Demographic Divergence: The Legacy of the Opioid Epidemic

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Abstract

In this paper, we study how the opioid epidemic influenced local population growth in the United States. Exploiting variation in exposure to the epidemic—driven by Purdue Pharma's targeted marketing of OxyContin and proxied by 1996 cancer mortality rates—we find that commuting zones with greater exposure experienced significant population declines. By 2020, areas more exposed to the epidemic experienced 2.5 percentage points lower population growth per one–standard-deviation increase in exposure, and this reduced growth was concentrated among individuals aged 18 to 44. Direct mortality from drug-induced deaths made only a limited contribution to these changes, particularly during the first decade of the epidemic. Instead, population losses were primarily driven by shifts in migration patterns: exposure increased out-migration rates, especially among women and college-educated individuals. We also document a rise in fertility that, by 2020, partially offset population losses among individuals under 18. Taken together, our findings show that the epidemic reshaped local demographic composition and contributed to the long-run divergence in population dynamics across U.S. commuting zones.

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I. Introduction

Regionally concentrated economic shocks have been one of the defining social and economic challenges of recent decades (Autor et al., 2025). A large body of research documents how globalization, automation, and the energy transition, among others, have created geographically uneven and long-lasting effects on local labor markets (Autor and Dorn, 2013; Acemoglu and Restrepo, 2020; Hansen et al., 2023). These shocks have not only depressed employment and earnings but have also had far-reaching consequences for health, family structure, and political polarization (Autor et al., 2019*b*; Adda and Fawaz, 2020; Autor et al., 2020; Pierce and Schott, 2020; Rodrik, 2021; Choi et al., 2024). During this period, the pace of income convergence across regions has slowed markedly, and widening economic disparities have been accompanied by demographic shifts (Moretti, 2011; Ganong and Shoag, 2017; Autor et al., 2025). Local population decline has emerged as a growing challenge, with more than half of all U.S. counties losing population between the 2010 and 2020 censuses (Mackun et al., 2021).

In this paper, we examine another regionally concentrated shock—the opioid epidemic—and study its effects on local population growth, mortality, internal migration, and fertility. The opioid epidemic is one of the most devastating and far-reaching public health crises in U.S. history. It has been accompanied by rising mortality, widespread addiction, economic hardship, and shifts in political preferences (Maclean et al., 2020; Arteaga and Barone, 2025). To isolate the effects of the epidemic from broader demographic and socioeconomic trends, our identification strategy draws on unsealed court records from litigation against Purdue Pharma—the manufacturer of OxyContin, the prescription opioid at the center of the crisis. These records reveal that Purdue initially targeted areas with high cancer pain incidence, with plans to expand to the larger non-cancer pain market within those same geographic areas. This strategy meant that non-cancer patients in high-cancer areas were disproportionately exposed to the opioid epidemic and the cascade of adverse outcomes that followed. Following Arteaga and Barone (2025), we use 1996 cancer mortality—measured prior to the onset of the crisis—as a proxy for exposure. This work and subsequent research, show that mid-nineties cancer mortality is a strong indicator of exogenous exposure to the opioid epidemic.[1]

Our results show that the opioid epidemic contributed to growing regional divergence across U.S. communities. Areas more exposed to the epidemic experienced significant *relative* population declines 10 and 20 years after the onset of the crisis. We estimate that by 2010, areas more exposed to the epidemic experienced 1.2 percentage points lower population growth per one–standard-deviation increase in exposure; by 2020, this effect was 2.5 percentage points. To put this number into context, between 2000 and 2020 the average local population change was 8.6%, our estimates suggest that one standard

[1]See, for example, Buckles et al. (2022); Cohle and Ortega (2023); Olvera et al. (2023); Siegal (2023).

deviation of epidemic exposure predicts a population decline of 28% of the observed average change.[2] These effects are stronger among individuals aged 18 to 44 years old and persist over time. By 2020, the young adult population declined by 4.1 percentage points for a one standard deviation of exposure–or 20% of the inter-quartile local population change.

A natural source of population decline in the context of the opioid epidemic is the direct increase in mortality induced by the crisis. Since 1999, more than 700,000 Americans have died from opioid overdoses (CDC, 2023*b*), and Arteaga and Barone (2025) show that high-exposure communities experienced drug-related deaths 46% higher than those in low-exposure areas. To assess the contribution of mortality to overall population loss, we compute counterfactual population counts by adding back accumulated drug-induced deaths and re-estimate our regressions. This counterfactual represents a scenario with *no opioid epidemic*, rather than one in which we remove deaths induced as a result of exposure to the epidemic, measured through mid-nineties cancer mortality. As such, it provides an *upper bound* on the contribution of mortality to the estimated population decline. Among individuals aged 18 to 44, we find that by 2010, population decline would have been only 2% smaller in the absence of drug-related mortality (2.42 percentage points instead of 2.47). By 2020, however, mortality accounts for roughly one-third of the observed decline (2.77 percentage points instead of 4.16), with particularly pronounced role among individuals without any college education.

While mortality directly reduced population growth, changes in migration patterns emerge as the main driver of local demographic adjustment. Consistent with models predicting that migration is a primary channel of adjustment to local economic shocks (Blanchard et al., 1992), we find that exposure to the epidemic induced significant migratory responses. A one–standard-deviation increase in exposure led to a 4% increase in out-migration rates by 2011, which persisted thereafter. These responses are driven by young women, for whom we estimate a 13% increase in out-migration rates over a similar period. The effects are strongest among women with some college education, while we find no statistically significant migration responses for men. Back-of-the-envelope calculations suggest that these migratory responses account for most of the observed population changes between 2000 and 2020.

To further understand the nature of these migratory adjustments, we explore the characteristics of destination communities and whether individuals leaving highly exposed areas moved to less exposed locations. On average, movers relocated to *better* areas—measured by lower exposure to the epidemic. However, these moves did not reflect a shift toward new destinations. Instead, individuals from highly exposed areas moved

[2]For further reference, Arteaga and Barone (2025) estimate that areas with a one–standard-deviation higher cancer mortality rate in 1996 experienced an additional 0.97 opioid doses prescribed per capita by 2012—the year when prescription rates peaked—equivalent to 65% more than the baseline mean. By 2017, this translated into a 46% increase in drug-related mortality relative to its pre-epidemic average.

more frequently to the *same* destinations as before the epidemic. In other words, greater exposure increased out-migration to historical destinations—a *push* factor—rather than attracting new inflows to less exposed areas—a *pull* factor.

Beyond migration, shifts in fertility behavior provide another important demographic response to local hardship. We find that exposure to the opioid epidemic led to higher fertility rates. By 2018, a one–standard deviation increase in exposure raised fertility rates at the commuting zone level by approximately 5.5%. This increase is primarily driven by unmarried women, for whom we detect rising fertility beginning in 2006, with effects that persist and grow over time. Among married women, we estimate fertility increases that are about half as large and statistically significant starting in 2013. The effects are concentrated among women in their late twenties and are higher for those without a college degree. Although these fertility responses have limited explanatory power for the population change up to 2010, they have growing importance over longer-run horizons, and contribute to the widening divergence in family formation patterns by education level (McLanahan, 2004; Lundberg and Pollak, 2014; Kearney, 2022).

Taken together, these patterns point to a pronounced gender dimension in the demographic response to the opioid epidemic. To understand the roots of this heterogeneity, we next examine how exposure differentially affected men and women through economic channels. We find that exposure to the epidemic generated greater economic hardship for women. Specifically, although employment declined across the board, the losses were larger for women than for men, and particularly pronounced among individuals without a college education. When examining the employment-to-population ratio, we observe a sustained decline among women without a college degree, as well as a reduction in average earnings among college-educated women. We complement these results by analyzing participation in the Supplemental Nutrition Assistance Program (SNAP)—the largest federal anti-poverty program in the United States. Consistent with the employment patterns, we find a strong link between opioid exposure and rising SNAP enrollment: exposure to the epidemic predicts increased applications for both men and women, with markedly stronger effects among women. The greater economic hardship experienced by women can explain their higher out-migration rates and increased fertility, as the opportunity cost of childbearing declined.

Our empirical analysis combines data from several sources. We use the Decennial Censuses, the American Community Survey (ACS) and Survey of Epidemiology and End Results (SEER) to construct local population counts and, jointly with IRS Statistics of Income (SOI) Tax Stats data, to measure migration behavior. To capture the economic impact of the epidemic, we rely on data from the Longitudinal Employer–Household Dynamics (LEHD) program, which provides detailed county-level information on employment and earnings. We complement these with data on Supplemental Nutrition Assistance Program (SNAP) receipt rates and applications from the Food and Nutrition

Service of the U.S. Department of Agriculture. Finally, we use data from the National Vital Statistics System (NVSS) to construct fertility outcomes, opioid-related mortality rates, and cancer mortality. Our analysis focuses on 587 commuting zones with populations exceeding 25,000 in 1996, observed annually from 1980 to 2020, resulting in a panel dataset comprising 24,067 CZ–year observations.[3]

We provide several tests to support our empirical design and the robustness of our results. To assess whether there is a systematic relationship between cancer rates and our outcome variables, we perform an out-of-sample exercise using 1976 cancer mortality data and replicate our empirical strategy for the pre-period. We find no evidence of a relationship between lagged cancer mortality and future population growth, out-migration rates, fertility, or drug mortality. We also construct placebo 1996 mortality rates from unrelated causes of death and replicate our main specification using these data. This exercise shows that our results are not driven by other health trends unrelated to the opioid epidemic, nor reflect underlying population health. We further rule out several alternative explanations that could account for our findings. Because cancer mortality is correlated with the share of the population aged 65 and older, one possible alternative hypothesis is that aging in areas with larger elderly populations explains both population decline and lower out-migration rates. This hypothesis is unlikely, as our effects are concentrated among younger populations. Nevertheless, we directly test it by controlling for the share of the population over 65 in 1996, interacted with year dummies to capture mechanical aging processes. Our results remain unchanged. We also account for geographic exposure to major economic shocks that could confound the relationship between opioid exposure and our outcomes. Specifically, we consider shocks such as increased competition from Chinese imports, the implementation of the North American Free Trade Agreement (NAFTA), the 2001 economic recession, and the Great Recession. Finally, we verify that our results are not sensitive to the exclusion of any single state.

This paper contributes to three distinct literatures. First, we contribute to the literature on the effects of local economic shocks and demographic adjustments to these shocks. This literature has documented how trade shocks (Autor and Dorn, 2013; Greenland et al., 2019), robots adoption (Acemoglu and Restrepo, 2020), the fracking boom (Wilson, 2022), and the Great Recession (Yagan, 2019) have shaped economic conditions of places and how workers have (or not) responded to these shocks. We extend this literature by showing that the opioid epidemic affected all major demographic margins of adjustment—mortality, migration, and fertility—in distinct ways across population subgroups, ultimately leading to sizable changes in local population growth and composition. In particular, we show that mortality played a role, especially among individuals without a college education. We also document large selective out-migration patterns that vary systematically by demographic and socioeconomic characteristics, and that ultimately

[3]These commuting zones account for over 97 percent of the total population in 1996.

are the central cause of lower population growth. This stands in contrast to prior work finding limited migration responses to negative economic shocks (Autor and Dorn, 2013; Yagan, 2019; Borusyak et al., 2022). Finally, we uncover meaningful fertility increases that will shape future population growth and composition.

Second, we contribute to the literature on the socioeconomic determinants of family formation and fertility. A large body of work has documented how economic conditions and individual socioeconomic status shape decisions around fertility. Foundational work by Becker (1960, 1973) emphasized the role of income, opportunity costs, and specialization in determining family behaviors. Subsequent research has shown that economic insecurity—particularly limited labor market prospects for less-educated men—increases nonmarital fertility (Wilson, 1987, 1996; Black et al., 2005; Autor et al., 2019*a*). This literature has also examined the family formation effects of positive economic shocks to men such as the fracking, coal and oil booms (Black et al., 2013; Brueckner and Schwandt, 2015; Kearney and Wilson, 2018), finding increases in fertility rates. On the other hand, another strand of the literature highlights that economic downturns can reduce the opportunity cost of time—especially for women with weaker labor market attachment—thereby increasing fertility through substitution effects, even as declining resources exert opposing income effects (Dehejia and Lleras-Muney, 2004; Perry, 2004; Schaller, 2016). Consistent with this view, we find that exposure to the epidemic reduced employment for women more than for men, helping to explain the observed fertility increases and, more broadly, contributing to divergent patterns of childbearing across the United States.

Finally, this paper is the first to estimate the overall population effects of the opioid epidemic. We extend prior research that has documented the epidemic's consequences for mortality, addiction, disability claims, municipal finances, housing prices, children's outcomes, and political behavior (Buckles et al., 2022; Park and Powell, 2021; Cornaggia et al., 2022; Arteaga and Barone, 2025). Our findings reveal that the epidemic triggered profound demographic adjustments, that ultimately reduced population growth in the communities most affected, and that will continue to shape their social and economic trajectories going forward.

II. Background: The Unfolding of the Opioid Epidemic

The United States has experienced an unprecedented crisis related to the misuse of and addiction to opioids. Over the past three decades, the crisis has evolved from the misuse of prescription opioids to the growing prevalence and use of heroin and fentanyl, leaving a profound impact on the families and communities most directly affected. As of 2022, over 700,000 lives had been lost to opioid overdoses (CDC, 2023*b*). A sizeable body of research has studied the origins of the opioid crisis and the factors that shaped its evolution and propagation. This literature has established that the pharmaceutical industry and

healthcare providers played a critical role in the origins of the crisis (Eichmeyer and Zhang, 2020; Miloucheva, 2021; Alpert et al., 2022; Arteaga and Barone, 2025). In particular, the aggressive and deceptive marketing of potent opioids with high potential for addiction directed toward physicians, in a setting with financial incentives for doctors to increase prescriptions and with weak monitoring, created the perfect platform for the crisis to unfold.

The beginning of the opioid epidemic is traced to the introduction of OxyContin to the market in 1996 (Quinones, 2015). OxyContin is a prescription opioid manufactured by Purdue Pharma that changed the standard of practice for the treatment of noncancer and nonterminal pain. Prior to the mid-1990s, pain management had focused on cancer and end-of-life pain treatment because of care providers' fears of the risk of severe addiction (Melzack, 1990). Soon after the introduction of OxyContin, reports of abuse and addiction started to be covered by the media. By 2001, West Virginia, Virginia, Ohio, and Kentucky were pursuing class-action lawsuits against Purdue Pharma and other pharmaceutical companies. During this time, United States Attorneys for Maine and Virginia, physicians, and community leaders raised concerns to the FDA and Congress about the level of OxyContin abuse (Meier, 2018). The prescription of opioids continued to increase during the 2000s with limited restrictions. At their peak in 2012, opioid prescriptions numbered 81.3 prescriptions per 100 persons (CDC, 2020). The rate of substance use disorder grew by a factor of six between 1999 and 2009 (Paulozzi et al., 2011), and prescription opioid mortality grew by a factor of five (Maclean et al., 2020).

In response to the widespread misuse of prescription opioids and in particular OxyContin, in 2010, Purdue Pharma introduced an abuse deterrent formulation of OxyContin. Evans et al. (2019) and Alpert et al. (2018) show that the reformulation unfortunately led many consumers to substitute toward a dangerous and inexpensive alternative: heroin. As a result, deaths, poisonings, emergency room visits, and enrollments in treatment programs for heroin abuse increased. In particular, between 2010 and 2013, heroin death rates increased by a factor of four with no reduction in the combined heroin and opioid death rate (Evans et al., 2019). From 2013 to this day, the epidemic has been characterized by surging deaths related to the use of synthetic opioids, particularly fentanyl. Fentanyl, an extremely potent synthetic opioid, is more profitable to manufacture and distribute than heroin and has a higher risk of overdose. Indeed, fentanyl-related deaths account for almost the entire increase in drug overdose mortality between 2014 and 2021.

Mortality from opioids is only one of the many social costs associated with the opioid epidemic. Some 10.1 million people aged 12 or older are estimated to have misused opioids in the past year in the U.S. (SAMHSA, 2020). These numbers are orders of magnitude larger than the number of deaths and help rationalize why the opioid epidemic has disrupted health and economic opportunities, affecting individuals and their communities. Notably, the epidemic has increased disability rates and Supplemental Nutrition

Assistance Program (SNAP) utilization (Powell et al., 2020; Savych et al., 2019; Arteaga and Barone, 2025) and affected labor force participation and employment rates (Krueger, 2017; Ouimet et al., 2020).

This evidence highlights the profound toll of the opioid epidemic on individuals, families, and communities. While its health, economic and political consequences are well documented, much less is known about how the epidemic has shaped key demographic adjustments. In the rest of the paper, we examine how exposure to the opioid crisis affected population growth through its effect on mortality, internal migration and fertility- critical processes that influence long-run community structure, social mobility, and economic development.

III. Data

We construct a comprehensive dataset at the commuting-zone level spanning three decades. The data combine a variety of administrative and survey sources that together capture the main margins of demographic adjustment—mortality, migration, and fertility—as well as local labor market and safety-net outcomes. This section describes the data sources, the construction of key measures, and the procedures used to harmonize information across time and geography.

