensions of the COVID-19 pandemic and pandemic response

(e.g., unprecedented levels of job loss, school closures, social isolation, disruptions in healthcare access) on despair-related mortality and morbidity. In general, most descriptive studies converge in documenting increases in opioid overdose and other adverse opioid-related outcomes in 2020 and early 2021 relative to previous years, although the extent to which these are continuations of existing trends or accelerations due to the pandemic is still unknown (Friedman and Akre, 2021; Holland et al., 2021). This includes provisional data from the CDC indicating that the number of drug overdoses surpassed 100,000 between March 2020 and April 2021, making the first year of the pandemic the deadliest one-year period for drug-related mortality on record (Centers for Disease Control and Prevention, 2021a).

There are also a growing number of studies showing that rates of depression, anxiety, and suicidal ideation have increased following the spread of COVID-19 (Czeisler et al., 2020; Farooq et al., 2021; Holland et al., 2021; Panchal et al., 2021). In one of the few recent studies directly linking changes in employment due to COVID-19 with mental health outcomes, Matthews et al. (2021) document increased psychological stress among individuals who reported experiencing permanent job loss, temporary employment, or a pay cut as a result of the pandemic. Despite this well-documented increase in emotional distress in the first years of the pandemic, overall rates of suicide in 2020 decreased by 3% relative to 2019 (Curtin and Hedegaard, 2021), and one recent working paper reports that suicide contributed to 9% fewer excess deaths in the first year of the pandemic (Ruhm, 2021b).

Early in the pandemic, researchers speculated that alcohol consumption could decrease in response to financial constraints and restricted access resulting from the closure of bars and restaurants (Rehm et al., 2020), yet increases in overall consumption have been widely reported, including the prevalence of heavy and problematic drinking (Barbosa et al., 2021; Hauck, 2021; Pollard et al., 2020). One cross-sectional study drawing on data from April through September 2020 reported that harmful alcohol use and alcohol dependence were strongly associated with job loss and found to be significantly higher among those experiencing longer periods of lockdown or "stay-at-home" orders (Killgore et al., 2021). However, that these trends have not directly translated to noticeable increases in alcohol-related mortality is unsurprising given the time lag linking increases in heavy drinking to chronic outcomes such as cirrhosis and alcoholic liver disease.

Taken at face value, the results from Chapter 2 in this dissertation would predict an increase in suicide rates accompanied by a decrease in rates of drug-related causes of death in response to the impacts of the pandemic on the labor market. The early evidence in the 18 months since the pandemic began suggests that just the opposite has occurred: rates of drug-related mortality in the US are at record highs, while the rate of suicide has decreased. However, that the early effects of the COVID-19 crisis on deaths of despair are inconsistent with the main conclusions from the analyses presented here is not entirely surprising given the unique nature of the current recession and the compounding effects of public health mitigation strategies that may have unintended consequences on mental and behavioral health.

Moreover, the analyses above point to a unique set of economic determinants for different deaths of despair and between demographic groups. The overall trends in cause-specific

mortality described above likely mask important heterogeneities across segments of the population who may be more vulnerable or suffer disproportionately in response to the economic impact of the virus and pandemic response. Developing a more nuanced understanding of these disparities—particularly along key dimensions such as race/ethnicity and socioeconomic status—will be critically important in designing equitable policies to help the country recover from the pandemic and reverse the disconcerting trends of increasing midlife mortality in the years to come.

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Appendix A

Chapter 2 – Supplementary Tables and Figures

Table A.1: Relationship between predicted employment, baseline employment shares, and changes in baseline characteristics

		NAICS Two-digit sector code				
	Predict. EPOP	**5**	**7**	**15**	**17**	**17**
Female	6.391***	0.185	-0.385***	-0.146*	0.086***	0.099
	(0.861)	(0.243)	(0.092)	(0.070)	(0.022)	(0.052)
25-34	3.060***	-0.693***	-0.165**	-0.016	0.093***	0.032
	(0.826)	(0.136)	(0.053)	(0.041)	(0.012)	(0.026)
35-44	2.635***	-0.229*	-0.196***	0.007	0.047***	0.051*
	(0.690)	(0.111)	(0.043)	(0.033)	(0.010)	(0.022)
45-54	0.745	0.582***	-0.088	-0.094*	0.057***	0.041
	(0.769)	(0.151)	(0.059)	(0.045)	(0.014)	(0.030)
55-64	3.191***	0.484**	-0.167*	-0.245***	-0.051**	0.037
	(0.865)	(0.170)	(0.066)	(0.051)	(0.016)	(0.034)
Hispanic	1.701***	-0.598***	-0.106***	-0.064***	0.029***	0.003
	(0.415)	(0.052)	(0.020)	(0.015)	(0.005)	(0.010)
NH Black	3.569***	-0.437***	0.276***	0.138***	0.032***	0.025
	(0.532)	(0.066)	(0.026)	(0.020)	(0.006)	(0.013)
NH Other	4.383***	-0.455***	-0.393***	0.187***	0.039***	0.094***
	(0.934)	(0.106)	(0.042)	(0.032)	(0.010)	(0.020)
Some coll.	0.044***	0.005***	-0.001**	0.000	-0.000***	0.001***
	(0.006)	(0.001)	(0.000)	(0.000)	(0.000)	(0.000)
Poverty	0.008***	-0.002***	-0.000**	0.000***	0.000***	0.000***
	(0.002)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
HH inc.	6.391***	0.185	-0.385***	-0.146*	0.086***	0.099
	(0.861)	(0.243)	(0.092)	(0.070)	(0.022)	(0.052)
Observations	3128	2750	2997	2896	2860	1685
R^2	0.073	0.199	0.129	0.095	0.204	0.119

Notes: Table displays estimated coefficients from regressions of the predicted employment rate on 2003–2017 changes in county characteristics (Col. 1) and from regressions of the largest 5 industry shares (based on total employment in 2002) on 2003–2017 changes in county characteristics (Col. 2-6). Regressions are weighted by the total working-age population in each county in 2002. Standard errors are clustered at the county level and presented in parentheses. NAICS codes refer to the following sectors: 5—Manufacturing; 7—Healthcare and social assistance; 15—Retail; 16—Accommodation and food services; 17—Education. Significance: * p<0.05, ** p<0.01, *** p<0.001.