Mortality. We use restricted-use data from the Detailed Multiple Cause of Death (DMCD) files from 1989 to 2018 to construct mortality measures. These files record every death in the United States, including the county of residence, the underlying cause of death, and up to 20 additional contributing causes, and thus represent a census of deaths in the U.S. The 1989–1998 data use ICD-9 codes to classify causes of death, while the 1999–2018 data use ICD-10 codes. We compute the 1996 cancer mortality rate to measure exposure to the opioid crisis.[4] Figure 1 shows the distribution of cancer mortality across geographies in 1996 and Table 1 shows summary statistics for cancer-mortality in the pre- and post-periods.

We construct a measure of drug-related overdose deaths to study the mortality effects of the epidemic. We define drug-induced mortality to include deaths from poisoning and medical conditions caused by the use of legal or illegal drugs, including prescription opioids, heroin, and synthetic opioids such as fentanyl.[5] We analyze drug-related deaths separately for women and men, and also leverage educational attainment data included in death certificates to document effects by education level—specifically, for individuals

[4]We define cancer deaths as those caused by malignant neoplasms (codes 140–208 in ICD-9 and C00–C97 in ICD-10) and in situ neoplasms, benign neoplasms, and neoplasms of uncertain or unknown behavior (codes 210–239 in ICD-9 and D00–D48 in ICD-10).

[5]Drug-related overdose deaths can be difficult to categorize; for this reason, we favor a broad measure of mortality. This category includes codes E850–E858, E950.0–E950.5, E962.0, and E980.0–E980.5 in ICD-9; and X40–X44, X60–X64, X85, Y10–Y14, and T36–T39, T40–T50 in ICD-10.

with some college or more, and those without any college education.[6] Panel (b) of Figure 1 presents the geographic distribution of the evolution of drug-induce mortality between 2018 and 1996. Table 1 presents summary statistics for drug-induced mortality overall and by sex. Mortality rates for men are twice as high as those of women before the start of the epidemic. As the epidemic unfolded, mortality rates for both sexes increased at similar rates.

Population counts. We use three data sources for population counts. First, the Survey of Epidemiology and End Results (SEER), which reports population at the county level and by age, race, sex, and Hispanic origin. We use these data to construct population counts for decennial census years and denominators for mortality rates, e.g., drug-induced mortality by sex. SEER does not provide population counts by educational attainment. For education-specific outcomes, we leverage data from the Census and the ACS. These data are at the county level and include the years 1990 and 2000 from Census and 2006 onward from the ACS. When using Census and ACS data to construct denominators, we linearly interpolate population counts.

Migration. We collect data on county-to-county migration flows from the IRS Statistics of Income (SOI) Tax Stats. These data are based on the universe of Forms 1040 filed and processed by the IRS during a calendar year and are available for filing years 1990 through 2021. The dataset includes the number of personal exemptions claimed, which approximates the number of individuals in a given county. Migration flows are identified based on year-to-year address changes reported on individual income tax returns. These data capture migration patterns across all U.S. counties and include both inflows—the number of new residents moving into a county and their origin—and outflows—the number of residents leaving a county and their destination. While these data cover most internal migration, they do not include demographic information. To examine migration flows for specific subpopulations, we complement the IRS data with information from the Census and the ACS. Specifically, we construct the number of moves from each geographic area based on individuals' reported locations in the previous year and rely on person weights to generate representative estimates.[7] Informed by Foschi et al. (2023), we drop the four least populous states of the contiguous United States (South and North Dakota, Vermont, and Wyoming) from our analysis sample due to the volatile state population swings observed in the ACS data.

Fertility. We use the National Center for Health Statistics (NCHS) Natality and Linked

[6]The 2018 data were the first for which educational attainment was consistently reported using the 2003 revision of the death certificate across all deaths (unless missing entirely). In prior years, some deaths still used the 1989 revision's categorization. This change prevents us from constructing more granular education categories. The next sub-section provides more detail on these definitions.

[7]Unfortunately, the 1990 and 2000 Censuses ask about residence five years prior. Therefore, for Census years, we construct the number of moves based on this retrospective question.

Births files to construct fertility rates. These data represent the universe of births in the United States and include geographic identifiers—such as the county of birth—as well as detailed demographic characteristics of the birthing individual, including age, marital status, and educational attainment. From these data, we compute average fertility rates at the commuting-zone level. The overall fertility rate is defined as the number of births to individuals in a given age group divided by the population in that age group.

We further disaggregate fertility rates by marital status and educational attainment.[8] For the analysis by marital status, we define the married (unmarried) fertility rate as the number of births to married (unmarried) individuals in a given age group divided by the population of married (unmarried) women in that age group. The numerator is obtained directly from the NCHS Files; the denominator is constructed using data from the U.S. Census and the American Community Survey (ACS).

To compute fertility rates by educational attainment, we classify mothers into two groups: those with some college or more, and those without any college education. The NCHS data include information on years of schooling, but we reclassify education levels to account for changes in how this information is reported across time and states. We define "some college or more" as having completed more than 13 years of education, any number of years of college without a degree, or any higher degree (e.g., a bachelor's or master's degree). If education is reported but does not meet this definition, we classify the individual as having "no college education." As with the marital status analysis, the denominator is constructed using Census and ACS data.

Economic outcomes. We collect data from the Longitudinal Employer-Household Dynamics (LEHD) program, which provides detailed information on employment and earnings at the county-level and by age, race, sex and education. Specifically, we use the Quarterly Workforce Indicators (QWI) employment counts and average monthly earnings. We construct sex-specific employment counts and employment-to-population ratios relying on the number of stable jobs divided and counts for the working age population. We also construct a measure of average earnings by sex as the ratio between earnings across all permanent jobs to the total number of permanent jobs.

To supplement our analysis on economic hardship we leverage data on SNAP benefit receipt rates and applications from the Food and Nutrition Service of the Department of Agriculture. We use data on county-level participation in the month of January for all years spanning 1989–2020, focusing on beneficiaries of food stamps (FSP) and electronic benefit transfer (EBT) in the context of the program. We then aggregate the county-level counts to compute the share of beneficiaries in the population at the CZ level.[9] These

[8]The Natality Detail Files include information obtained from each U.S. birth certificate. There is some variation across states and over time in the availability of specific variables. In some cases, we exclude states from specific analyses due to data limitations; we note these exclusions when relevant.

[9]When information at the local level is not available, we impute the state-level share of SNAP

data allow us to test for differential trends before the epidemic but do not allow for the estimation of sex-specific impacts. To do so, we use the Quality Control (QC) database which contains detailed information on the economic and demographic circumstances of a sample of households selected for review as part of the SNAP QC System.

Geographic harmonization. We use the crosswalks developed by Autor and Dorn (2013) to aggregate all data to CZ level. Some CZs cross state borders; when this happens, the CZ is assigned to the state with the larger share of the CZ population. This criterion helps preserve the strong within-cluster and weak between-cluster commuting ties.

In sum, we build a dataset at the commuting-zone level, covering the period from 1980 to 2020 for our outcome variables and the measure of exposure to the epidemic. We choose commuting zones as our unit of observation since it is the geographic space that captures one's economic life—which often spans beyond county borders. We restrict our sample to areas with more than 25,000 residents. This represents 97% of the total population. Our final dataset is a panel of 587 commuting zones and consists of over 24,067 observations. Table 1 presents summary statistics for key variables for our analysis sample, in pre- and post-periods.

IV. Empirical Strategy

To identify the effect of the opioid epidemic on population changes we exploit rich geographic variation in the early promotional efforts of prescription opioids as an exogenous source of exposure. Following the approach in Arteaga and Barone (2025), we use 1996 cancer mortality as a proxy for local exposure to the opioid epidemic. The rationale behind this strategy is that the size of the cancer pain market in the mid-1990s was a key determinant of where pharmaceutical companies initially concentrated their marketing efforts for opioids. A large body of research—and multiple legal rulings—has established a strong link between these early promotional campaigns and the subsequent unfolding of the opioid crisis.[10]

Our empirical strategy interacts cancer mortality rates in 1996 with period dummies in a continuous-treatment event-study design, as follows:

$$\Delta\, y_{ct} = \sum_{\tau=\underline{t}}^{T} \phi_\tau\; CancerMR_{c,1996}\mathbf{1}(Period = \tau) + \sum_{\tau=\underline{t}}^{T} \alpha_t\; X_{ct_0}\mathbf{1}(Period = \tau) + \gamma_{st} + \upsilon_{ct}\ , \quad (1)$$

where c indexes the commuting zone, s the state, and period t. t_0 corresponds to 1996—the year of OxyContin's launch—or the closest year with available data; e.g., 2000

recipients.

[10] Appendix B presents additional empirical evidence probing the validity of this empirical approach.

when Census data are used. We define Δ as the long-change operator: for any random variable W_{ct}, $\Delta W_{ct} = W_{ct} - W_{ct_0}$.

The outcome of interest is given by y_{ct}. X_{ct_0} is a vector of baseline demographic variables such as the share female, White, and age groups. $CancerMR_{c,1996}$ is the cancer mortality rate in commuting zone c in 1996 and it is interacted with a full set of year fixed effects indexed by τ. In this specification, the coefficients for the pre-epidemic period, test whether outcomes in higher and lower cancer mortality areas followed similar trends before 1996. In the result section, we present scaled estimates of the ϕ_τ coefficients, we refer to these as *effect sizes*. Specifically, we multiply the estimated coefficient by the standard deviation of 1996 cancer mortality rates and divide by the mean of the outcome variable in the baseline year t_0. This yields the percent change in the outcome associated with a one standard deviation increase in exposure.

This research design allows us to control for state-specific trends and state-level policy changes which were common during this period, as well as time invariant commuting zone characteristics. This specification is equivalent to stacked cross-section estimates of Equation (1), and when estimated on a balanced panel, this to a regression in levels with commuting zone fixed effects. However, our panel is not perfectly balanced for some outcomes—e.g., fertility by marital status cannot be constructed for California after 2016. In such cases, the specification in Equation (1) is more efficient, and we adopt it as our baseline approach.

Identifying assumptions. Our causal effects of interest are average changes in outcomes (e.g., population growth rates) resulting from a marginal increase in exposure to the opioid epidemic. Our identification discussion follows Callaway et al. (2024) and we target Average Causal Response (ACR) parameters, which relies on three key identifying assumptions.

Our first assumption is that of *no anticipation*: exposure to the opioid epidemic must not affect outcomes prior to the unfolding of the epidemic (i.e., prior to 1996). Our second assumption is that of *strong parallel trends*: the average change in (potential) outcome associated with experiencing any level of exposure must be the same for all CZs, regardless of the level of exposure they actually experienced. Put differently, this assumption requires the standard parallel trends assumption (which only concerns untreated *dose* groups) to hold across all dose groups.[11]

We follow Callaway et al. (2024) and Goldsmith-Pinkham et al. (2020) and assess the plausibility of our assumptions using several complementary strategies. First, we test the exogeneity of our cancer-based exposure measure by examining pre-trends within a parametric event-study framework. We complement this analysis with a series of placebo

[11] In our context, the standard parallel trends assumption would require that, absent exposure to the opioid epidemic, the *untreated potential outcomes* in high- and low-exposure CZs would have trended similarly.

tests, out-of-sample exercises, and specifications that include additional controls for alternative local shocks (e.g., the China shock) and baseline commuting-zone characteristics such as industrial composition and poverty rates. These exercises also provide evidence of the exclusion restriction—that cancer exposure solely affects our outcomes via exposure to the opioid epidemic.

Finally, we further assess *strong parallel trends* and linearity (of treatment effects) by estimating treatment effects semi-parametrically *across the distribution of treatment exposure* in pre- and post-periods. In practice, we implement this using binned scatter methods following Cattaneo et al. (2024). Section VIII. describes in detail the results of these exercises and provide support for the validity of our identification strategy.

V. Population Decline and the Opioid Epidemic

In this section, we examine how exposure to the opioid epidemic translated into changes in local population growth. Panel (c) of Figure 1 plots population growth rates from 2000 to 2020 across CZs. The map shows that a large share of communities experienced population decline during this period, despite overall national population growth of 17.8% during this period. Figure 2 further illustrates these dynamics by showing the evolution of log total population (panel a) and log population aged 18 to 44 (panel b) for commuting zones in the top and bottom quartiles of opioid exposure. Before the onset of the epidemic, population growth rates were similar across exposure levels. Shortly thereafter, however, growth slowed markedly in more exposed areas—and even turned negative for the 18–44 age group.

To estimate the effect of the opioid epidemic on local population growth, we employ only decadal U.S. Census data on CZ population counts to avoid using intercensal data imputations. To account for the autoregressive structure of population growth (Greenland et al. (2019)), we estimate a generalized version of Equation (1) as follows:[12]

$$\triangle \ln(pop_{c,t}) = \phi \; CancerMR_{c,1996} + \beta \triangle \ln(pop_{c,t-10}) + \alpha X_{c,1990} + \gamma_s + \epsilon_{ct}, \qquad (2)$$

where $\triangle \ln pop_{c,t}$ represents the change in CZ's c log population between years t and $t-10$: $\triangle \ln(pop_{c,t}) = \ln(pop_{c,t}) - \ln(pop_{c,t-10})$. We first examine changes in log population between 1990 and 2000 to capture pre-epidemic differential trends across CZs. We then analyze changes between 2010 and 2000, and between 2020 and 2000. To capture long-term effects, we focus on 20-year changes in population, which helps avoid the inclusion

[12]To see this, consider that population changes could be model as an AR(1) process. That is, $\triangle \ln(pop_{c,t}) = \theta \triangle \ln(pop_{c,t-1}) + \mu_{ct}$. Not including the lagged population change when estimating the effects of exposure to the epidemic may mistakenly attribute effects of prior growth to this shock. Thus, the estimates of ϕ from the following regression may be biased: $\triangle \ln(pop_{c,t}) = \phi_t \; CancerMR_{c,1996} + \alpha X_{c,1990} + \gamma_s + \epsilon_{ct}$

of shorter-run lags already influenced by the epidemic. Specifically, when we define the outcome as the change in log population from 2020 to 2000, $\triangle \ln(pop_{c,2020})$, the relevant lag is the 20-year lag, $\triangle \ln(pop_{c,2000})$. We control for a vector of demographic characteristics $X_{c,1990}$ and state-level fixed effects.

Table 2 reports the estimated effects of exposure to the opioid epidemic on local population growth Equation (2). Each coefficient reflects the change in log population growth associated with a one–standard-deviation increase in 1996 cancer mortality, our measure of exposure. The table shows that prior to the onset of the epidemic, there were no systematic differences in population growth across exposure levels. Between 1990 and 2000, the estimated coefficients are small and statistically insignificant across all age groups, indicating that high- and low-exposure areas followed similar demographic trends before the diffusion of OxyContin. In contrast, after 2000—when the epidemic began to unfold—population growth diverged sharply. Between 2000 and 2010, commuting zones with higher exposure experienced 1.2 percentage points lower population growth, and by 2020 this effect grew to 2.5 percentage points. These declines are concentrated among individuals aged 18–44, whose population fell by 2.5 p.p. in the 2000–2010 period and by 4.1 p.p. between 2000 and 2020. Panels (b) and (c) of Table 2 split the population by sex. Effects are of similar magnitude through 2010; however, by 2020, the decline in population growth among men aged 18–44 (4.6 p.p.) exceeds that among women (3.7 p.p.).

Next, Table 3 examines how the effects of exposure to the opioid epidemic on population growth varied by educational attainment. Panel (a) presents results for individuals without any college education. The population declines in response to the epidemic were substantial in this group: between 2000 and 2010, high-exposure areas experienced a 2.1 p.p. decline in the 18–44 population, and by 2020 this effect had roughly doubled to 4.2 p.p. Declines are evident for both men and women and are of similar magnitude, with somewhat larger effects among men in the early period.

Panel (b) turns to individuals with some college education or more. Population declines are of similar magnitude in this group. Between 2000 and 2020, high-exposure areas saw a 3.8 p.p. decline in the 18–44 population with some college, compared with a 4.2 p.p. decline among those without college. Among women with some college, the estimated decline is larger than for men—4.2 p.p. compared to 3.6 p.p.—consistent with stronger migration responses among educated women documented below.

VI. Margins of Adjustment: Deaths, Moves, and Births

Having established the impacts on local population and economic outcomes, we now turn to potential explanations for these changes. We organize the discussion around three margins of adjustment. First, we explore a direct channel: deaths. Second, we examine

migration responses to the epidemic. Finally, we consider the role of fertility.

VI.a Deaths

The opioid epidemic has been characterized by record levels of drug-related mortality. Prior research has documented its large effects on excess deaths attributable to opioids. As such, mortality provides a natural starting point for unpacking the population decline we estimate. However, mortality effects are not unique to local health crises: studies examining exposure to import competition from China and Mexico have also found increases in mortality (Adda and Fawaz, 2020; Pierce and Schott, 2020; Finkelstein et al., 2025). In this section, we provide new estimates of the effects of the opioid epidemic on mortality across population subgroups, recognizing that both the magnitude and significance of these effects vary by sex and education level. We then assess the contribution of these direct mortality effects to the overall decline in population growth, both in aggregate and across demographic groups.

Panels (a) and (c) of Figure 3 show the evolution of drug-induced mortality rates for men and women, and for individuals with and without a college degree, across high- and low-cancer commuting zones. Men and those without a college degree consistently exhibit higher levels of drug-induced mortality. In 2018, the mortality rate among men was 2.5 times that of women. However, when examining the effects of exposure to the opioid epidemic, we find that the magnitudes are broadly similar across sexes (Panel b of Figure 3). By contrast, the differences by educational attainment are more pronounced: the estimated effects are statistically insignificant for those with any college education, but positive and statistically significant for individuals without any college education. By 2010, a one–standard-deviation increase in exposure led to a 57% increase in drug-induced mortality rates among the latter group. Table A1 reports coefficients grouped into three post-periods, facilitating clearer comparisons across subgroups.

To assess whether mortality impacts can explain the population decline estimated in Section V., we construct counterfactual population counts for commuting zone c in year t under the assumption that mortality from drug-induced causes is zero. This exercise provides a stringent *upper bound* on the contribution of mortality to the observed population decline, as it assumes the complete absence of opioid-related deaths. In other words, we simulate a scenario in which the opioid epidemic did not occur at all, rather than one in which we remove only the deaths attributable to exposure predicted by mid-nineties cancer mortality. The latter exercise is infeasible, as it would require estimating commuting zone– and time–specific treatment effects of exposure on mortality.

Specifically, we add back the cumulative number of deaths of individuals who would fall within the age bracket of interest to the observed population counts. For instance, when analyzing the population aged 18 to 44 years, the counterfactual population count

is given by:

$$\widetilde{\text{pop}}_{ct,18-44} = \text{pop}_{ct,18-44} + \text{drug-induced deaths}_{c,[18-44]_t} \tag{3}$$

where drug-induced deaths$_{c,[18-44]_t}$ is the sum of deaths from drug-induced causes among people who would be between 18 and 44 by year t.[13] In figure A3 we plot observed population trends and the counterfactual counts and Table 4 shows the results when we re-estimate Equation 2.

For the population 18 to 44, between 2000 and 2010 the observed effect is –0.0247, compared to –0.0242 under the counterfactual, implying that drug-induced deaths account for roughly 2% of the total decline. By 2020, and as drug deaths accumulate at higher rates, the importance of direct mortality effect increases. The observed effect is –0.0416 and the counterfactual –0.0277, suggesting that mortality explains about 33% of the population loss. When disaggregated by sex, the results show that mortality accounts for a similar share of the population decline among men and women. Table 5 further disaggregates the results by education; showing that mortality accounts for a larger share of the population decline in the population without any college education.

Taken together, these findings indicate that although the opioid epidemic generated severe mortality shocks—particularly among less-educated adults—these deaths explain only part of the overall population losses in high-exposure areas. To understand how the remaining adjustment occurred, we next examine migration changes.

VI.b Moves

Migration is often considered a primary margin of adjustment to local economic shocks (Blanchard et al., 1992). Prior research, however, has found only limited migration responses to major economic disruptions such as trade liberalization or recessions (Autor and Dorn, 2013; Yagan, 2019; Borusyak et al., 2022). The opioid epidemic differs from these shocks in both its geographic and industrial scope, as well as in the social distress it generated alongside its economic impacts—factors that may have produced a distinct migratory response. We now examine how local exposure to the opioid epidemic affected in- and out-migration patterns across commuting zones.

To capture the bilateral nature of migration, we analyze origin-destination migration flows between pairs of locations and allow for migration to depend on both origin and destination shocks and characteristics (Borusyak et al., 2022). Our regression of interest for an annual cross-section of CZ-pairs in year t is:

[13] In this computation we accumulated deaths from 1989, the first year of the mortality data. While this approach could introduce truncation concerns, this issue is mitigated by the fact that deaths from these causes began to rise only after 2000.

$$\Delta\, y_{ol,t} = \phi_{o,t}\, Cancer_{o96} + \phi_{l,t}\, Cancer_{l96} + \alpha_{o,t}\, X_{o96} + \alpha_{l,t}\, X_{l96} + \gamma_{so,t} + \gamma_{sl,t} + \upsilon_{ol,t}\ . \quad (4)$$

$y_{ol,t}$ is the log probability of migrating from CZ o to l in year t, and $\Delta\, y_{ol,t}$ is the change in this variable between year t and the baseline year (t_0). $Cancer_{o96}$ and $Cancer_{l96}$ measure exposure to the epidemic in the origin and destination, respectively.[14] $\gamma_{so,t}$ and $\gamma_{sl,t}$ are origin and destination state fixed effects. We include demographic controls in both origin and destination commuting zones at baseline (X_{o96} and X_{l96}). We cluster standard errors two-way at the origin and destination level.[15]

$\phi_{o,t}$ and $\phi_{l,t}$ are the main coefficients of interest and can be interpreted as migration semi-elasticities with respect to epidemic exposure: all else constant, a unit increase in origin (destination) cancer mortality leads to a $\phi_{o,t}$ ($\phi_{l,t}$) percent change in the probability of out- (in-) migration by year t, relative to the baseline year.

Figure 4 presents semiparametric estimates of $\phi_{o,t}$ (Panel (a)) and $\phi_{l,t}$ (Panel (b)) for inter-county migration using IRS data on the full population of tax filing adults, using binned scatter plots following the method of Cattaneo et al. (2024).[16] We group the data into deciles of exposure and focus on changes in migration rates across deciles (i.e., the slope). Consistent with an absence of pre-trends across the distribution, both Panels (a) and (b), show no relation between changes in exposure and changes in migration rates from 1993—1994. By 2009—2010, a distinct positive slope emerges in Panel (a): increases in origin exposure lead to increased out-migration rates. This effect persists through 2019—2020. In contrast, Panel (b) reveals no evidence of a relationship between destination exposure and in-migration rates in either of the post-epidemic periods indicating $\phi_{l,t} \approx 0$.

Figure 5 presents parametric estimates of $\phi_{o,t}$ from Equation 4. To ease interpretation, coefficients are scaled by the standard deviation of cancer mortality in 1996. Panel (a) is a parametric version of Figure 4(a) for all years. We find a one-standard deviation

[14] Consistent with, e.g., Moretti and Wilson (2017) (among others), we study proportional changes in migration rates thereby focusing on location-pairs with migration connections (non-zero flows) in 1996. While this significantly decreases the number of location-pairs in our sample, this has almost no bearing on the number of moves observed. In our IRS migration data, 96.5% of the moves occur between counties with non-zero flows in 1996.

[15] Our use of cross-sectional regressions in our migration analysis is motivated by computational and notational efficiency. It can be readily shown that estimating Equation 4 over all cross-sections is numerically equivalent to a stacked "event-study style" panel regression, analogous to Equation 1.

[16] The IRS migration data suppresses county-to-county migration flows below certain thresholds (e.g., < 10 prior to 2012) for confidentiality purposes. The thresholds only suppress a small share of the overall moves—we observe 85% of the moves throughout our sample period—however, differential missingness across CZs makes aggregating to CZ-to-CZ migration flows challenging, as this can sometimes aggregate suppression. This is not a concern for inter-county migration therefore, at this paper's current stage, when using the IRS migration data we focus on inter-county migration. The mean and median share of movers we observe across counties is 70% and there is no correlation between the share of moves observed and epidemic exposure.

increase in exposure to the epidemic led to a 3% increase in out-migration rates in 2010; this effect increased to nearly 4% in 2011 and persisted thereafter. Panel (b) presents estimates of Equation 4 for inter-CZ migration using data from the ACS and decennial census, separately by sex.[17] Out-migration effects are more pronounced among women: a one-standard deviation increase in exposure led to an 8% increase in out-migration rates among women in 2010 with no detectable effects among men. This effect grew slightly over time, reaching 13% by 2017. Panels (c) and (d) present estimates by educational attainment separately for women and men, respectively; among both men and women we find that out-migration responses are concentrated among those with some college education, but we only detect statistically significant effects among women.

Where Do People Move? We characterize the destination of these moves in terms of their exposure to the opioid epidemic using the origin-by-destination migration data from the IRS. We group origin communities into decile of exposure groups (g). For each group, we compute the migration-weighted average destination-exposure, where weights ($w_{o,l}$) correspond to the share of movers from a given origin o who relocated to destination $l \neq o$ ($m_{o,l}$):

$$\overline{\text{Exposure}}_g^{dest} = \sum_{o \in g} \sum_{l} w_{o,l} \, \text{Exposure}_l \quad \text{where} \quad w_{o,l} = \frac{m_{o,l}}{\sum_{l' \neq o} m_{o,l'}}$$

Figure 6 illustrates this relationship. For each decile of exposure, the squares plot the average exposure to the opioid epidemic in the origin commuting zone (x-axis) against that in the destination commuting zone (y-axis), based on 2010 migration patterns. The figure shows that for communities above the third decile of exposure, migration reduced exposure to the epidemic, as all squares lie below the 45-degree line.

To assess whether these destination choices were specific to the opioid epidemic, we compare the exposure of 2010 destinations to historical (pre-epidemic) migration patterns. Specifically, we examine whether migrants from high-exposure areas began moving to new destinations after the onset of the crisis or continued relocating to the same places as before. Using 1994 migration flows—when the epidemic had not yet emerged— and which are represented by the circles in Figure 6, we find that migrants in 2010 largely moved to the same destinations as they had historically. Thus, while greater exposure increased the frequency of moves, it did not alter where people moved: the set of destinations remained stable.

This result underscores that migration decisions are often constrained by established

[17] Our inter-CZ migration analysis uses 5-year data from the 2000 decennial census and 1-year data from the ACS. For comparability, we pool ACS data into multi-year windows (e.g., 2005–2010) over which the underlying Public Use Microdata Area (PUMA) geographies, used for mapping to CZs, remain constant.

social and informational networks (Borusyak et al., 2022; Porcher, 2022). To further examine whether individuals selected relatively less exposed destinations within these existing networks, we construct a placebo exercise that benchmarks the lowest possible destination exposure among the top five locations where migration flows had historically been established. In this *optimal* migration pattern, each origin's top five historical destinations are ranked by their exposure to the epidemic, and the lowest-exposure destination is identified as the "best" option. The triangles in Figure 6 illustrate this comparison. The gap between the optimal and actual destinations shows that there was substantial scope for migrants to relocate to less exposed areas within their existing networks, yet they did not. Even when restricting the choice set to historically established destinations, individuals did not disproportionately move to the least exposed locations.

In summary, exposure to the opioid epidemic led individuals to move more frequently but not to new or less affected destinations. Although migrants generally ended up in less exposed locations in absolute terms, these movements primarily reflected historical migration patterns rather than a targeted response to the epidemic.

Implications for regional convergence. Our finding of significant out-migration responses to epidemic exposure is consistent with standard labor supply models (e.g., Blanchard et al., 1992). Different from these models, however, is our finding of exposure serving solely as a "push" factor for migration.[18] This lack of directed migration negates the role of migration as an equilibrating force to dissipate regional disparities over time. Consistent with this implication, throughout this paper we document persistent, and often growing, disparities between CZs along the dimension of epidemic exposure continuing through 2020. This finding coincides with the general lack of regional income convergence seen in the U.S. of the time period of our study (e.g., Autor et al., 2025; Ganong and Shoag, 2017).

Magnitudes and back of the envelope computations. We conduct a back-of-the-envelope exercise to assess whether these migration responses are large enough to account for the observed population decline. The core idea of our exercise is that absent changes in mortality, changes in population growth rates attributable to the opioid epidemic can be decomposed into excess in- and out-migration induced by the epidemic.[19] For simplicity, we assume that, absent the opioid epidemic, inter-CZ migration would be net-zero. In such a scenario, the change in population growth attributable to epidemic-induced migration can be expressed as:

[18] See Claassen (2025) on the implication in standard labor supply and migration models that local shocks serve symmetrically as both a push- and pull-factor.

[19] Since the IRS migration data includes the population of tax-filers, changes in fertility would not yet have scope to affect this population (i.e., the "excess births" would not yet be tax-filers).

$$\Delta\text{Pop. Growth}_{c,T} = ln\left(\Pi_{t=2000}^{T}\left(1 - \underbrace{\text{Out-Mig. Rate}_{c,t_0} \times \phi_{o,t}}_{\text{Excess out-mig.}} + \underbrace{\text{In-Mig. Rate}_{c,t_0} \times \phi_{l,t}}_{\text{Excess in-mig.}}\right)\right) \quad (5)$$

We use as inputs: (i) Out-migration rate of 7% which corresponds to the average out-migration rate in 1996; (ii) Parametric annual estimates of $\phi_{o,t}$ from 2000 to 2020 (shown in Figure 5(a)); and (iii) Semi-parametric estimates of $\phi_{l,t} = 0$ (shown in Figure 4(b)).

Since mortality effects of the epidemic mostly began unfolding after 2010 (Table 4) we first focus on 2000–2010. In this period, out-migration responses from a one-standard deviation increase in exposure led to a 1 percentage point reduction in population growth rates. Over the same period, estimates of Equation 2 among the population aged 30 or above, proxying the tax-filing population, indicate a one-standard deviation increase in exposure led to a 1.2 percentage point reduction in population growth rates. Thus, from 2000–2010, migration responses can account for nearly all of the decline in population growth (among adults) in areas more exposed to the opioid epidemic.

Extending the exercise to 2010–2020, while accounting for the excess mortality induced by local exposure to the epidemic, suggests migration responses can generally account for the *non-mortality* portion of population decline occuring in more exposed areas over this period.

VI.c Births

Fertility constitutes a third major margin through which the opioid epidemic may have altered local demographic trajectories. Beyond its effects on mortality and migration, the crisis could have influenced reproductive behavior through changes in economic stability, health, and household formation. We next analyze how fertility evolved across areas with different levels of exposure to the epidemic.

We begin by examining the evolution of fertility rates in areas more exposed to the epidemic—those in the top quartile of 1996 cancer mortality—relative to areas with lower exposure, corresponding to the bottom quartile. Panel (a) of Figure 7 shows that prior to the crisis, fertility rates in high- and low-exposure communities followed similar trends, albeit from different levels, with low-exposure areas exhibiting higher fertility. As the epidemic unfolded, however, fertility declined more sharply in low-exposure communities. Estimating Equation (1), we find that exposure to the opioid epidemic led to a substantial (relative) increase in fertility among women of reproductive age. Panel (b) of Figure 7 shows that a one standard deviation increase in exposure—proxied by local 1996 cancer mortality rates—led to an approximately 5.5% increase in the overall fertility rate among women aged 15 to 44 by 2018, relative to its 1996 baseline. These effects start to be significant in 2005, and have steadily grown since then. These relative increases in fertility

are driven by women aged 25 to 29. Figure A4 presents a breakdown of the fertility results by age and shows that this is the only group in which we observe a significant effect of exposure to the epidemic.

Panel (a) of Figure A5 presents the effects of exposure on fertility by marital status. We find that a one–standard deviation increase in exposure raised fertility among unmarried women by 4.7% by 2018, compared to a 2.8% increase among married women. The dynamics of these effects also differ meaningfully: the increase in fertility among unmarried women becomes statistically significant starting in 2005, whereas it takes an additional eight years—until 2013—for the effect on married women's fertility to reach statistical significance.

When we disaggregate the effects by education level, we find that the increase in fertility is more pronounced among women without any college education, particularly during the decade from 2005 to 2015. Panel (b) of Figure A5 shows that in areas more exposed to the opioid epidemic, fertility among women without a college degree increased by 5% by 2010. In contrast, the increase among women with some college education was only 1.8% over the same period. Over time, fertility rates for both groups continued to rise. By 2018, fertility had increased by 6% among women without any college education and by 5% among those with some college or more.

These effects on fertility rates have two important implications. First, in terms of population declined, the direction of these changes indicate that (relative) higher fertility induced by the epidemic could attenuate its impacts on population growth. Second, the heterogeneous fertility changes will contribute to long term changes in population composition in areas more affected by the epidemic.

A potential direct channel linking the opioid epidemic to fertility operates through opioid use disorder (OUD). Drug use is associated with lower contraceptive use and higher rates of unintended pregnancy (Terplan et al., 2015). Consequently, exposure to the epidemic could, in principle, increase fertility if it led to reduced contraception. However, the magnitude of this channel is likely limited. In 2018, 0.9% of women aged 15–44 had an OUD NSDUH (2020), while births diagnosed with neonatal abstinence syndrome (NAS) accounted for 0.6% of all births (CDC, 2023*a*). Given that withdrawal symptoms develop in 55% to 94% of neonates exposed to opioids in utero (Hudak et al., 2012), a back-of-the-envelope calculation suggests that women with OUD have fertility rates approximately 88%, thus lower than the general population. An alternative mechanism could operate through changes in abortion rates or access to abortion services. Using data from the Myers Abortion Facility Database, we find no evidence of changes in abortion rates as a function of exposure to the epidemic (Figure A6). Moreover, we do not observe any differential effects between areas with high and low access to abortion services.

VII. Mechanisms: Economic Effects

So far, we have shown that the opioid epidemic induced substantial demographic changes that extend beyond the direct effects of excess mortality. Understanding the mechanisms behind these changes, particularly, the migration responses of women and the rise in fertility, requires examining how the epidemic generated differential economic impacts by sex. Economic theory predicts that shocks to local labor markets can affect fertility differently for men and women (Becker, 1973). For men, economic hardship typically reduces fertility through an income effect, as lower earnings decrease the ability to support a family. For women, by contrast, declines in labor market opportunities can reduce the opportunity cost of childbearing, potentially increasing fertility if the substitution effect dominates. Motivated by these insights, we next examine the economic hardship induced by the opioid epidemic, focusing on how its effects differ for men and women.