Table A.2: Relationship between 1999–2002 difference in mortality and value of the shift-share instrument in 2003

	Log(Pred. EPOP)	*Log*(Pred. EPOP)
Alcohol	0.458	0.596
	(0.268)	(0.315)
Drug non-suicide	-1.051***	-0.158
	(0.255)	(0.287)
Non-drug suicide	-0.462	-0.304
	(0.329)	(0.387)
Drug suicide	0.033	0.168
	(0.108)	(0.128)
County dem. controls	N	Y
Observations	3089	3089

Notes: Each cell presents the estimated coefficient from the regression of the 1999–2002 three-year difference in cause-specific mortality rate on the value of the shift-share instrument (log predicted EPOP) in 2003. The rightmost column controls for county-level baseline characteristics listed in the footnote to Table 2. Standard errors are clustered at the county level and presented in parentheses. All regressions are weighted by the county-level working-age population in 2003. Significance: * p<0.05, ** p<0.01, *** p<0.001.

Table A.3: First stage regression of actual county-level employment rates on predicted county-level employment rates, 2003–2017

	Model 1	Model 2	Model 3
Log shift-share instrument	41.96***	19.83***	18.87***
	(1.367)	(1.532)	(1.449)
Share female	-161.1***	-84.69**	-47.97*
	(10.98)	(26.22)	(22.02)
Share ages 25–34	30.87	7.715	-23.39
	(36.19)	(12.39)	(12.00)
Share ages 35–44	41.71**	-6.866	-22.84
	(13.27)	(10.83)	(13.71)
Share ages 45–54	-68.62***	22.05	11.60
	(16.09)	(12.51)	(12.79)
Share ages 55–64	-55.62***	6.547	-26.90
	(10.82)	(23.43)	(22.31)
Share Hispanic	-14.34***	-30.95*	-28.55*
	(3.085)	(14.96)	(14.10)
Black non-Hispanic	-5.769	-40.08***	-23.41*
	(5.685)	(11.17)	(9.802)
Other non-White non-Hispanic	-9.860*	24.78	12.22
	(4.200)	(17.98)	(19.16)
Share with some college	-0.522*	0.0902	0.0888
	(0.251)	(0.0803)	(0.0700)
County and year fixed effects	N	Y	Y
State-by-year fixed effects	N	N	Y
First stage F-statistic	991.7	167.4	169.7
Observations	46596	46595	46595

Notes: Table displays all regression coefficients (net the fixed effects) from the first stage regressions of the observed county-level predicted employment-population ratio (EPOP) on the log of the predicted EPOP, controlling for county-level demographic characteristics (all models), county and year fixed effects (Models 2 and 3) and state-by-year fixed effects (Model 3). Regressions 1-3 correspond to the first stages of the two-stage least squares regressions presented in Table 2 (columns 1b, 2b, and 3b). The unit of analysis is the county-year. Standard errors are clustered at the county level and presented in parentheses. Significance: * p<0.05, ** p<0.01, *** p<0.001.

Table A.4: Reduced form regression of aggregate mortality on predicted county-level employment, 2003–2017

		Age 19–44		Age 45–64	
	Overall	**Male**	**Female**	**Male**	**Female**
Alcohol	-0.427	0.708	1.348	-2.331	-1.173
	(0.965)	(1.034)	(0.833)	(3.053)	(2.166)
Drug non-suicide	5.281**	5.686	5.545**	8.359*	0.767
	(1.908)	(3.371)	(1.963)	(3.263)	(2.247)
Non-drug suicide	-3.044**	-3.747	-0.998	-6.466*	-0.0885
	(1.037)	(2.295)	(1.162)	(2.785)	(1.135)
Drug suicide	-0.312	0.00198	-0.137	-1.651*	0.421
	(0.340)	(0.555)	(0.633)	(0.691)	(0.795)
Observations	46595	46773	46773	46773	46773

Notes: Table presents estimates from separate reduced form regressions of the cause-specific mortality rate (per 100,000) on the log of the shift-share instrument. The unit of analysis is the county-year cell for the first column and the county-year-demographic cell column for remaining columns. All regressions include county, year, and state-by-year fixed effects and the county-level controls listed in the footnote to Table 2. Each regression is weighted by total working-age population in each cell. Standard errors are clustered at the county level and presented in parentheses. Significance: * p<0.05, ** p<0.01, *** p<0.001.

Table A.5: Effect of one-year lagged employment rates on mortality, aggregate sample (2003–2017)

	(1a) **OLS**	(1b) **IV**	(2a) **OLS**	(2b) **IV**	(3a) **OLS**	(3b) **IV**
Alcohol	-0.0110	-0.0128	-0.0112	-0.0450	-0.00320	0.00101
(mean = 12.25)	(0.00879)	(0.00986)	(0.0132)	(0.0577)	(0.0117)	(0.0551)
Drug non-suicide	-0.0146	0.0341	-0.0511	0.437***	-0.0225	0.270**
(mean = 18.55)	(0.0264)	(0.0202)	(0.0354)	(0.123)	(0.0350)	(0.103)
Non-drug suicide	-0.0303***	-0.0433***	0.0135	-0.225***	0.0131	-0.179**
(mean = 14.00)	(0.00861)	(0.0102)	(0.0118)	(0.0550)	(0.0107)	(0.0562)
Drug suicide	0.00585***	0.00562***	0.00249	-0.0150	-0.00103	-0.0266
(mean = 2.28)	(0.00125)	(0.00135)	(0.00419)	(0.0188)	(0.00432)	(0.0191)
County + year FE	N	N	Y	Y	Y	Y
State-by-year FE	N	N	N	N	Y	Y
First stage F-stat.		1028.0		152.2		161.8
Observations	43549	43499	43549	43499	43549	43499

Notes: Each cell presents an estimate from a separate regression of the cause-specific mortality rate (per 100,000) on the one-year lag of the employment-population ratio (EPOP). The unit of analysis is the county-year. The sample mean of EPOP is 59.56. In the IV models, the one-year lag of EPOP is instrumented using the one-year lag of the log of the shift-share instrument. All models control for the county, year, and state-by-year fixed effects and the county-level controls listed in the footnote to Table 2. Each regression is weighted by total working age population (ages 19–64) at the county level. Standard errors are clustered at the county level and presented in parentheses. Significance: * p<0.05, ** p<0.01, *** p<0.001.