We consider a more parsimonious specification that aggregates effects for time periods but still permits some heterogeneity. We define four time bins [1990-1995], [1996 - 2000], [2001- 2010], and [2011 - 2018]:

$$\begin{aligned}\Delta y_{ct} = \phi_{95}\, CancerMR_{c,1996}\, D_{[90\text{–}95]}(t) + \phi_{10}\, CancerMR_{c,1996}\, D_{[01\text{–}10]}(t) \\ + \phi_{18}\, CancerMR_{c,1996}\, D_{[11\text{–}18]}(t) + \sum_g \alpha_g X_{ct_0}\, D_g(t) + \gamma_{st} + \nu_{ct},\end{aligned} \tag{6}$$

where $D_g(t) = \mathbf{1}\{t \in g\}$ is the indicator for time bin g, and the excluded category is 1996-2000 to ease comparison with the rest of the analysis. The rest of the variables are defined as in Equation (1).

Employment and earnings. Changes in population growth might alter local labor markets and thus may confound estimates of the *direct* impact of the epidemic on economic conditions. To see this, note that the employment-to-population ratio can be decomposed as follows:

$$\frac{emp_t}{pop_t} - \frac{emp_0}{pop_0} = \underbrace{\frac{emp_t - emp_0}{pop_t}}_{\text{net emp change}} + \underbrace{\frac{emp_0}{pop_t} - \frac{emp_0}{pop_0}}_{\text{effect of pop change on emp/pop}} \tag{7}$$

We document effects on *net* employment growth defining the outcome of interest as the change in log employment in year t relative to log employment in 2000 (top panel of Figure 8). In Figure A1, we present estimates of the change in the employment-to-popuation ratio.

Before 1996, we find no significant differences in employment changes between men and women in more and less exposed communities. By 2010, both groups experienced declines in employment growth, and this trend persisted through 2018. A one standard deviation increase in exposure to the epidemic led to a reduction in employment growth of

2-percentage-points or 18% among women and and 1.4 percentage-points or 13% among men about 10 years after the onset of the epidemic (see panel (a) of Figure 8). The declines persisted, and by 2018 we observe a reduction in employment growth of 6 percentage points for women and 4 percentage points for men. When we disaggregate the outcomes by education, we find that the opioid epidemic led to larger reductions in employment growth among non-college individuals than among college-educated individuals.

Next, we analyze changes in the employment-to-population ratio, negative estimates in this outcome would indicate that the relative losses in employment were stronger than those in population (Figure A1). We find no detectable effects of the opioid epidemic on this ratio for men. For women, we estimate negative effects, though they are not statistically significant. However, when we split the data by education, we observe that among women without a college education, the opioid epidemic induced a continuous decline in the employment-to-population ratio. By 2018, a one standard deviation increase in exposure to the epidemic led to an 8% reduction in employment-to-population ratio for this group. We estimate a smaller impact of the opioid epidemic on men without college education, a one standard deviation increase in exposure to the epidemic lead to a 3% reduction in employment-to-population.

Given these extensive margin changes, we ask how the earnings of these populations have changed. We find no detectable earning effects for women or men. When we zoom into education groups, we find changes that are consistent with women with some college education facing lower average earnings and displacing less educated women out of the labor force. We find that the epidemic did not affected men earning significantly.

How large are the employment and earning estimates? Our estimates are difficult to compare to earlier work because of differences in identifying variation, we target the *direct effects* of increased access to opioids rather than the effects of *policies* implemented to curb the epidemic on these outcomes.[20] Our variation is closer to that induced by the triplicate state variation: Alpert et al. (2022), find that opioids distribution was approximately 50% lower in triplicate states in the years after the launch of OxyContin.[21] Exploiting this variation, Powell (2021) and Mukherjee et al. (2025) find comparable effects, thought their results are not desagregated by sex.[22] Our results are also comparable to the employment effects of NAFTA vulnerability, Choi et al. (2024) estimates that the average county in the top vulnerability quartile lost about three to

[20] See: Currie et al. (2019); Park and Powell (2021); Kaestner and Ziedan (2023)

[21] Arteaga and Barone (2025) finds that one-standard-deviation increase in the 1996 cancer mortality rate led to an additional 0.97 opioid doses prescribed per capita, which is 65% higher than the baseline mean.

[22] Powell (2021) estimates that triplicate states had an additional 3.8 percentage points of labor force participation, on average, due to its reduced exposure to prescription opioids. Mukherjee et al. (2025) document that triplicate states experienced a 8.2% increase in the flows from unemployment to employment between 1996 and 2019, which translates to a 1.11 percentage point increase in the steady-state employment-to-population rate.

seven log points of employment compared to the average county in the bottom quartile.

Reliance on public transfer programs. The reduction on economic opportunities could increase the need for participation in public transfer programs. Arteaga and Barone (2025) show that, by 2020, in communities more affected by the opioid epidemic the populations applying for SSDI and SSI benefits rose by 15% and of SSI by 3.2% per one standard deviation increase in exposure. The corresponding increase in the proportion of the population receiving SNAP benefits in these CZs was 8%. We extend these analyses by studying differential responses of men and women. These results are informative not only on the impact of exposure to the epidemic on adult wellbeing, but also shed light on the forces shaping the migration responses we study in the next section. Notowidigdo (2020) shows that aggregate transfer program expenditures are highly responsive to local labor market conditions compensating low-skill workers and making them less likely to out-migrate. Panel (a) of Figure A2 provides evidence that prior to the onset of the opioid epidemic, receipt rates evolved similarly across communities with different baseline cancer mortality. Following the epidemic's emergence, 1996 cancer mortality predicts a continuous increase in SNAP takeup in the subsequent two decades. In panel (b) we leverage data on the sex of applicants to document that this increased takeup was mainly driven by women.

VIII. Robustness Checks

In this section, we provide evidence in support of our identification strategy, explore alternative explanations for our findings and test the sensitivity of our results.

VIII.a Evidence on the Exogeneity Assumption and Exclusion Restriction

The validity of the identification strategy requires that, in the absence of prescription opioid marketing, areas with higher cancer mortality in 1996 would have followed the same trends in health and economic outcomes as areas with lower cancer mortality—all along the support of the 1996 cancer mortality distribution. To support this assumption, we present several exercises.[23]

Out-of-sample. This exercise tests whether lagged cancer mortality predicts future population growth and migration prior to the onset of the opioid epidemic. In Table A3 we show that 1976 cancer mortality does not predict population growth between 2000 and 1990, i.e., there is no relationship between cancer mortality rates and growth in the next 20 years or so. Similarly, in panel (a) of Figure 9, we find that cancer 1976 is not predictive of out-migration probabilities in 1994 to 1996—the estimated coefficients are

[23] In this draft we focus on population growth and migration as the main outcomes of analysis. Arteaga and Barone (2025) provide complementary evidence on the validity of this strategy and present results for these tests on mortality, economic, and political outcomes.

statistically indistinguishable from zero, and there is no evidence of a pattern—providing evidence of a lack of mechanical relationship between cancer mortality and moves.

Placebo. We further probe the validity of our design by estimating event-study regressions with placebo instruments—i.e., mid-1990s mortality from causes unrelated to cancer.[24] Finding a good placebo instrument is challenging given that the causes that underlie the incidence of cancer and of other conditions such as heart disease are not independent (Chiang, 1991; Honoré and Lleras-Muney, 2006). As a result, there is substantial overlap across underlying causes, and the correlation across measures is very high, especially among elderly age groups. With this caveat, in Table 6 we present placebo instrument regressions for mortality from hypertension and pneumonia, which are less likely to be affected by the previous concern but still capture community-level health trends. We find no relationship between mortality from pneumonia and population growth. When looking at hypertension, we find some statistically weak predictive power of this measure on the 20 year population growth rate. These coefficients are less than half the size of the baseline estimates and are only significantly different from zero at the 10 percent level. Panel (b) of Figure 9 presents this exercise for migration rates, the event study graph shows no effects of placebo mortality on this outcome.

Additional covariates. We also expand the set of covariates to account for health behaviors, access to healthcare and economic factors and the correlation of these with cancer. Panel (c) of Figure 9 presents baseline estimates along with four additional models. First, we control for the share of population older than 65 years, since this is a strong predictor of cancer mortality. Second, we control for contemporaneous cancer mortality rates to account for the association between cancer and other socio-economic outcomes. Third, we expand the set of controls in our baseline to include the share of smokers, the share of adults with overweight, the share of primary care physicians, and the infant mortality rate, all measured at the CZ level at baseline or the earliest available data point. Four, we expand the set of controls to account for economic covariates, these controls include the unemployment rate, the share of employment in the manufacturing sector, the poverty rate, and the share of the population that has completed some college. Panel (c) of Figure 9 shows that our results are robust to the inclusion of these covariates.[25]

Alternative samples. Finally, we assess whether our results are sensitive to the exclusion of any single state. Figure 10 shows the estimated effect on out-migration probabilities for the year 2010 when we exclude one state at the time from the sample. This figure provides evidence that our results are not driven by a particular state, estimates are similar to the full sample ones.

[24] These placebo instruments are also known as negative instruments (Danieli et al., 2023).

[25] All of these controls are measured at the CZ level at baseline or the first available year.

VIII.b Exposure to Economic Shocks

We account for geographic exposure to major economic shocks that could confound the relationship between opioid exposure and our outcomes. For each shock, we include a measure of local exposure interacted with year dummies to flexibly control for its varying geographic impact.[26] Contemporaneous with the unfolding of the opioid epidemic, the United States economy was affected by the formation of the NAFTA, rising import competition from China and two major recessions (2001 and 2007–09). These developments could influence relocation decisions. To address rising import competition from the NAFTA and from China joining the WTO, we control for exposure to the regional import competition measure from Choi et al. (2024) and Pierce and Schott (2020) respectively. Exposure to the 2001 and Great Recession is measured using changes in CZ-level unemployment around each downturn, as in Yagan (2019). Panel (d) of Figure 9 presents these estimates and provides evidence that our results are not capturing exposure to these other local shocks.

IX. Discussion

This paper documents how the opioid epidemic—one of the most consequential public health crises in recent U.S. history—reshaped fundamental demographic processes through changes in internal migration and fertility. Using quasi-exogenous variation in local exposure to Purdue Pharma's marketing of OxyContin, we show that communities more heavily exposed to the epidemic experienced both significant selective outmigration—concentrated among women and college-educated adults—and substantial increases in fertility, particularly among younger, unmarried, and less-educated women. Together, these shifts reveal how the epidemic altered the composition of local populations and, in doing so, contributed to the growing regional demographic divergence within the United States.

Our findings point to a novel mechanism through which public health crises shape long-run social and economic trajectories. The demographic impact of the opioid epidemic operated not only through excess mortality but also through economic and behavioral responses that influenced migration and family formation. This highlights the close interconnections between health and economic inequality: communities most exposed to opioids not only suffered higher mortality but also lost key segments of their working-age and educated populations. In this sense, the epidemic amplified regional disparities and entrenched patterns of economic stagnation and demographic decline.

[26] We follow the literature to construct these measures; (Arteaga and Barone, 2025) provides the details. Correlations with opioid exposure range from –0.04 to 0.18: NAFTA: –0.04, China shock: 0.18, 2001 recession: 0.05, Great Recession: 0.02.

A central contribution of this work is to underscore the gendered nature of these adjustments. The economic hardship induced by the epidemic fell disproportionately on women, in contrast to the pattern observed in other regionally concentrated shocks—such as trade or automation—which primarily affected men. These differences help explain the distinctive demographic responses we observe: unlike most negative economic shocks, the opioid epidemic generated both substantial migration responses and increases in fertility. These gender differences open new avenues for understanding how the intersection of health and economic shocks reshapes family and labor dynamics. They also raise important questions about why women bore a disproportionate share of the economic burden of the epidemic, even though men were more likely to die directly from opioid overdoses.

Future research could build on our findings by examining how these shifts in migration and fertility interact with other long-run outcomes, such as child development, educational attainment, and intergenerational mobility. Another promising avenue involves studying how institutional responses—such as access to addiction treatment, economic support programs, or housing policies—mitigated or amplified these effects across places and populations.

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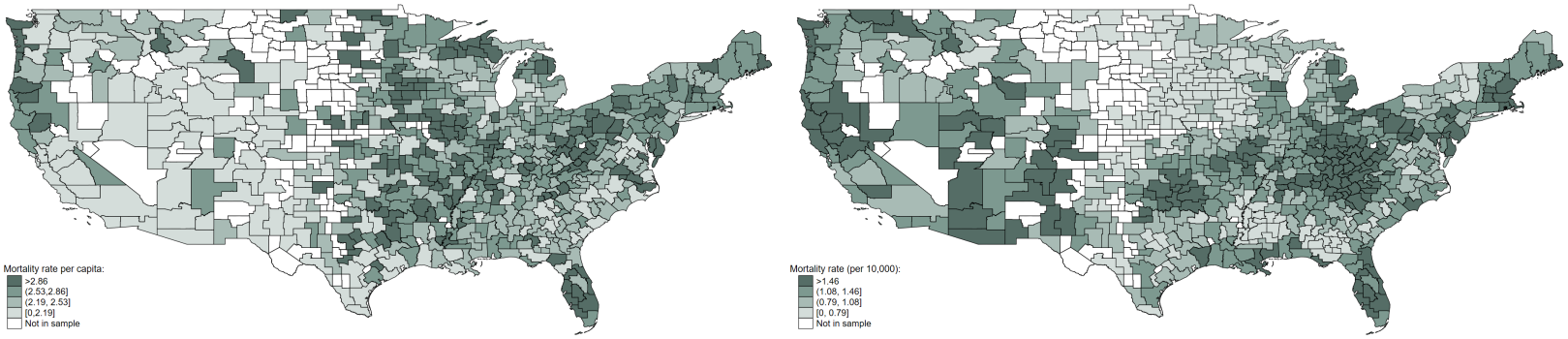
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X. Figures

Figure 1: Geographic Variation

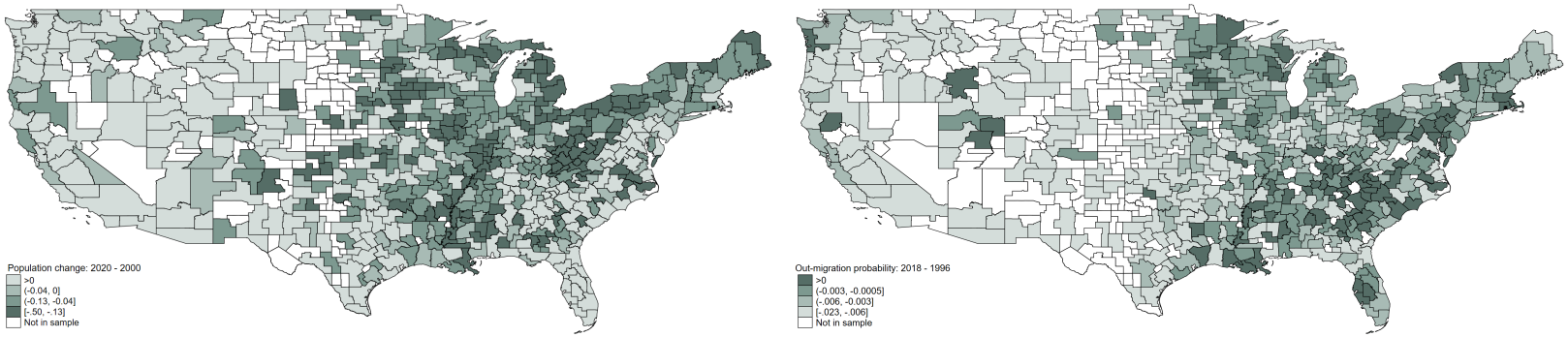
(a) Cancer Mortality Rate, 1996

(b) Drug-Induced Mortality per 10,000, 2018-1996



(c) Population change, 2020-2000

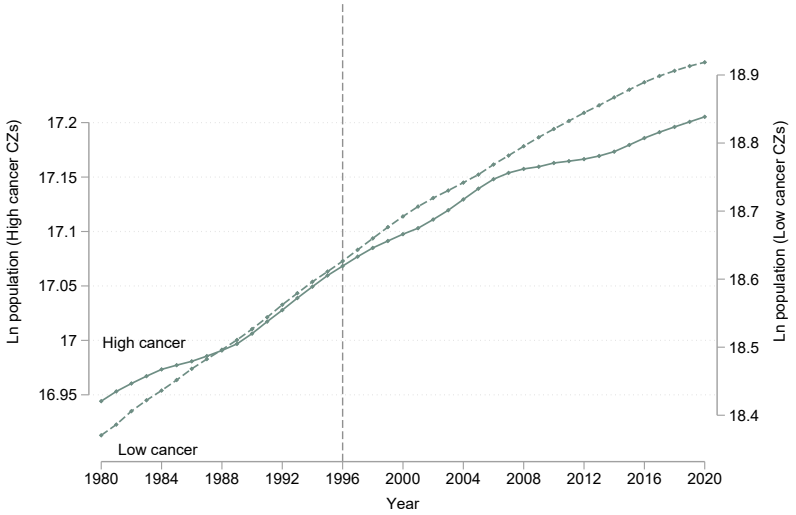
(d) Out-migration Rate, 2018-1996



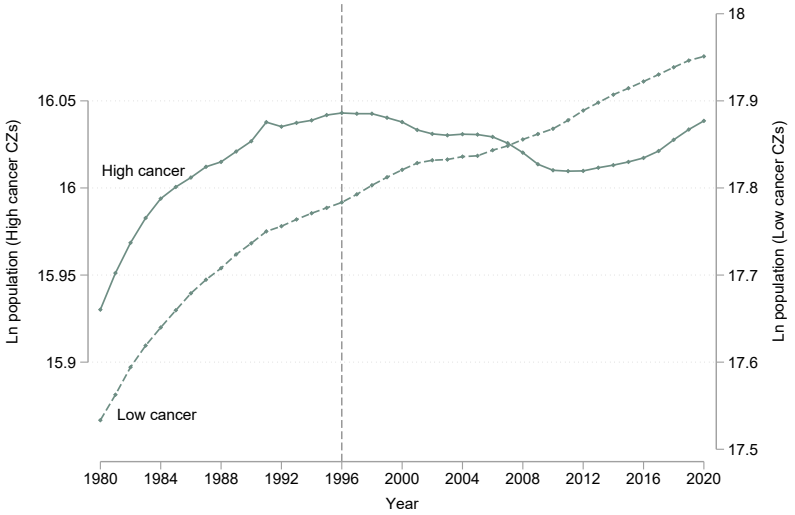
Notes: Panel (a) of this figure shows the geographic distribution of the cancer mortality rate (per 1,000) in 1996, our measure of exposure to the opioid epidemic. Panel (b) shows the geographic distribution of drug-induced mortality (per 10,000) between 1996 and 2018. Panel (c) shows the change in the (natural log of) population aged 18 to 44 years old between the decennial years 2020 and 2000. Panel (d) shows the change in out migration probabilities from 1996 to 2018. The break points correspond to the quartiles of the variable's distribution. In the bottom panels, we use zero as a break point to ease interpretation of population decline (panel c) and net-out migration (panel d). The sample is restricted to areas with more than 25,000 residents in 1996; these account for more than 97% of the total population in 1996. Commuting zones not included in the sample are white in the figure. We use geocoded National Vital Statistics System (NVSS) data to construct death counts; see Section III. for the ICD codes used. This figure is referenced in Section III.

Figure 2: Evolution of Local Population in High- vs Low-Exposure Commuting Zones

(a) Overall Population



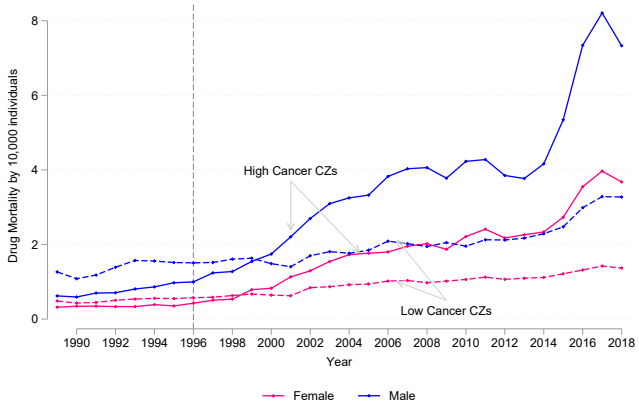
(b) Population aged 18-44 years



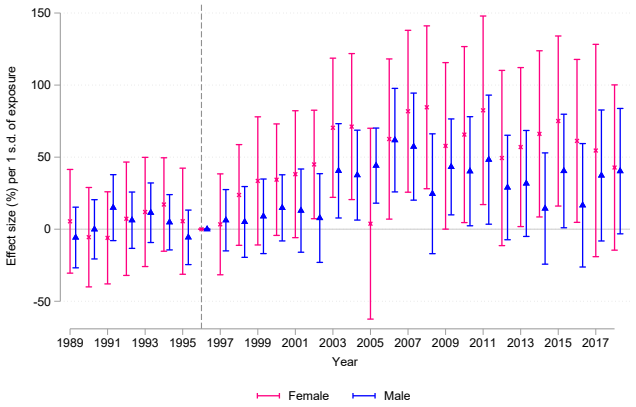
Notes: This figure shows the evolution of log total population and the log population aged 18 to 44 years in CZs in the bottom (dashed lines) and top (solid lines) quartiles of cancer mortality before the launch of OxyContin. This comparison is based on dichotomous high-versus-low categorization of CZs, and the outcome is weighted by population. The series for high-cancer-mortality CZs are plotted on the left vertical axis, while those for low-cancer-mortality CZs are shown on the right. This figure is referenced in Section V.

Figure 3: Exposure to the Opioid Epidemic and Drug Induced Mortality by Sex and Education

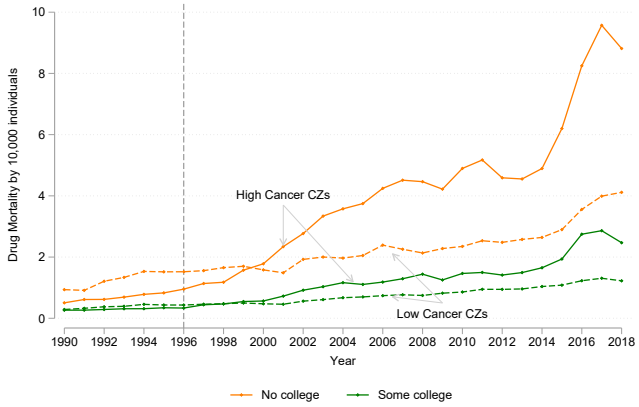
(a) Trends in Drug Induced Mortality by Sex



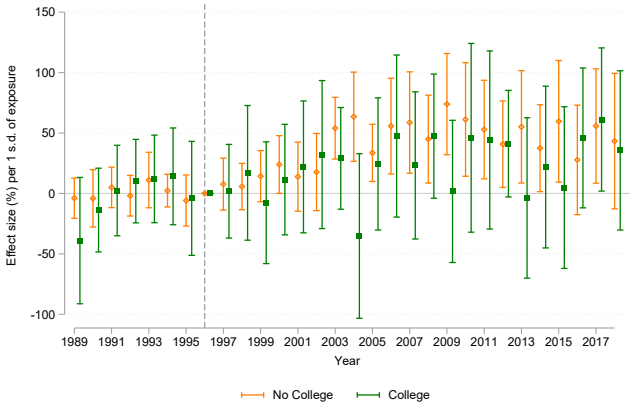
(b) Event-study by Sex



(c) Trends in Drug Induced Mortality by Education

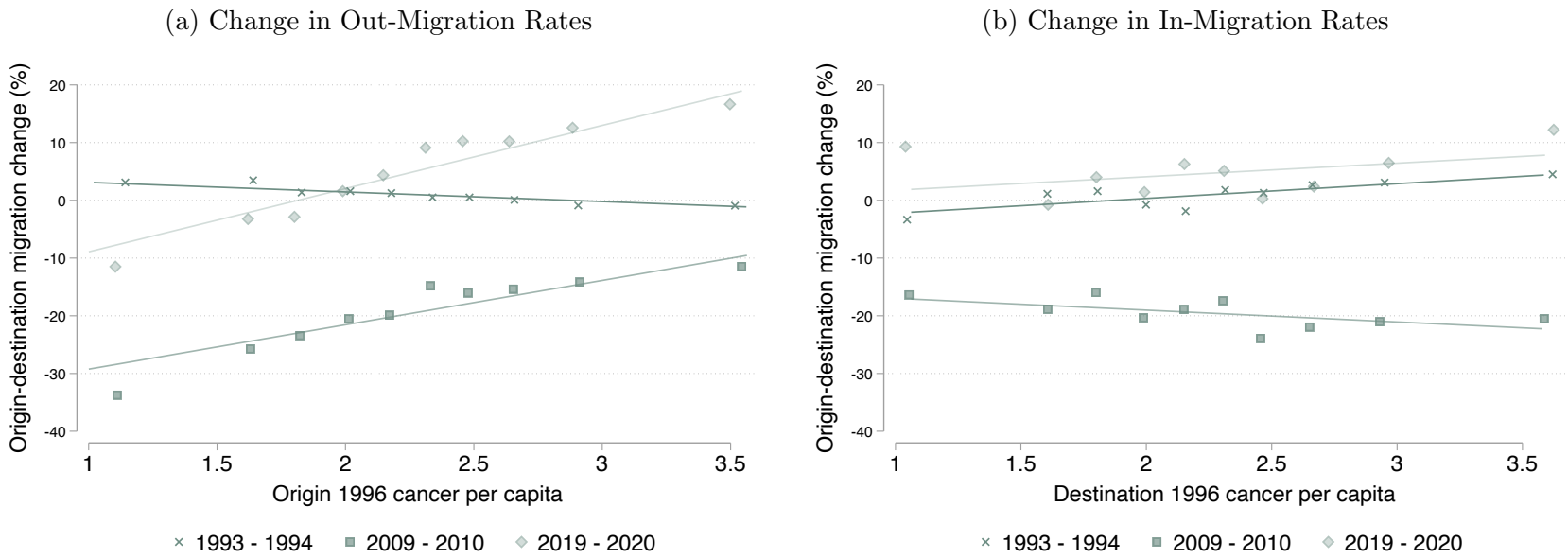


(d) Event-study by Education



Notes: This figure shows the effects of the opioid epidemic on drug-induced mortality of women and men aged 20 to 44 years old. The effect size (expressed as a percentage) corresponds to the scaled coefficients from Equation (1). Specifically, we multiply the estimated coefficient by the standard deviation of 1996 cancer mortality rates and divide by the mean of the outcome variable in 1996. These estimates exploit continuous variation in cancer mortality in 1996 and control for baseline characteristics and state-by-year fixed effects. Standard errors are clustered at the CZ level. This figure is referenced in Section VI.a.

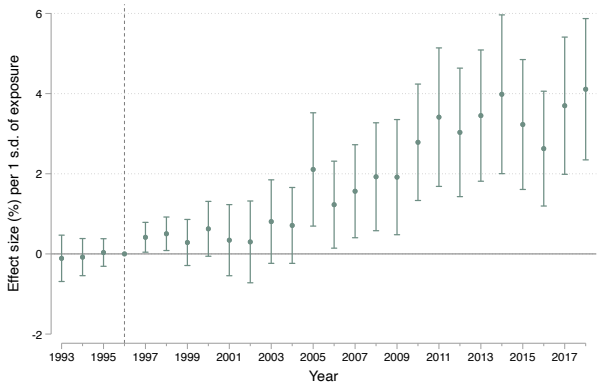
Figure 4: Effects of the Opioid Epidemic on Out- and In-Migration (IRS)



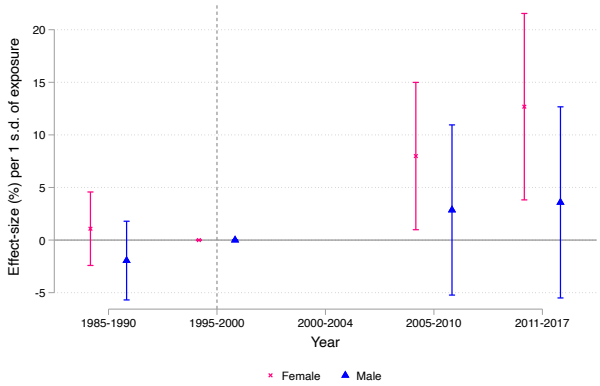
Notes: This figure presents binned scatter plots of proportional changes in origin-by-destination migration rates, relative to 1996, along the distribution of origin (panel a) and destination (panel b) cancer mortality rates in 1996 (exposure). Each bin corresponds to a decile of the exposure distribution. We follow the method of Cattaneo et al. (2024) to absorb origin and destination state fixed effects. The change in proportional migration rates across bins can be interpreted as semi-parametric estimates of $\phi_{o,t}$ (panel a) and $\phi_{l,t}$ (panel b) from Equation 4. This figure uses IRS migration data which covers inter-county internal migration for the adult population (tax-filers) and provides year-to-year moves. This figure is referenced in Section VI.b.

Figure 5: Effects of the Opioid Epidemic on Out-Migration

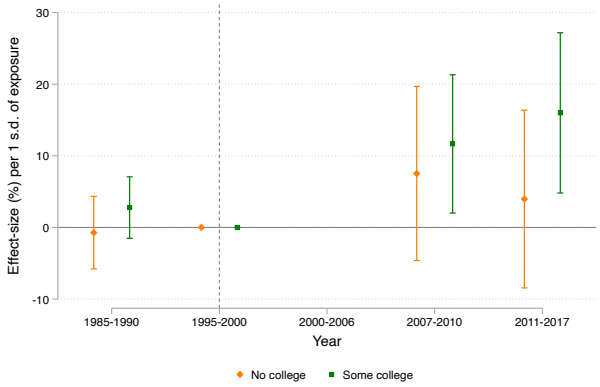
(a) Adult population (IRS)



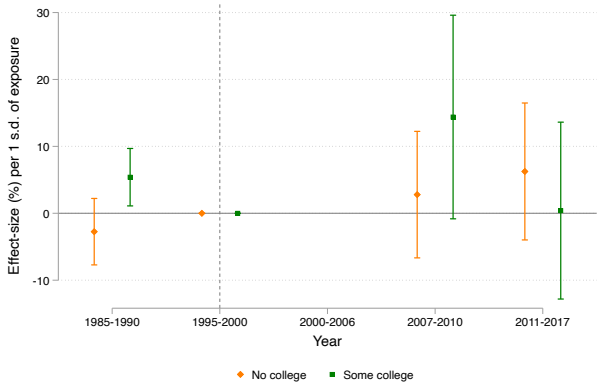
(b) Adult population, by sex (Census and ACS)



(c) Female adult population, by education

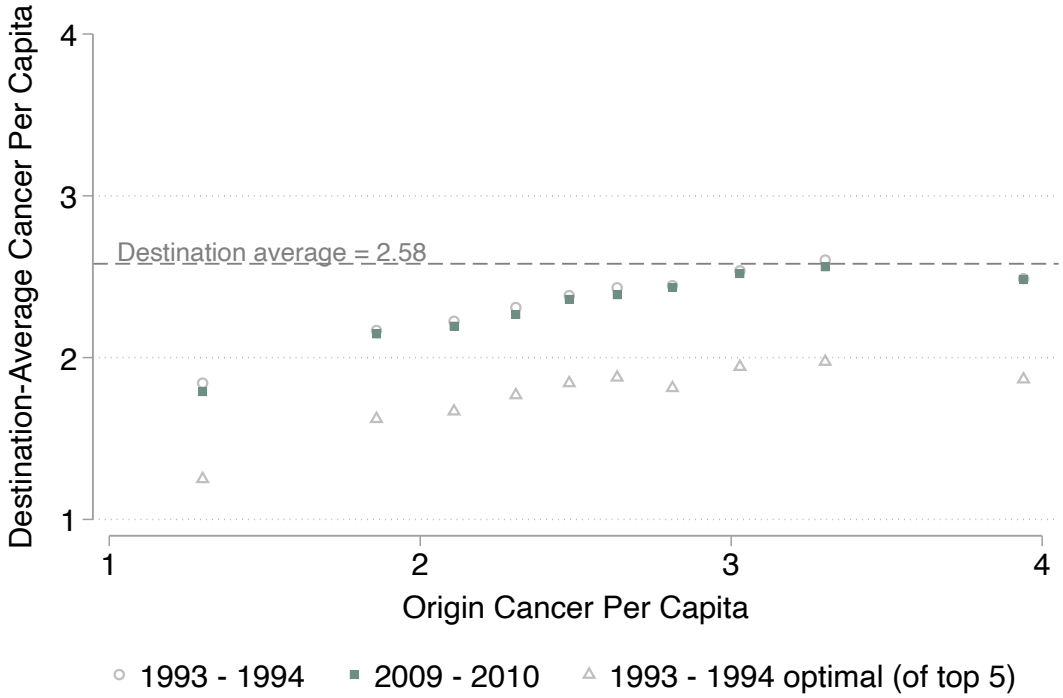


(d) Male adult population, by education



Notes: This figure presents the effects of exposure to the opioid epidemic on out-migration rates. Data from the IRS (panel a) covers inter-county internal migration for the adult population (tax-filers) and provides year-to-year moves. Data from the decennial census and ACS (panels b to d) are used to construct inter-CZ migration of adults by sex and education. Decennial census data (1990, 2000) provides 5-year migration patterns and ACS data (2005, onward) provides 1-year migration patterns. For comparability, we pool ACS data into multi-year windows (e.g., 2005–2010) over which the underlying Public Use Microdata Area (PUMA) geographies, used for mapping to CZs, remain constant. The effect size (expressed as a percentage) corresponds to the scaled coefficients ϕ_o from Equation (4). Specifically, we multiply the estimated coefficient by the standard deviation of 1996 cancer mortality rates. Standard errors are clustered two-way at the origin-destination level. This figure is referenced in Section VI.b

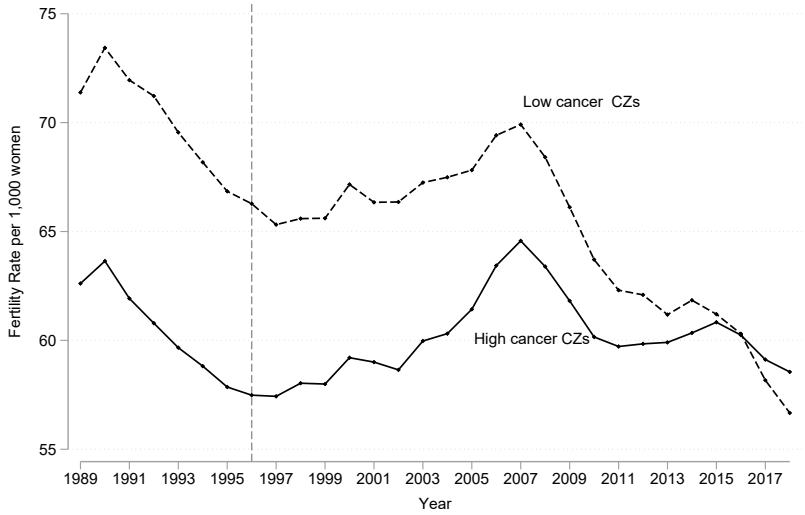
Figure 6: Migration Directedness (IRS)