Table A.6: Estimates from fixed effects (FE) and stacked long difference IV models (2004 start year)

		Stacked long difference models				
	Year FE Model	**1-year**	**2-year**	**3-year**	**4-year**	**5-year**
Alcohol	-0.0336	-0.179	-0.0570	-0.195	-0.0917	-0.0278
	(0.0541)	(0.151)	(0.116)	(0.109)	(0.112)	(0.125)
Drug non-suicide	0.286**	0.106	0.0653	0.143	0.188	-0.244
	(0.108)	(0.169)	(0.146)	(0.137)	(0.171)	(0.153)
Non-drug suicide	-0.166**	-0.161	-0.111	-0.180	-0.0426	-0.234
	(0.0603)	(0.147)	(0.135)	(0.125)	(0.137)	(0.127)
Drug suicide	-0.01000	-0.0229	-0.0745	-0.0391	-0.0866*	-0.0231
	(0.0191)	(0.0475)	(0.0387)	(0.0435)	(0.0414)	(0.0472)
First stage F stat.	163.6	90.26	119.0	117.2	77.87	141.6
Observations	43508	40536	18714	12478	9350	6219

Notes: Each cell presents an estimate from a separate IV regression of the period difference in cause-specific mortality rates (per 100,000) on the period difference in the employment-population ratio (EPOP). The yearly fixed effect (FE) model for the 2004–2017 period is included for comparison. The year FE model includes county, year, and state-by-year fixed effects and county-level controls listed in the footnote to Table 2. The long difference IV models instrument for the difference in EPOP using the difference in the log of the predicted EPOP and control for period fixed effects and start-of-period county-level controls. Each regression is weighted by total working-age population (ages 19–64) at the county level. Standard errors are clustered at the county level and presented in parentheses. Significance: * $p<0.05$, ** $p<0.01$, *** $p<0.001$.

Table A.7: Estimates from instrumented distributed lag models of aggregate alcohol mortality rates on county-level employment

Lag term	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
r = -1	0.0538	0.104	0.118	0.168	0.164	0.149
	(0.0564)	(0.103)	(0.113)	(0.125)	(0.131)	(0.146)
r = 0		-0.0658	-0.164	-0.238	-0.264	-0.279
		(0.0959)	(0.149)	(0.161)	(0.173)	(0.195)
r = 1			0.109	0.0783	0.0708	0.0418
			(0.106)	(0.163)	(0.175)	(0.188)
r = 2				0.0474	0.0428	0.0381
				(0.109)	(0.154)	(0.162)
r = 3					0.0701	0.109
					(0.111)	(0.184)
r = 4						-0.0339
						(0.136)
First stage F	158.2	53.39	22.56	14.20	12.45	7.370
Observations	43506	43466	40345	37226	34109	30995

Notes: Table presents IV estimates from the distributed lag regression of the aggregate alcohol mortality rate (per 100,000) on the employment-population ratio. The leftmost column indicates the number of lag terms included in the model ($r = -1$ denotes a one-year lead and $r = 0$ denotes the contemporaneous term). All regressions include county, year, and state-by-year fixed effects and control for the county-level covariates listed in the footnote to Table 2. Each regression is weighted by total working-age population (ages 19–64) at the county level. Standard errors are clustered at the county level and presented in parentheses. Significance: * p<0.05, ** p<0.01, *** p<0.001.

Table A.8: Estimates from instrumented distributed lag models of aggregate drug non-suicide mortality rates on county-level employment

Lag term	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
r = -1	0.138	0.00231	0.0381	0.130	0.156	0.308
	(0.102)	(0.139)	(0.151)	(0.165)	(0.178)	(0.198)
r = 0		0.170	0.0312	-0.00476	0.0753	0.0423
		(0.124)	(0.181)	(0.193)	(0.207)	(0.229)
r = 1			0.154	-0.174	-0.185	-0.144
			(0.131)	(0.185)	(0.191)	(0.204)
r = 2				0.435**	0.195	0.210
				(0.137)	(0.171)	(0.178)
r = 3					0.384**	0.276
					(0.141)	(0.175)
r = 4						0.218
						(0.132)
First stage F	158.2	53.39	22.56	14.20	12.45	7.370
Observations	43506	43466	40345	37226	34109	30995

Notes: Table presents IV estimates from the distributed lag regression of the aggregate drug non-suicide mortality rate (per 100,000) on the employment-population ratio. The leftmost column indicates the number of lag terms included in the model ($r = -1$ denotes a one-year lead and $r = 0$ denotes the contemporaneous term). All regressions include county, year, and state-by-year fixed effects and control for the county-level covariates listed in the footnote to Table 2. Each regression is weighted by total working-age population (ages 19–64) at the county level. Standard errors are clustered at the county level and presented in parentheses. Significance: * p<0.05, ** p<0.01, *** p<0.001.

Table A.9: Estimates from instrumented distributed lag models of aggregate non-drug suicide mortality rates on county-level employment

Lag term	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
r = -1	-0.0740	0.0911	0.0773	0.0563	-0.0350	-0.0719
	(0.0597)	(0.101)	(0.109)	(0.121)	(0.126)	(0.132)
r = 0		-0.210*	-0.0893	-0.128	-0.0561	0.0250
		(0.0967)	(0.146)	(0.155)	(0.160)	(0.175)
r = 1			-0.150	-0.145	-0.144	-0.252
			(0.103)	(0.172)	(0.179)	(0.192)
r = 2				-0.0123	-0.0443	-0.0604
				(0.128)	(0.182)	(0.195)
r = 3					0.0350	0.00795
					(0.114)	(0.173)
r = 4						0.0538
						(0.130)
First stage F	158.2	53.39	22.56	14.20	12.45	7.370
Observations	43506	43466	40345	37226	34109	30995

Notes: Table presents IV estimates from the distributed lag regression of the aggregate non-drug suicide mortality rate (per 100,000) on the employment-population ratio. The leftmost column indicates the number of lag terms included in the model ($r = -1$ denotes a one-year lead and $r = 0$ denotes the contemporaneous term). All regressions include county, year, and state-by-year fixed effects and control for the county-level covariates listed in the footnote to Table 2. Each regression is weighted by total working-age population (ages 19–64) at the county level. Standard errors are clustered at the county level and presented in parentheses. Significance: * p<0.05, ** p<0.01, *** p<0.001.