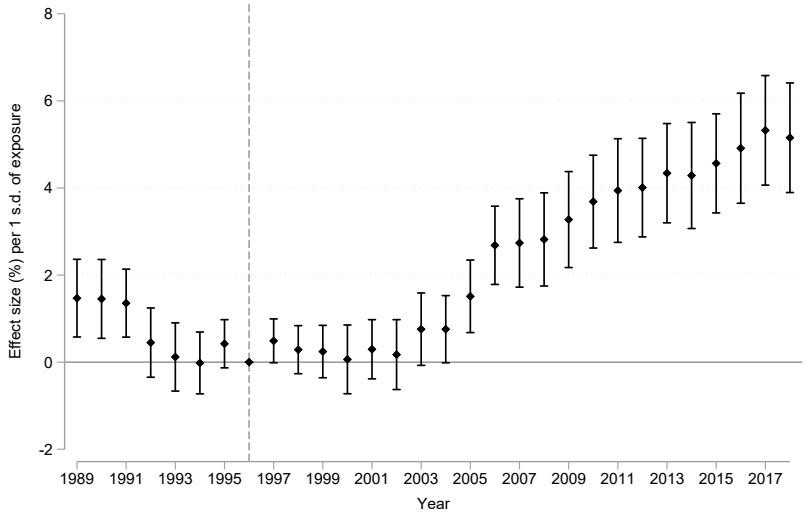
Notes: This figure shows a binned scatter plot of average exposure to the opioid epidemic (cancer mortality per capita in 1996) in destination counties by exposure in origin counties. Each bin corresponds to a decile origin cancer exposure. For each decile, we compute the migration-weighted average exposure in destinations. For "1993–1994" (hollow, grey circles), we construct weights based on actual origin-destination migration patterns from 1993–1994. For, "1993 - 1994 optimal (of top 5)" (hollow, grey triangles) we construct weights based on if individuals in a given origin moved to the lowest-exposure destination county of the top five most popular destinations from that origin in 1994. For "2009–2010" (solid, teal squares) we construct weights based on actual origin-destination migration patterns from 2009–2010. A slope of one corresponds to "perfect non-targeting": migrants relocate to identically exposed locations, on average. A slope of negative one corresponds to "perfect targeting": migrants in the most exposed areas relocate to the least exposed areas, on average. We find a slope of 0.26 (s.e. 0.01) indicating that migration was largely non-targeted; despite this, by the 30th percentile of exposure, individuals ended up in less exposed destinations, on average. This figure uses IRS migration data which covers inter-county internal migration for the adult population (tax-filers) and provides year-to-year moves. This figure is referenced in Section VI.b.

Figure 7: Effects of the Opioid Epidemic on Total Fertility

(a) Trends in High- vs Low-Cancer-Mortality CZs

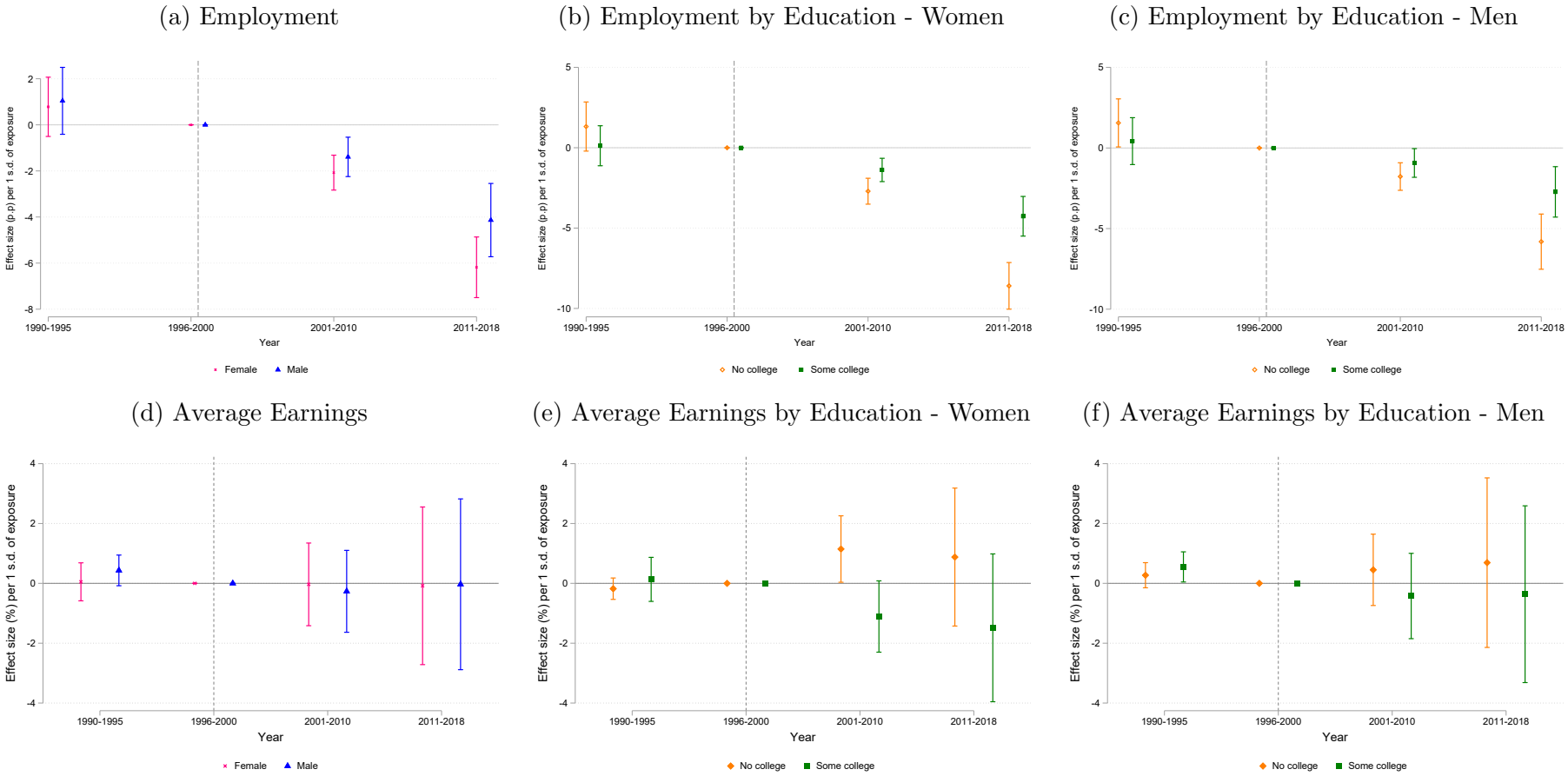


(b) Event Study



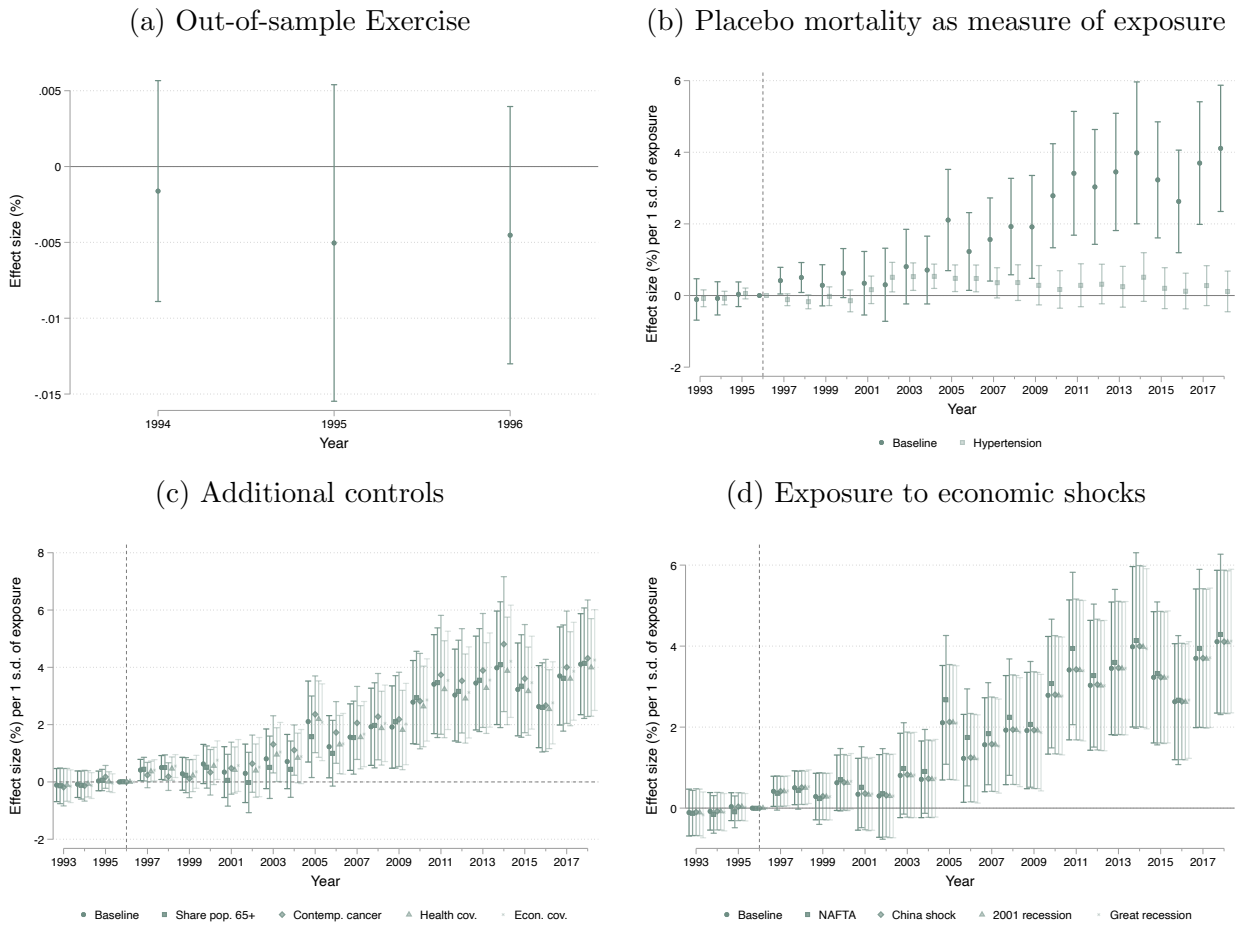
Notes: Total fertility is defined as the ratio of births to women aged 15 to 44 to the female population in this age group. Panel (a) shows the evolution of the fertility rate per 1,000 women in commuting zones (CZs) in the bottom (dashed lines) and top (solid lines) quartiles of cancer mortality prior to the launch of OxyContin. This comparison is based on a binary high-versus-low classification of CZs, with the outcome weighted by population. Panel (b) presents estimates of the effect of exposure to the opioid epidemic on fertility rates. The effect size (expressed as a percentage) corresponds to the scaled coefficients from Equation (1). Specifically, we multiply the estimated coefficient by the standard deviation of 1996 cancer mortality rates and divide by the mean of the outcome variable in 1996. These estimates exploit continuous variation in cancer mortality in 1996 and control for baseline characteristics and state-by-year fixed effects. This figure is referenced in Section VI.c.

Figure 8: Exposure to the Opioid Epidemic and Employment Rates and Earnings by Sex and Education



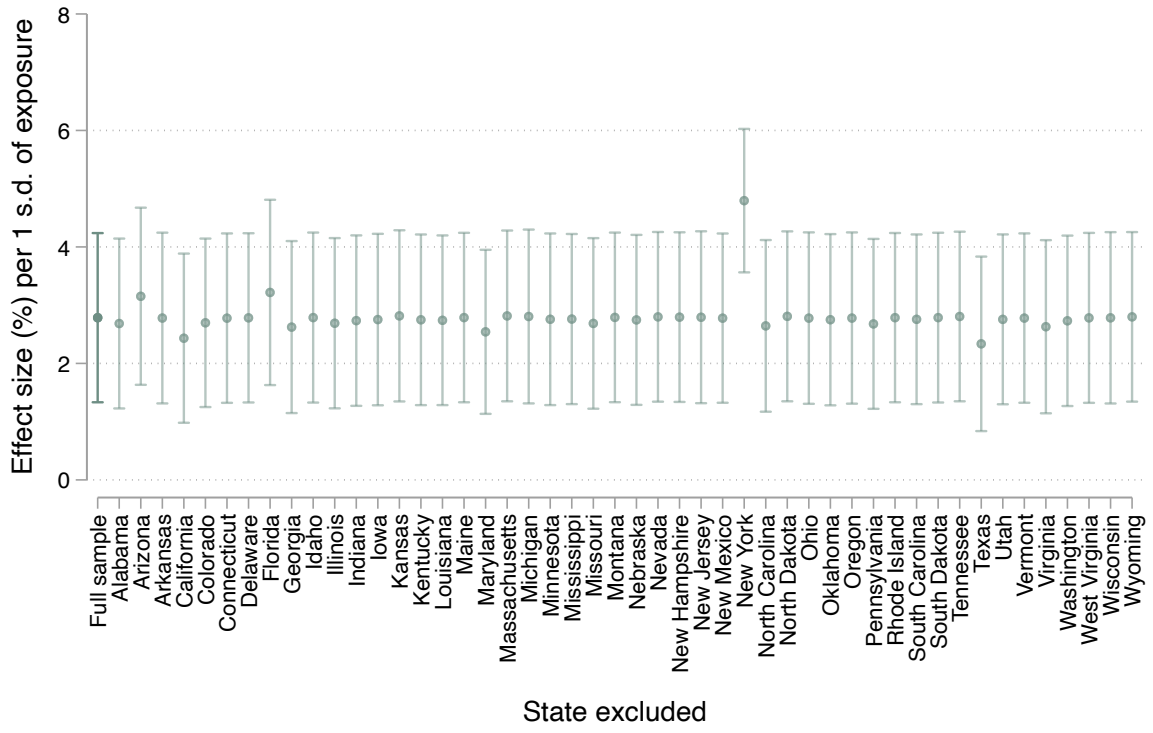
Notes: This figure presents the effects of exposure to the opioid epidemic on employment and average earnings, by sex and by sex–education group. In the top panels, the outcomes are log employment–that is, the log of the number of permanent jobs. In the bottom panels, the outcomes are the ratio of total earnings across all permanent jobs to the total number of permanent jobs. We estimate Equation (6) and present scaled ϕ_g coefficients or effect sizes (expressed as a percentage). Specifically, we multiply the estimated coefficients by the standard deviation of 1996 cancer mortality rates and divide by the mean of the outcome variable in 1996. These estimates exploit continuous variation in cancer mortality in 1996 and control for baseline characteristics and state-by-year fixed effects. Standard errors are clustered at the CZ level. This figure is referenced in Section VII.

Figure 9: Robustness Checks: Out-Migration (IRS)



Notes: This figure presents robustness checks that probe the validity of our empirical strategy. The outcome of interest in each regression is proportional changes in origin-by-destination migration rates. Panel (a) shows an out-of-sample exercise where we use 1976 cancer mortality rates as our measure of exposure to the epidemic and estimate effects on migration rates relative to 1993. Panel (b) presents a placebo exercise where we use 1996 mortality from hypertension as the measure of exposure. Panel (c) expands the set of controls to include: the share of the population over 65; contemporaneous cancer mortality; and broad sets of health covariates and economic covariates. Panel (d) adds measures of exposure to various economic shocks. For the regressions in panels (c) and (d), we allow covariates to vary flexibly over time. The effect sizes correspond to scaled coefficients ($\phi_{o,t}$) from Equation 4. This figure uses IRS migration data which covers inter-county internal migration for the adult population (tax-filers) and provides year-to-year moves. This figure is referenced in Section VIII.

Figure 10: Estimation on Alternative Samples: Out-Migration (IRS)



Notes: This figure presents estimates of the effects of exposure to the opioid epidemic on out-migration rates among the adult population (tax filers) on alternative geographic samples. The effects shown correspond to scaled coefficients ($\phi_{o,t}$) from Equation 4. This figure shows estimates for 2010, leaving one state out at a time. This figure uses IRS migration data which covers inter-county internal migration for the adult population (tax-filers) and provides year-to-year moves. This figure is referenced in Section VIII..

XI. Tables

Table 1: Summary Statistics

	Before 1996			After 2010		
	Mean	Median	SD	Mean	Median	SD
	(1)	(2)	(3)	(4)	(5)	(6)
Cancer mortality rate per 1,000	2.5218	2.5227	0.5477	2.4858	2.4998	0.5517
Local population growth						
Overall population	0.0982	0.0920	0.0945	0.0270	0.0136	0.1005
Ages 18–44	0.0331	0.0280	0.1056	0.0173	0.0079	0.1108
Female	0.0204	0.0139	0.1026	0.0052	-0.0038	0.1104
Male	0.0449	0.0380	0.1156	0.0284	0.0166	0.1133
Mortality (per 10,000)						
Drug-induced all	0.0506	0.0416	0.0501	0.2695	0.2230	0.2057
Drug-induced female	0.0331	0.0193	0.0452	0.1980	0.1668	0.1695
Drug-induced male	0.0681	0.0503	0.0790	0.3386	0.2720	0.2829
Out-migration probability	0.0675	0.0621	0.0245	0.0638	0.0603	0.0212
Fertility Rate (per 1,000 women)						
Women aged 15–44	63.6284	62.3441	10.1162	63.4227	62.5304	9.4574
Women aged 25–29	128.1476	129.8867	35.7958	100.4293	100.2160	34.0146
Economic outcomes						
Employment female (ln)	10.1690	9.8816	1.3862	10.2178	9.9826	1.3587
Employment male (ln)	10.1251	9.8304	1.3461	10.2366	10.0174	1.3437

Notes: This table presents summary statistics for the main dependent variables and our measure of exposure to the opioid epidemic for the periods before and after the launch of OxyContin. This table is referenced in Section III.

Table 2: Effects of Exposure to the Opioid Epidemic on Population Growth Rates

1996 cancer mortality ×	Change in log population per 1 s.d. exposure			
	All ages (1)	Ages 0-17 (2)	Ages 18-44 (3)	Ages 45-64 (4)
A. Overall Population				
1990 – 2000	-0.0015	-0.0025	-0.0134*	-0.0012
	(0.0056)	(0.0077)	(0.0072)	(0.0052)
2000 – 2010	-0.0123***	-0.0183***	-0.0247***	-0.022***
	(0.0034)	(0.0049)	(0.0048)	(0.0034)
2000 – 2020	-0.0253***	-0.0307***	-0.0416***	-0.0524***
	(0.009)	(0.0104)	(0.0103)	(0.0095)
B. Female Population				
1990 – 2000	0.0001	-0.001	-0.0107	0.0023
	(0.0056)	(0.0077)	(0.0069)	(0.0055)
2000 – 2010	-0.014***	-0.0191***	-0.0253***	-0.0233***
	(0.0034)	(0.0049)	(0.0047)	(0.0036)
2000 – 2020	-0.0257***	-0.0317***	-0.0373***	-0.0517***
	(0.009)	(0.0103)	(0.0104)	(0.0096)
C. Male Population				
1990 – 2000	-0.0029	-0.0041	-0.0151*	-0.0049
	(0.0059)	(0.0077)	(0.0084)	(0.0052)
2000 – 2010	-0.011***	-0.0179***	-0.0254***	-0.021***
	(0.0036)	(0.005)	(0.0053)	(0.0035)
2000 – 2020	-0.0251***	-0.0299***	-0.046***	-0.0532***
	(0.009)	(0.0105)	(0.0106)	(0.0096)

Notes: This table presents estimates of the effects of exposure to the opioid epidemic on the change in log population. We consider changes in the total population by sex and age groups. Each cell reports estimates of the coefficient ϕ from Equation (2). All specifications include a set of demographic controls measured in 1990 and state-level fixed effects. Standard errors are clustered at the CZ level. *p<0.10, **p<0.05, *** p<0.01. This table is referenced in Section V..

Table 3: Effects of Exposure to the Opioid Epidemic on Population Growth Rates by Education

	Change in log population per 1 s.d. exposure		
1996 cancer mortality ×	Ages 18+	Ages 18-44	Ages 45-64
	(1)	(2)	(3)
A. Non College Education			
Overall Population			
2000 – 2010	-0.0091**	-0.0214***	-0.0165***
	(0.004)	(0.0063)	(0.0052)
2000 – 2020	-0.0312***	-0.0421***	-0.045***
	(0.0082)	(0.0109)	(0.0095)
Female Population			
2000 – 2010	-0.0098**	-0.0242***	-0.0191***
	(0.0043)	(0.0072)	(0.0054)
2000 – 2020	-0.0341***	-0.0442***	-0.0526***
	(0.0094)	(0.0131)	(0.0102)
Male Population			
2000 – 2010	-0.0103**	-0.0217***	-0.0156**
	(0.0044)	(0.0065)	(0.0062)
2000 – 2020	-0.0315***	-0.0443***	-0.0408***
	(0.0082)	(0.0106)	(0.0107)
B. College Education			
Overall Population			
2000 – 2010	-0.0198***	-0.0246***	-0.033***
	(0.0049)	(0.0065)	(0.0061)
2010 – 2020	-0.0341***	-0.0379***	-0.06***
	(0.009)	(0.0094)	(0.0136)
Female Population			
2000 – 2010	-0.0241***	-0.0321***	-0.0343***
	(0.005)	(0.0063)	(0.0063)
2000 – 2020	-0.0363***	-0.0423***	-0.059***
	(0.009)	(0.0091)	(0.0138)
Male Population			
2000 – 2010	-0.0136**	-0.0183**	-0.0307***
	(0.0056)	(0.0077)	(0.0071)
2000 – 2020	-0.0298***	-0.0358***	-0.0592***
	(0.0099)	(0.0112)	(0.0146)

Notes: This table presents estimates of the effects of exposure to the opioid epidemic on the change in log population. We consider changes in the total population by education, sex and age groups. Each cell reports estimates of the coefficient ϕ from Equation (2). All specifications include a set of demographic controls measured in 1990 and state-level fixed effects. Standard errors are clustered at the CZ level. *p<0.10, **p<0.05, *** p<0.01. This table is referenced in Section V..

Table 4: Counterfactual Population Growth Rates

1996 cancer mortality ×	Change in log population per 1 s.d. exposure			
	All ages		Ages 18-44	
	Observed	Counterfactual	Observed	Counterfactual
	(1)	(2)	(3)	(4)
A. Overall Population				
2000 – 2010	-0.0123***	-0.0104***	-0.0247***	-0.0242***
	(0.0034)	(0.0036)	(0.0048)	(0.0049)
2000 – 2020	-0.0253***	-0.0116**	-0.0416***	-0.0277***
	(0.009)	(0.0052)	(0.0103)	(0.0074)
B. Female Population				
2000 – 2010	-0.014***	-0.0116***	-0.0253***	-0.0241***
	(0.0034)	(0.0035)	(0.0047)	(0.0047)
2000 – 2020	-0.0257***	-0.012**	-0.0373***	-0.0235***
	(0.009)	(0.0051)	(0.0104)	(0.007)
C. Male Population				
2000 – 2010	-0.011***	-0.0097**	-0.0254***	-0.025***
	(0.0036)	(0.0038)	(0.0053)	(0.0054)
2000 – 2020	-0.0251***	-0.0118**	-0.046***	-0.0333***
	(0.009)	(0.0054)	(0.0106)	(0.0082)

Notes: This table presents estimates of the effects of exposure to the opioid epidemic on the change in log population and on the change in log counterfactual population. It also provides results of these exercises by sex and age groups. Equation (3) details the computation of counterfactual population counts. Each cell reports estimates of the coefficient ϕ from Equation (2). All specifications include a set of demographic controls measured in 1990 and state-level fixed effects. Standard errors are clustered at the CZ level. *p<0.10, **p<0.05, *** p<0.01. This table is referenced in Section VI.a.

Table 5: Counterfactual Population Growth Rate by Education

	Change in log population per 1 s.d. exposure	
1996 cancer mortality ×	Observed	Counterfactual
	(1)	(2)
A. Non College Education		
Overall Population		
2000 – 2010	-0.0214***	-0.0214***
	(0.0063)	(0.0063)
2000 – 2020	-0.0421***	-0.034***
	(0.0109)	(0.0093)
Female Population		
2000 – 2010	-0.0242***	-0.0237***
	(0.0072)	(0.0072)
2000 – 2020	-0.0442***	-0.0343***
	(0.0131)	(0.0108)
Male Population		
2000 – 2010	-0.0217***	-0.0217***
	(0.0065)	(0.0064)
2000 – 2020	-0.0443***	-0.037***
	(0.0106)	(0.0095)
B. College Education		
Overall Population		
2000 – 2010	-0.0246***	-0.0252***
	(0.0065)	(0.0064)
2000 – 2020	-0.0379***	-0.0325***
	(0.0094)	(0.0081)
Female Population		
2000 – 2010	-0.0321***	-0.0327***
	(0.0063)	(0.0062)
2000 – 2020	-0.0423***	-0.0363***
	(0.0091)	(0.0077)
Male Population		
2000 – 2010	-0.0183**	-0.0189**
	(0.0077)	(0.0076)
2000 – 2020	-0.0358***	-0.0309***
	(0.0112)	(0.0101)

Notes: This table presents estimates of the effects of exposure to the opioid epidemic on the change in log population on the change in log counterfactual population. We consider changes in the total population by education, sex and age groups. Equation (3) details the computation of counterfactual population counts. Each cell reports estimates of the coefficient ϕ from Equation 2. All specifications include a set of demographic controls measured in 1990 and state-level fixed effects. Standard errors are clustered at the CZ level. *p<0.10, **p<0.05, *** p<0.01. This table is referenced in Section VI.a.

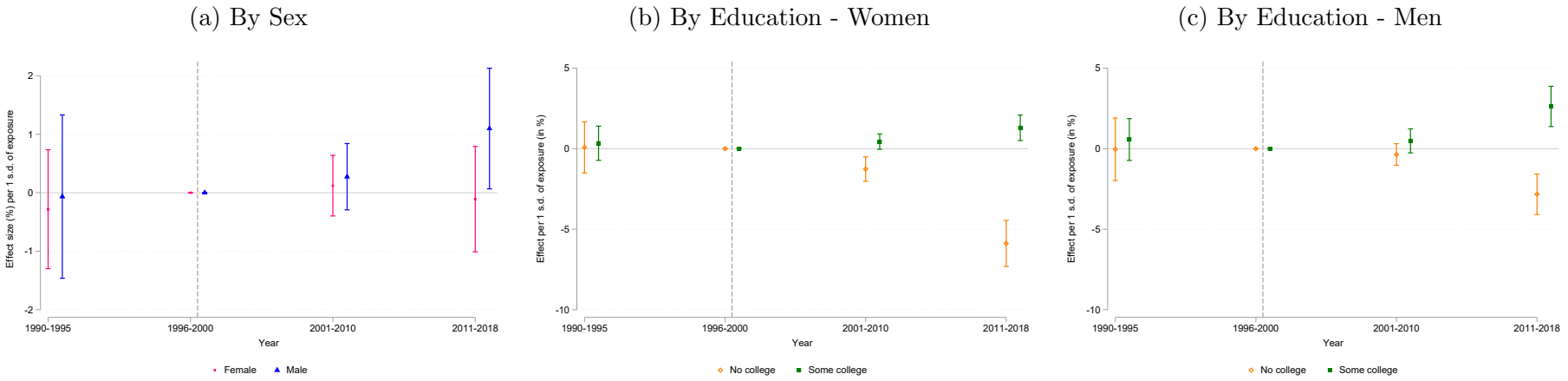
Table 6: Placebo Tests: Placebo Mortality and Population Growth Rate

	Cancer 1996 (1)	Hypertension 1996 (2)	Pneumonia 1996 (3)
Panel A. Overall Population			
1990 – 2000	-0.0015	-0.0081**	-0.0049*
	(0.0056)	(0.004)	(0.003)
2000 – 2010	-0.0123***	-0.0003	0.0015
	(0.0034)	(0.0025)	(0.0022)
2000 – 2020	-0.0253***	-0.0127*	-0.0082
	(0.009)	(0.0071)	(0.0057)
Panel B. Female Population			
1990 – 2000	0.0001	-0.0074*	-0.0056**
	(0.0056)	(0.004)	(0.0028)
2000 – 2010	-0.014***	-0.0006	0.0011
	(0.0034)	(0.0026)	(0.002)
2000 – 2020	-0.0258***	-0.0129*	-0.0091
	(0.009)	(0.0072)	(0.0058)
Panel C: Male Population			
1990 – 2000	-0.0029	-0.0087**	-0.0041
	(0.0059)	(0.004)	(0.0033)
2000 – 2010	-0.011***	-0.0006	0.0017
	(0.0036)	(0.0025)	(0.0024)
2000 – 2020	-0.0251***	-0.0128*	-0.0075
	(0.009)	(0.0069)	(0.0057)

Notes: This table presents estimates of the effects of placebo mortality rates on the change in CZ log population. We consider changes in the total population by sex and age groups. Each cell reports estimates of the coefficient ϕ from Equation (2). All specifications include a set of demographic controls measured in 1990 and state-level fixed effects. Standard errors are clustered at the CZ level. *p<0.10, **p<0.05, *** p<0.01. This table is referenced in Section VIII..

A. Additional Figures and Tables

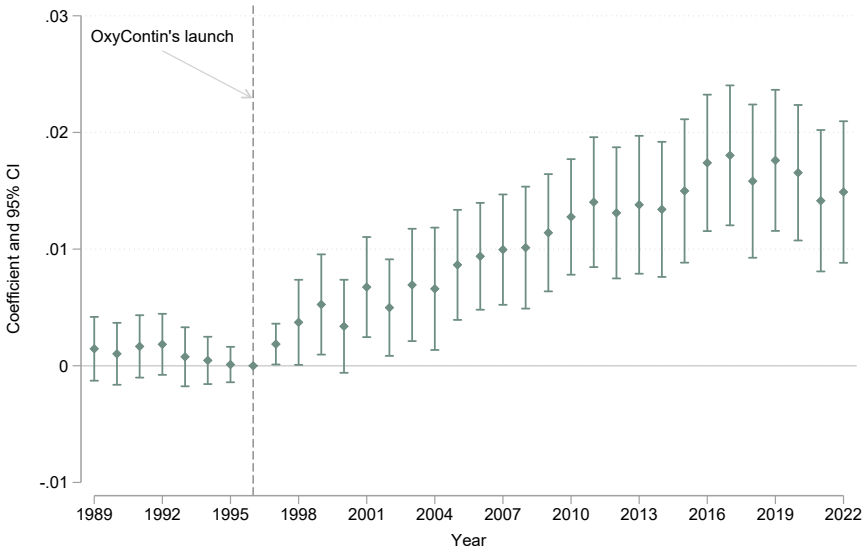
Figure A1: Exposure to the Opioid Epidemic on Employment-to-Population Ratio



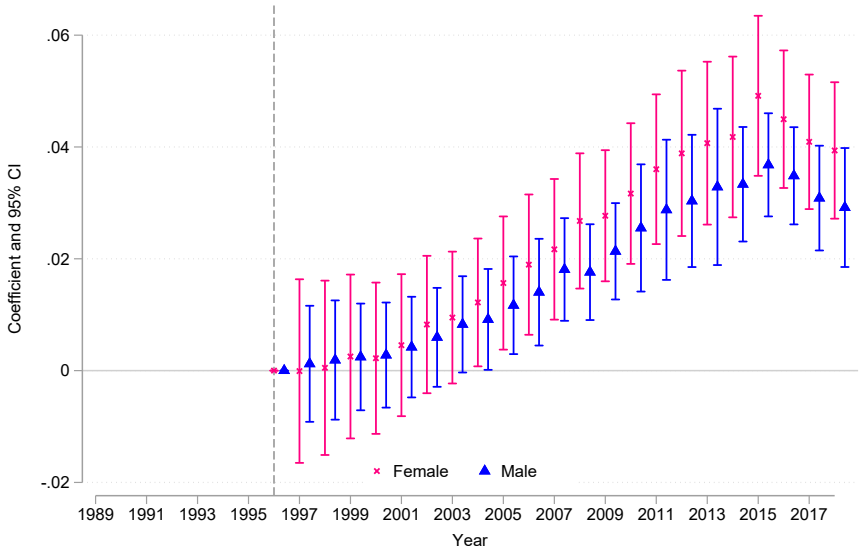
Notes: This figure presents the effects of exposure to the opioid epidemic on employment by sex and by sex–education group. The outcomes are employment-to-population ratios. Where employment is the number of permanent jobs. We estimate Equation (6) and present scaled ϕ_g coefficients or effect sizes (expressed as a percentage). Specifically, we multiply the estimated coefficients by the standard deviation of 1996 cancer mortality rates and divide by the mean of the outcome variable in 1996. These estimates exploit continuous variation in cancer mortality in 1996 and control for baseline characteristics and state-by-year fixed effects. Standard errors are clustered at the CZ level. This figure is referenced in Section VII.