Table A.10: Estimates from instrumented distributed lag models of aggregate drug suicide mortality rates on county-level employment

Lag term	**(1)**	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
r = -1	0.00301	0.0343	0.0338	0.0294	0.0209	0.0151
	(0.0194)	(0.0341)	(0.0373)	(0.0405)	(0.0430)	(0.0477)
r = 0		-0.0394	-0.00600	-3.23E-6	-0.0125	-0.0261
		(0.0322)	(0.0473)	(0.0496)	(0.0529)	(0.0573)
r = 1			-0.0338	-0.0947	-0.0759	-0.0559
			(0.0334)	(0.0547)	(0.0580)	(0.0599)
r = 2				0.0777	0.0734	0.0581
				(0.0417)	(0.0639)	(0.0656)
r = 3					-0.00543	-0.0180
					(0.0452)	(0.0670)
r = 4						0.0255
						(0.0435)
First stage F	158.2	53.39	22.56	14.20	12.45	7.370
Observations	43506	43466	40345	37226	34109	30995

Notes: Table presents IV estimates from the distributed lag regression of the aggregate drug suicide mortality rate (per 100,000) on the employment-population ratio. The leftmost column indicates the number of lag terms included in the model ($r = -1$ denotes a one-year lead and $r = 0$ denotes the contemporaneous term). All regressions include county, year, and state-by-year fixed effects and control for the county-level covariates listed in the footnote to Table 2. Each regression is weighted by total working-age population (ages 19–64) at the county level. Standard errors are clustered at the county level and presented in parentheses. Significance: * p<0.05, ** p<0.01, *** p<0.001.

Table A.11: Estimated contemporaneous coefficients and Westfall-Young adjusted p-values, fully disaggregated model

	Panel A: Males, 19–44				Panel B. Females, 19–44			
	Own	**F1**	**M2**	**F2**	**M1**	**Own**	**M2**	**F2**
Alcohol	0.0498	0.114*	0.0252	0.0368	0.0540	0.0313	-0.0180	0.0478*
Adjusted p-value	*0.999*	*0.699*	*0.999*	*0.999*	*0.987*	*0.999*	*0.999*	*0.864*
Drug non-suicide	0.301	0.403*	0.211*	0.160	0.235*	0.214*	0.0548	0.0230
Adjusted p-value	*0.966*	*0.695*	*0.560*	*0.969*	*0.709*	*0.852*	*0.999*	*0.999*
Non-drug suicide	-0.251*	0.0300	-0.0783	-0.0441	-0.0297	0.0483	-0.0412	0.0152
Adjusted p-value	*0.569*	*0.999*	*0.999*	*0.999*	*0.999*	*0.999*	*0.994*	*0.999*
Drug suicide	0.0418	0.00906	0.00616	0.000332	-0.0137	-0.00332	-0.000302	-0.0306
Adjusted p-value	*0.999*	*0.999*	*0.999*	*0.999*	*0.999*	*0.999*	*0.999*	*0.980*
	Panel C: Males, 45–64				**Panel D: Males, 45–64**			
	M1	**F1**	**Own**	**F2**	**M1**	**F1**	**M2**	**Own**
Alcohol	-0.146	0.00446	-0.0356	-0.172	0.000818	0.107	0.00340	0.0486
Adjusted p-value	*0.999*	*0.999*	*0.999*	*0.987*	*0.999*	*0.999*	*0.999*	*0.999*
Drug non-suicide	0.219	0.501**	-0.00506	0.144	0.0830	0.129	-0.0169	0.0823
Adjusted p-value	*0.999*	*0.090*	*0.999*	*0.966*	*0.999*	*0.999*	*0.999*	*0.996*
Non-drug suicide	-0.374**	-0.198	-0.0810	-0.0376	0.00298	0.0377	-0.0530	0.0393
Adjusted p-value	*0.122*	*0.999*	*0.999*	*0.999*	*0.999*	*0.999*	*0.987*	*0.999*
Drug suicide	-0.0417	-0.0380	-0.0129	-0.00735	0.0221	0.0251	0.0228	0.0435
Adjusted p-value	*0.999*	*0.999*	*0.999*	*0.999*	*0.999*	*0.999*	*0.999*	*0.969*

Notes: Table presents estimated coefficients and Westfall-Young adjusted p-values (in italics) from the IV regressions of demographic group-specific mortality rates on the group-specific employment-population ratios. Adjusted p-values are calculated using the *rwyoung* Stata command (Jones et al. 2019) with 1,000 replications. Asterisks on the point estimates indicate statistical significance based on the naive (unadjusted) p-values (* p<0.05, ** p<0.01, *** p<0.001).

Abbreviations: M1 (Male, 19–44), F1 (Female, 19–44), M2 (Male, 45–64), F2 (Female, 45–64)

Table A.12: Robustness exercises with preferred IV models, 2003–2017

	Alcohol	Drug non-suicide	Non-drug suicide	Drug suicide
A. Model with 1-year lead				
EPOP (lead term)	0.104	0.00231	0.0911	0.0343
	(0.103)	(0.139)	(0.101)	(0.0341)
EPOP	-0.0658	0.170	-0.210*	-0.0394
	(0.0959)	(0.124)	(0.0967)	(0.0322)
First-stage F-statistic	53.39	53.39	53.39	53.39
B. Model with housing price				
EPOP	-0.0312	0.233*	-0.183***	-0.0106
	(0.0538)	(0.106)	(0.0550)	(0.0179)
First-stage F-statistic	129.8	129.8	129.8	129.8
C. Winsorized model				
EPOP	-0.0216	0.268**	-0.154**	-0.0158
	(0.0490)	(0.0976)	(0.0534)	(0.0171)
First-stage F-statistic	204.2	204.2	204.2	204.2
D. Inverse hyperbolic sine model				
EPOP	-0.171	0.0498	-0.254**	-0.0186
	(0.104)	(0.162)	(0.0906)	(0.145)
First-stage F-statistic	531.5	531.5	531.5	531.5
E. Commuting zone-level				
EPOP	-0.202	1.271*	-0.310*	-0.0665
	(0.160)	(0.534)	(0.143)	(0.0509)
First-stage F-statistic	24.37	24.37	24.37	24.37

Notes: Table presents coefficient estimates and (standard errors in parentheses) from five sets of IV regressions of the aggregate cause-specific mortality rate (per 100,000) on the aggregate employment-population ratio (EPOP). The unit of analysis for all regressions in panels A–D is the county-year, and the unit of analysis in panel E is the commuting zone (CZ)-year. Regressions in panels A–D include county, year, and state-by-year fixed effects and control for the county-level covariates listed in the footnote to Table 2. In panel A, I substitute the contemporaneous and instrumented EPOPs with the one-year leads for each variable while simultaneously controlling for the observed EPOP in the current time period (N = 43,506). Panel B presents estimates from the preferred model in Table 2 while also controlling for an index of county-level housing prices (N=43,008). Panel C presents estimates from the preferred model in Table 2 where the contemporaneous EPOP is winsorized at the top and bottom 0.5% of the distribution (N=46,595). Panel D presents estimates from the regression of the inverse hyperbolic sine of the count of deaths in each county-year cell on the log employment-population ratio (N =46,595). Panel E presents estimates from the IV model of mortality on EPOP at the commuting-zone level. Models in Panel E control for CZ-level demographic controls, CZ and year fixed effects, and state-by-year fixed effects. * p<0.05, ** p<0.01, *** p<0.001.

Table A.13: Main OLS and IV models with and without county-level controls, 2003–2017

	(1a) **OLS**	(1b) **IV**	(2a) **OLS**	(2b) **IV**
Alcohol	-0.00408	-0.128*	-0.00429	-0.0226
	(0.0146)	(0.0593)	(0.0114)	(0.0513)
Drug non-suicide	-0.0255	0.196	-0.000454	0.280**
	(0.0468)	(0.120)	(0.0368)	(0.103)
Non-drug suicide	0.0121	-0.259***	0.0113	-0.161**
	(0.0116)	(0.0647)	(0.0102)	(0.0562)
Drug suicide	-0.00358	-0.0329	-0.00319	-0.0166
	(0.00449)	(0.0191)	(0.00423)	(0.0179)
Demographic controls	N	N	Y	Y
County + year FEs	Y	Y	Y	Y
State-by-year FEs	Y	Y	Y	Y
First stage F-statistic		132.0		169.7
Observations	46646	46595	46646	46595

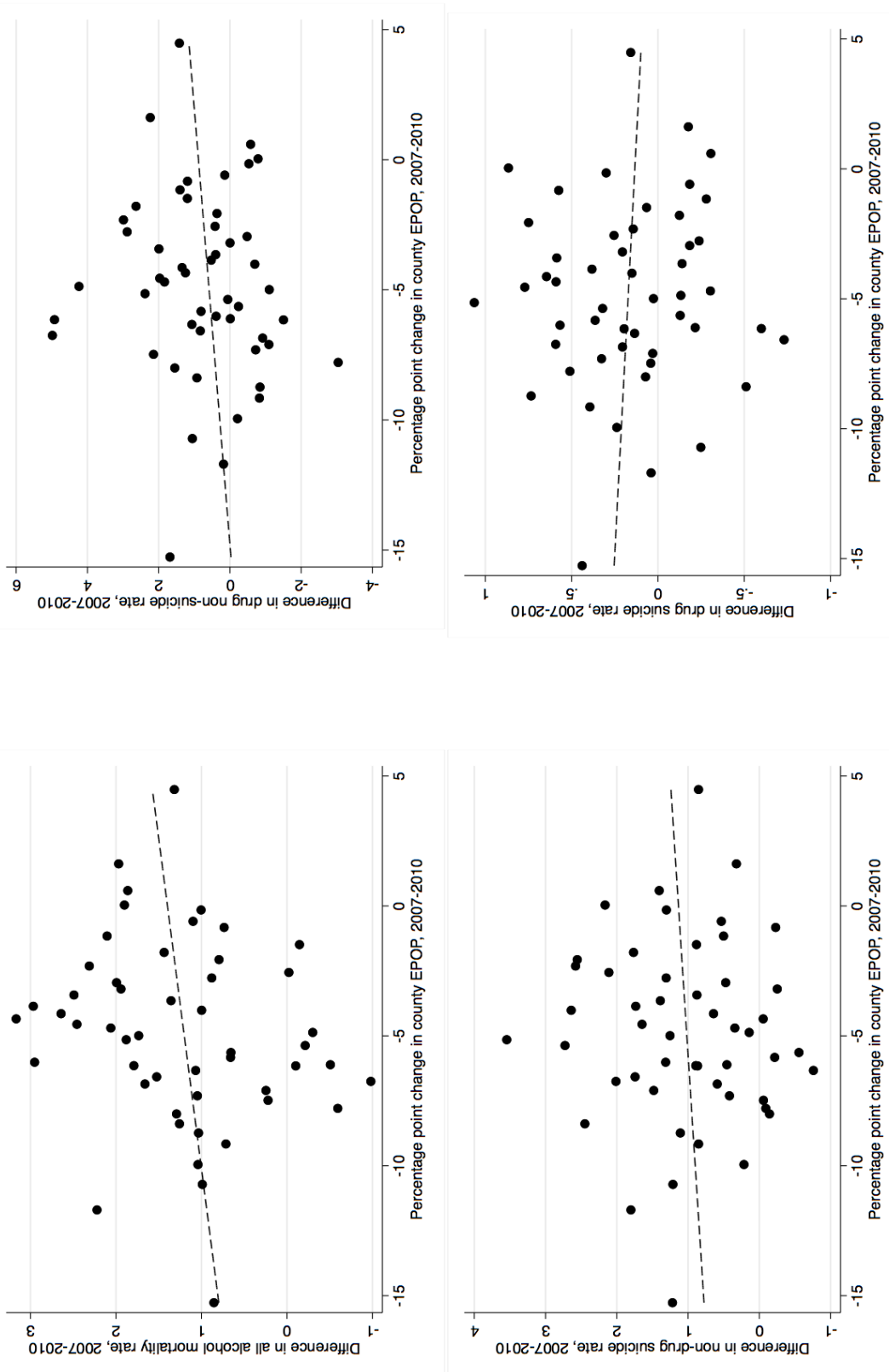
Notes: Each cell presents an estimate from a separate regression of the cause-specific mortality rate (per 100,000) on the employment-population ratio (EPOP). The unit of analysis is the county-year. In the IV models, EPOP is instrumented using the log of the shift-share instrument described in Section 3. County-level characteristics included in columns 2a and 2b include the share of the total working-age population that is female; share ages 25–34, 35–44, 45–54, and 55–65; share Hispanic, non-Hispanic Black, and other non-Hispanic non-White; and share with some college education. Each regression is weighted by total working age population (ages 19-64) at the county level. Robust standard errors are clustered at the county level and presented in parentheses. Significance: * p<0.05, ** p<0.01, *** p<0.001.