Figure A2: Effects of Exposure to the Opioid Epidemic on SNAP Receipt Rate

(a) All households

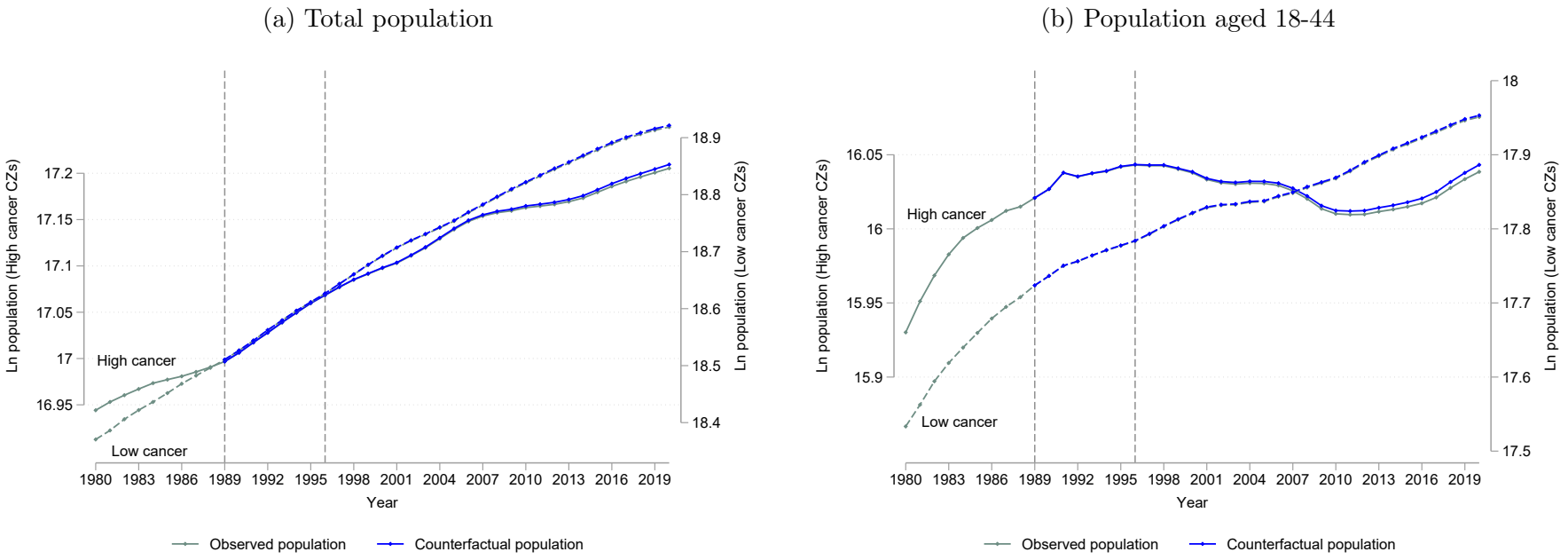


(b) By sex of the household head



Notes: Panel (a) of this figure shows the effects of exposure to the opioid epidemic on SNAP receipt rates, which corresponds to the ratio between number of beneficiaries of the SNAP program and the population of the CZ. This event-study exploit continuous variation in 1996 cancer mortality and include time-varying controls and CZ and state–year fixed effects. Standard errors are clustered at the CZ level. Panel (b) of this figure shows the effects on SNAP receipt rates by sex of the head of the household participating in the program. Due to data limitations, we estimate these effects at the state level using the following specification: $y_{st} = \sum_{\tau=1997}^{2018} \phi_\tau \; CancerMR_{c,1996} \mathbf{1}(Year = \tau) + X_{st} + \gamma_s + \gamma_t + \upsilon_{st}$. Where the vector X_{st} includes the contemporaneous cancer mortality, the white and female population shares, and the shares of the population aged 18–29, 30–49, 50–64, above 65 years, and under 1 year. This figure is referenced in Section VII.

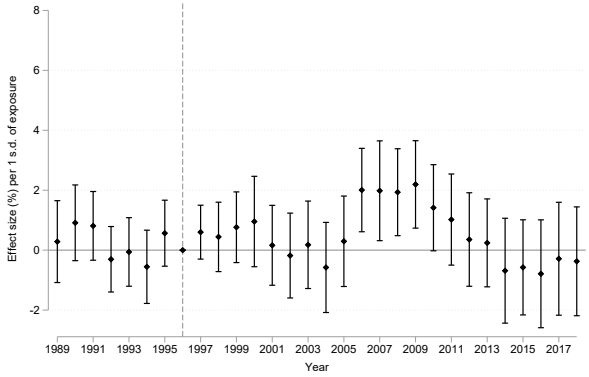
Figure A3: Population Trends: Observed vs Counterfactual



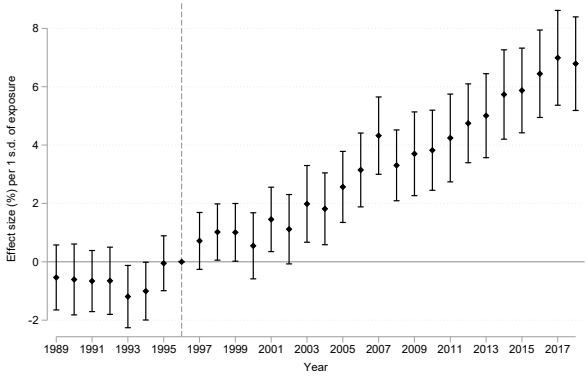
Notes: This figure shows the evolution of observed and counterfactual log total population and log population ages 18 to 44 across CZs in the bottom (dashed lines) and top (solid lines) quartiles of cancer mortality before the launch of OxyContin. This comparison is based on dichotomous high-versus-low categorization of CZs, and the outcome is weighted by population. Equation (3) details the computation of counterfactual population counts. Counterfactual counts are constructed beginning in 1989, as indicated by the first vertical line. The second vertical line marks the 1996 launch of OxyContin. The series for high-cancer-mortality CZs are plotted on the left vertical axis, while those for low-cancer-mortality CZs are shown on the right vertical axis. This figure is referenced in Section VI.a .

Figure A4: Effects of the Opioid Epidemic on Fertility by Age Groups

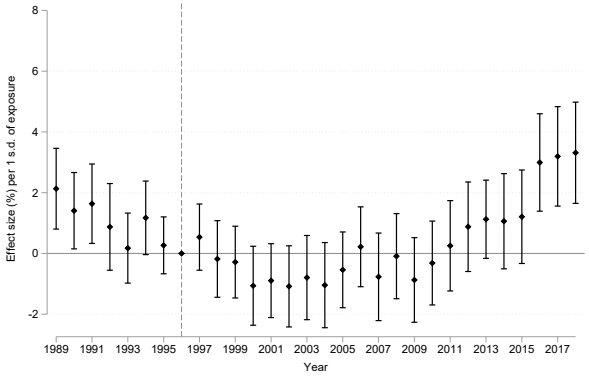
(a) Ages 20-24



(b) Ages 25-29



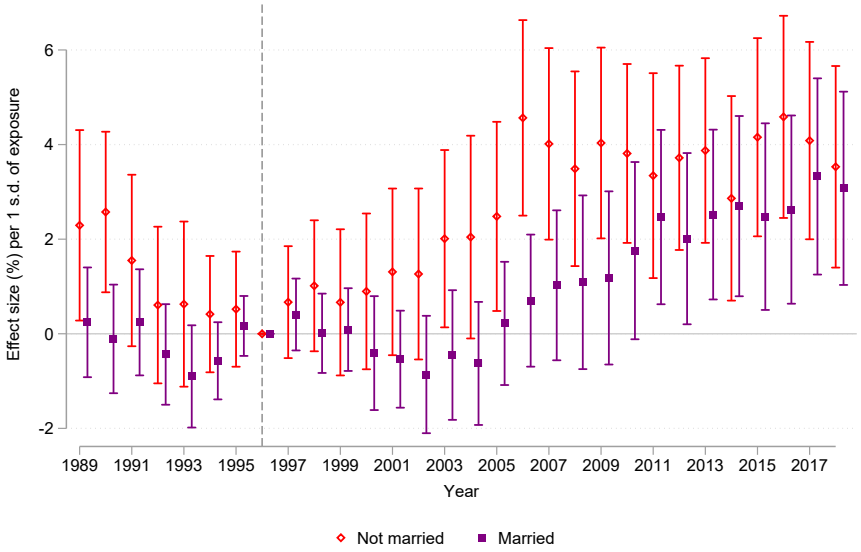
(c) Ages 30-34



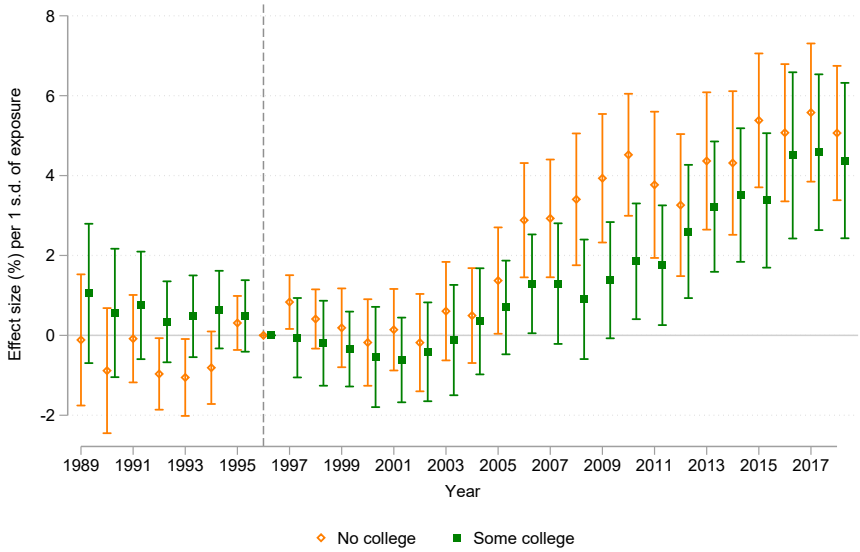
Notes: This figure presents the effects of exposure to the opioid epidemic on fertility rates by age group. The three groups together accounted for 76% of births to women aged 15–44 in 1996: 25% were to mothers aged 20–24, 28% to those aged 25–29, and 23% to those aged 30–34. Figure **??** shows the evolution of these shares. The effect size (expressed as a percentage) corresponds to the scaled coefficients from Equation (1). Specifically, we multiply the estimated coefficient by the standard deviation of 1996 cancer mortality rates and divide by the mean of the outcome variable in 1996. These estimates exploit continuous variation in cancer mortality in 1996 and control for baseline characteristics and state-by-year fixed effects. Standard errors are clustered at the CZ level. This figure is referenced in Section VI.c

Figure A5: Effects of the Opioid Epidemic on Fertility by Marital Status and Education

(a) Marital Status



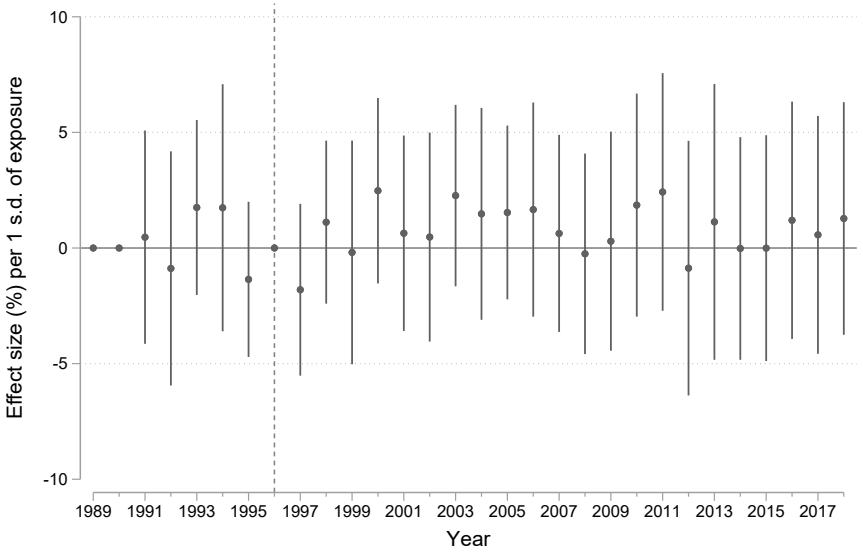
(b) Education



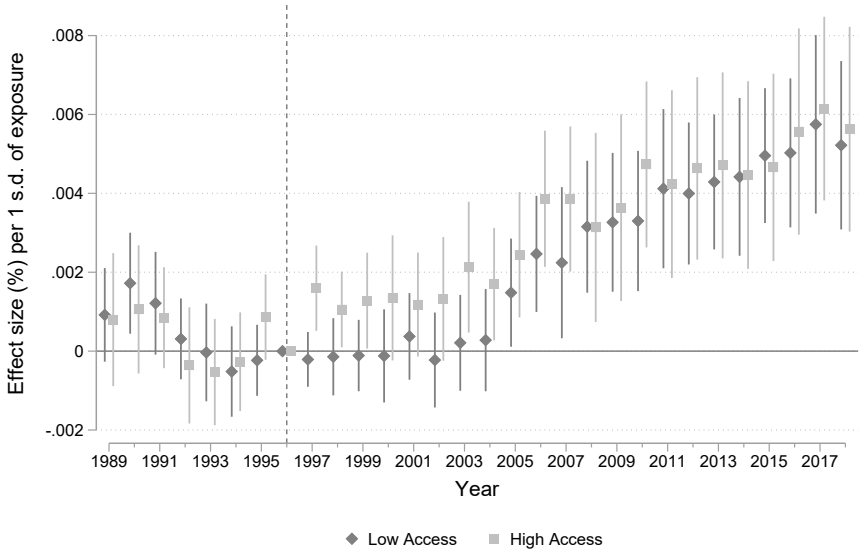
Notes: This figure shows the effects of exposure to the opioid epidemic on fertility rates by marital status (panel a) and educational level of the mother (panel b). The effect size (expressed as a percentage) corresponds to the scaled coefficients from Equation (1). Specifically, we multiply the estimated coefficient by the standard deviation of 1996 cancer mortality rates and divide by the mean of the outcome variable in 1996. These estimates exploit continuous variation in cancer mortality in 1996 and control for baseline characteristics and state-by-year fixed effects. The married (unmarried) fertility rate is defined as the number of births to married (unmarried) individuals aged 15 to 44 years old divided by the population of married (unmarried) women in that age group. Similarly, fertility rates by education are the ratio of births to individuals with a no college (some college) to the number of women with no college (some college). Standard errors are clustered at the CZ level. This figure is referenced in Section VI.c.

Figure A6: Effects of the Opioid Epidemic on Abortion Rates and Fertility Heterogeneity

(a) Abortion Rated



(b) Heterogeneity by Abortion Access



Notes: This figure shows the effects of exposure to the opioid epidemic on abortion rates (panel a) and fertility rates by access to abortion (panel b). Access to abortion is defined by above or below median distance to an abortion clinic. Standard errors are clustered at the CZ level. This figure is referenced in Section VI.c.

Table A1: Effects of Exposure to the Opioid Epidemic on Drug Induced Mortality by Sex and Education

1996 cancer mortality x	Female	Male	No college	Some college
	(1)	(2)	(3)	(4)
1989 - 1995	1.8843	2.2421	0.6782	-2.7407
	(15.7529)	(7.668)	(9.11)	(16.6325)
1997 - 2003	30.066*	12.768	19.5308**	7.9099
	(16.1488)	(10.1008)	(9.7978)	(19.119)
2004 - 2010	55.2389***	44.9236***	57.6871***	18.6983
	(19.4514)	(12.5563)	(14.6056)	(20.8508)
2011 - 2018	55.2467***	31.3286**	45.7283**	21.13
	(19.2814)	(14.5314)	(18.5686)	(21.0003)
Mean of dep variable in 1996	0.0413	0.0821	0.0767	0.0263
Number of CZs	587	587	586	586
Observations	17,608	17,608	17,578	17,578

Notes: This table shows the effects of the opioid epidemic on drug-induced mortality by sex and by education for the period 1989 to 2018. Effects are estimated following Equation (6) where years are grouped as listed in the left column of the table. We report effect sizes constructed by multiplying the estimated coefficient by the standard deviation of 1996 cancer mortality rates and dividing by the mean of the outcome variable in 1996. These estimates exploit continuous variation in cancer mortality in 1996 and control for baseline characteristics and state-by-year fixed effects. Standard errors are clustered at the CZ level. This figure is referenced in Section VI.a.

Table A2: Effects of Exposure to the Opioid Epidemic on Fertility by Marital Status and Education

1996 cancer mortality ×	Unmarried	Married	No college	Some college
	(1)	(2)	(3)	(4)
1989 - 1995	1.2266*	-0.1957	-0.5143	0.6154
	(0.6745)	(0.412)	(0.3722)	(0.4827)
1997 - 2003	1.1179	-0.2501	0.2599	-0.3265
	(0.7219)	(0.476)	(0.4317)	(0.4784)
2004 - 2010	3.4912***	0.7633	2.7909***	1.1104*
	(0.9081)	(0.7316)	(0.6378)	(0.6137)
2011 - 2018	3.7671***	2.646***	4.6007***	3.4917***
	(0.9647)	(0.9248)	(0.7506)	(0.7613)
Mean of dep variable in 1996	0.0481	0.0752	0.083	0.0531
Number of CZs	583	583	586	586
Observations	17,456	17,456	17,580	17,580

Notes: This table shows the effects of the opioid epidemic on fertility rates by marital status and by education for the period 1989 to 2018. Effects are estimated following Equation (6) where years are grouped as listed in the left column of the table. We report effect sizes constructed by multiplying the estimated coefficient by the standard deviation of 1996 cancer mortality rates and dividing by the mean of the outcome variable in 1996. These estimates exploit continuous variation in cancer mortality in 1996 and control for baseline characteristics and state-by-year fixed effects. Standard errors are clustered at the CZ level. This figure is referenced in Section VI.a.

Table A3: Out-of-sample: 1976 Cancer Mortality on Population Growth Rates

	Change in log population per 1 s.d. exposure		
	All	Female	Male
1976 cancer mortality x	(1)	(2)	(3)
1990 – 2000	0.0044	0.0048	0.004
	(0.0035)	(0.0037)	(0.0034)

Notes: This table presents estimates of the effects of 1976 cancer mortality rates on the change in CZ log population. We consider changes in the total population by sex. Each cell reports estimates of the coefficient ϕ from Equation (2) using cancer mortality in 1976 as our measure of exposure. All specifications include a set of demographic controls measured in 1990 and state-level fixed effects. Standard errors are clustered at the CZ level. *p<0.10, **p<0.05, *** p<0.01. This table is referenced in Section VIII..