Table A.14: Main OLS and IV models with county-level linear trends, 2003–2017

	(1a) **OLS**	(1b) **IV**	(2a) **OLS**	(2b) **IV**	(3a) **OLS**	(3b) **IV**
Alcohol	-0.00429	-0.0226	0.00227	-0.0422	0.00432	-0.0615
	(0.0114)	(0.0513)	(0.0141)	(0.0724)	(0.0144)	(0.0760)
Drug non-suicide	-0.000454	0.280**	0.0741	0.0867	0.0847*	0.0638
	(0.0368)	(0.103)	(0.0470)	(0.114)	(0.0390)	(0.113)
Non-drug suicide	0.0113	-0.161**	-0.00711	-0.0718	0.0104	-0.0893
	(0.0102)	(0.0562)	(0.0148)	(0.0747)	(0.0156)	(0.0797)
Drug suicide	-0.00319	-0.0166	-0.00594	-0.0331	0.00381	-0.0228
	(0.0102)	(0.0562)	(0.00567)	(0.0254)	(0.00586)	(0.0269)
State-by-year FE	Y	Y	N	N	Y	Y
County linear trends	N	N	Y	Y	Y	Y
First stage F-stat.		169.7		132.9		138.1
Observations	46646	46595	46646	46595	46646	46595

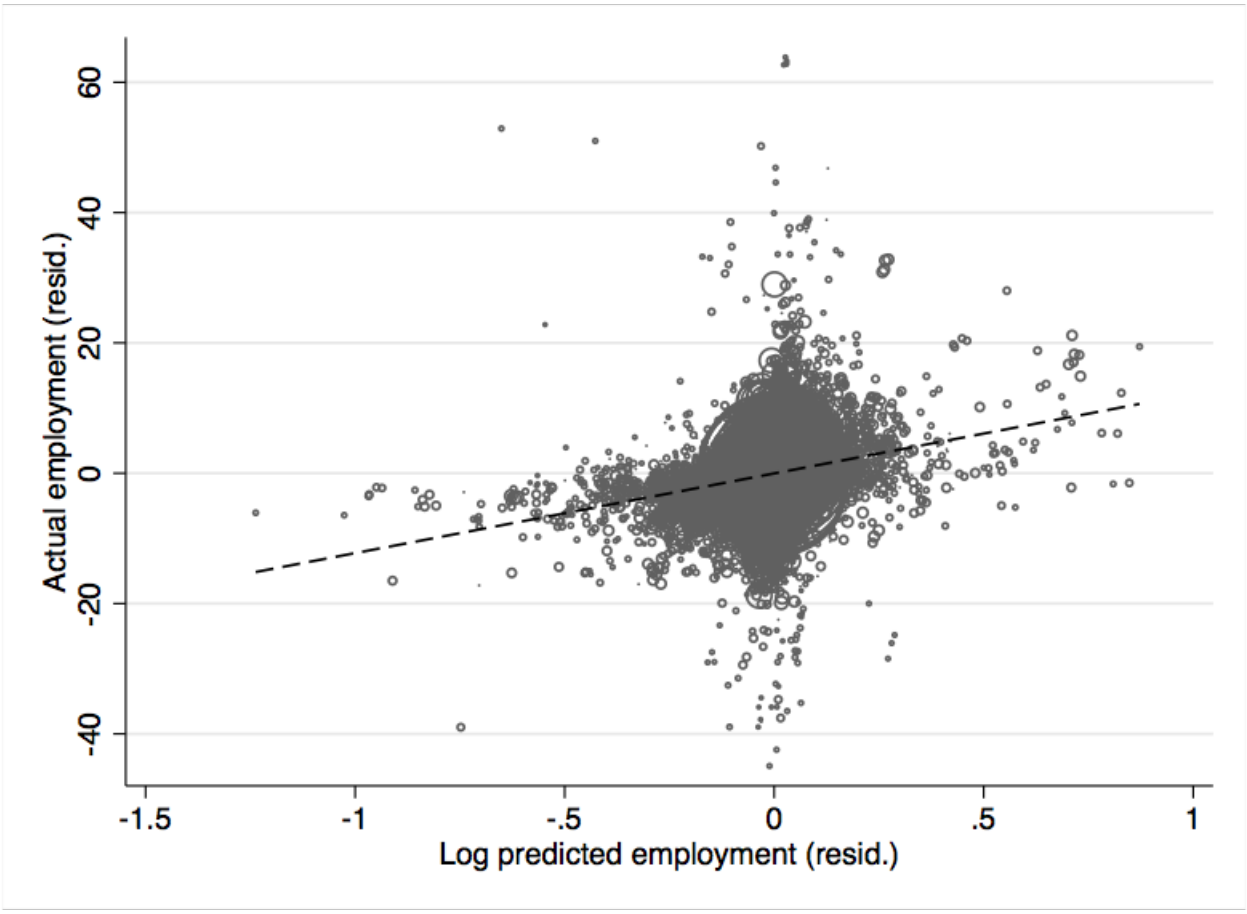
Notes: Each cell presents an estimate from a separate regression of the cause-specific mortality rate (per 100,000) on the employment-population ratio (EPOP). The unit of analysis is the county-year. In the IV models, EPOP is instrumented using the log of the shift-share instrument described in Section 3. County-level characteristics included in columns 2a and 2b include the share of the total working-age population that is female; share ages 25–34, 35–44, 45–54, and 55–65; share Hispanic, non-Hispanic Black, and other non-Hispanic non-White; and share with some college education. Each regression is weighted by total working-age population (ages 19-64) at the county level. Robust standard errors are clustered at the county level and presented in parentheses. Significance: * p<0.05, ** p<0.01, *** p<0.001.

Figure A.1: Relationship between changes in cause-specific mortality and employment-population ratios, 2007–2010



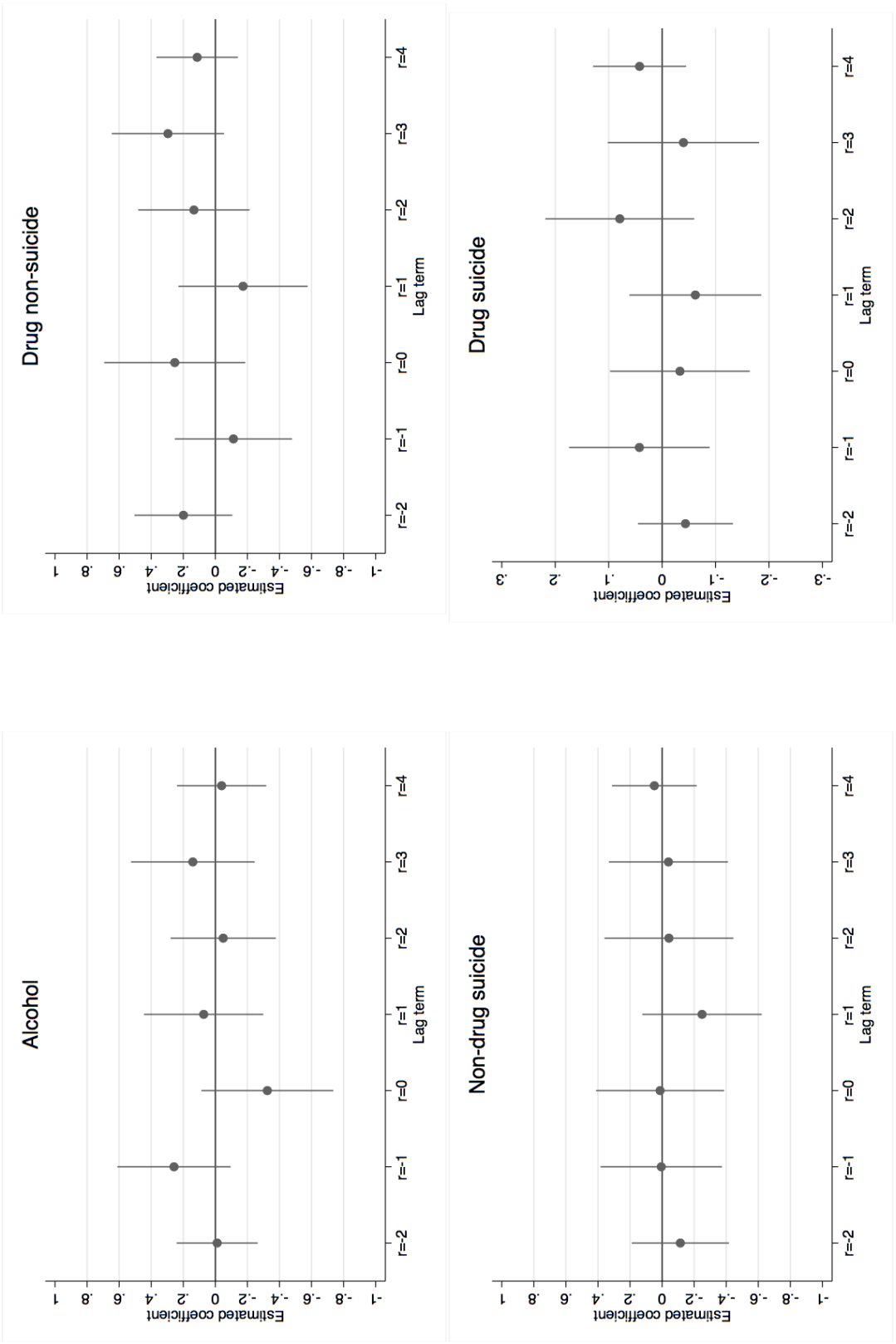
Notes: Figures show the non-parametric relationship between changes in county-level employment-population ratio and cause-specific mortality rates from 2007 to 2010. Points represent mean values of the outcome within 50 evenly sized bins of changes in EPOP. The dashed line depicts a linear fit line weighted by the estimated 2007 population in each county-year observation.

Figure A.2: Relationship between actual and predicted employment rates (log), residualized



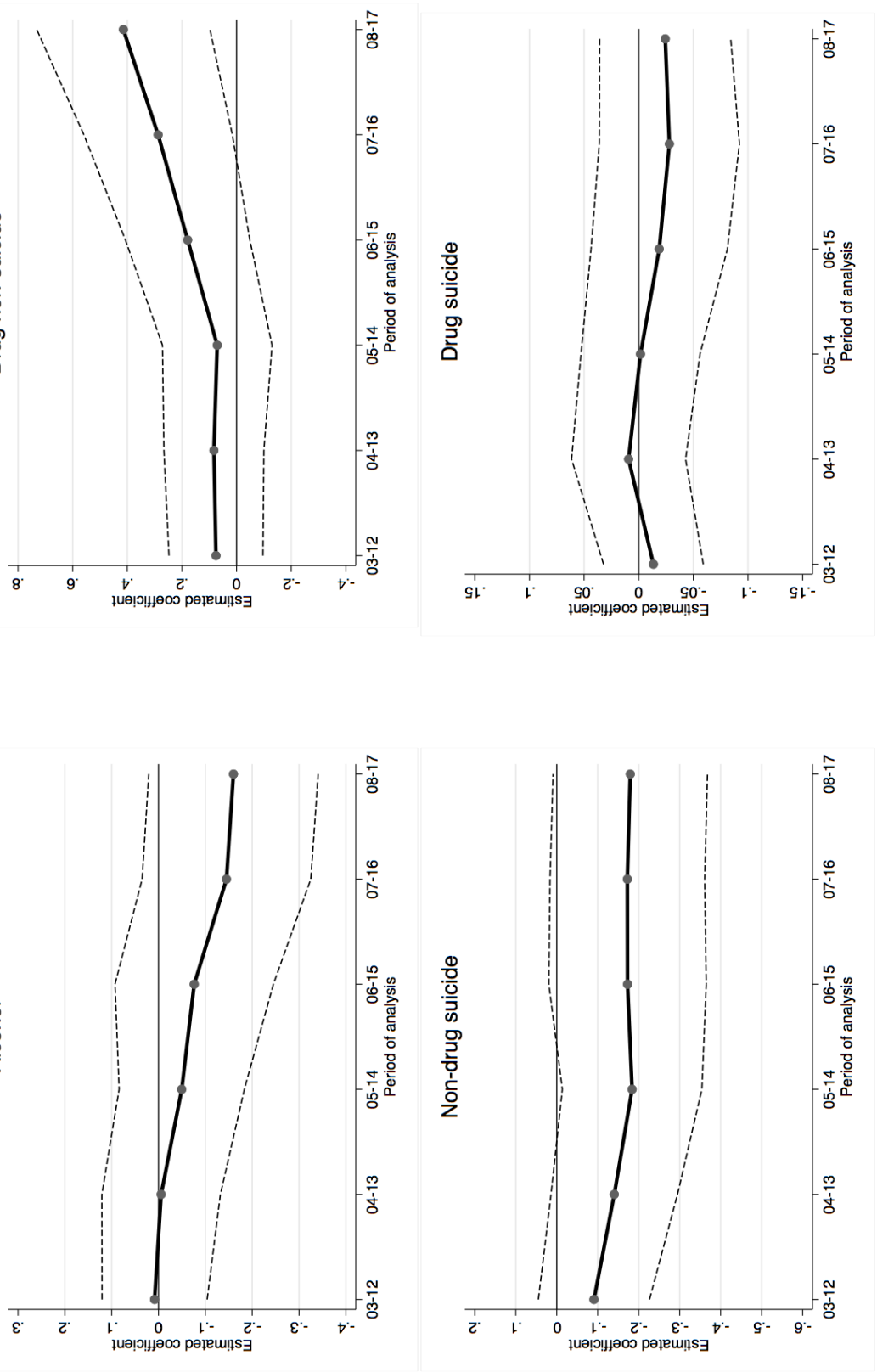
Notes: Figure presents the residuals from the partial first-stage regression of the actual employment rate on the log of the predicted employment rate. Regression controls for county, year, and state-by-year fixed effects.

Figure A.3: Estimated dynamic effect of short-term employment increase on cause-specific mortality



Notes: Figures display the estimated contemporaneous effect and distribution of lag coefficients (with 95% confidence intervals) from the IV model that includes two lead years, a contemporaneous term, and four lag terms of the shift-share instrument. The dependent variable is the cause-specific mortality rate (per 100,000 at the county-year level). Models include county, year, and state-by year fixed effects and the county-year controls listed in Table 2. Data years include 2003–2017.

Figure A.4: Estimated effect of aggregate employment rates on cause-specific mortality across 10-year panels



Notes: Figures present point estimates and 95% confidence interval bands from the IV regression of each cause-specific mortality rate (per 100,000) on the aggregate county-level employment-population ratio. All periods of analysis are 10 years long, as denoted on the horizontal axis. All models control for county, year, and state-by-year fixed effects and the county-year controls listed in Table 2.

Appendix B

Chapter 3 – Case definitions

Additional information on exclusion restrictions and case definitions:

Hospice or palliative care is identified using the following CPT/HCPCS codes: 99377-99378, Q5001-Q5010, G9524, S9126, G947X, or T2042-T2046.

Opioid exposure is determined in the pharmacy claims database by matching on any of 4,824 unique national drug codes (NDCs) for the following products:

- Butorphanol, Codeine, Dihydrocodeine, Levorphanol, Meperidine, Methadone, Opium, Pentazocine, Propoxyphene, and both long- and short-acting versions of the following: Fentanyl, Hydrocodone, Hydromorphone, Morphine, Oxycodone, Oxymorphone, Tapentadol, and Tramadol.

Benzodiazepine exposure is determined in the pharmacy claims database by matching on any of 6,447 unique NDCs for the following drugs:

- Alprazolam, Chlordiazepoxide, Chlordiazepoxide Hydrochloride, Clobazam, Clonazepam, Clorazepate Dipotassium, Diazepam, Estazolam, Flurazepam Hydrochloride, Halazepam, Lorazepam, Oxazepam, Prazepam, Quazepam, Temazepam, and Triazolam.

Evidence of malignant cancer is defined using the following ICD-9 codes: 140-172, 174-194, 195, 200-209, 231; and the following ICD-10 codes: C00-C43, C38.8, C45-C75, C76, C81-C94, C96-C99, D02.

Chemotherapy was identified using the following HCPS codes: C1166, C1167, C1178, C9021, C9025, C9027 C9120, C9127, C9129, C9131, C9205, C9213, C9215, C9218, C9231, C9235, C9240, C9243, C9253, C9260, C9262, C9265, C9273, C9276, C9280, C9284, C9287, C9289, C9292, C9295, C9296, C9414, C9415, C9416, C9418, C9419, C9420, C9421, C9422, C9423, C9424, C9425, C9426, C9427, C9428, C9429, C9431, C9433, C9437, C9440, C9442, C9449, J0594, J0894, J3315, J7309, J8510, J8520, J8521, J8530, J8560, J8562, J8565, J8600, J8700, J8705, J9000, J9001, J9002, J9015, J9017, J9019, J9020, J9025, J9027, J9031, J9032, J9033, J9039, J9042, J9043, J9045, J9047, J9050, J9055, J9060, J9062, J9065, J9070, J9080, J9090, J9091,J9092, J9093, J9094, J9095, J9096, J9097, J9098, J9100, J9110, J9120, J9130, J9140, J9150, J9151, J9155, J9160, J9170, J9171, J9178, J9179, J9180, J9181, J9182, J9185, J9190, J9200, J9201, J9202, J9206, J9207, J9208, J9209, J9211, J9228, J9230, J9245, J9261, J9263, J9264, J9265, J9267, J9271, J9300, J9301, J9302, J9303, J9305, J9306, J9308, J9310, J9315, J9328, J9340, J9350, J9351, J9354, J9355, J9360, J9370, J9371, J9375, J9380, J9390, J9395, J9400, Q2017, Q2025, Q2043, Q2048, Q2049, Q2050, S0088, S0156, S0168, S0172, S0175, S0178, S0182

Radiation therapy is identified using the following procedure, diagnostic, and revenue codes:

- CPT/HCPCS procedure codes: 19296-19298, 20555, 32553, 41019, 49411, 55875, 55876, 55920, 57155, 58346, 61770, 61796-61799, 63620-63621, 76370, 76950, 76965, 77014, 77261-77799, 79000-79999, 0520F, 4165F, 4181F, 4200F-4201F, 0190T, 0197T, C1325, C1348, C1350, C1700-C1712, C1715-C1720, C1728, C1790-C1806, C2616, C2632, C2633, C9714, C9715, G0174, G0178, G0179, G0261, G0256, G0273, G0274, G0338-G0340

- ICD-9 diagnostic codes: 508.0, 508.1, 558.1, 990, V58.0, V66.1, V67.1, E926.5

- ICD-9 procedure codes: 92.2X

- ICD-10 diagnostic codes: J70.0, J70.1, K52.0, T66XXXA, Z51.0, Z51.89, Z08, Z09, W881XXA

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