The Indirect Effects of Opioid Supply on U.S. Suicide and Alcohol-Related Deaths

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Abstract

We provide evidence that drug abuse resulting from the opioid epidemic contributed to rising non-drug suicide (NDS) and alcohol-related mortality (ARM) rates during the last 20 years. Our underlying assumption for the relationship between the opioid epidemic and these non-drug deaths relies on existent public health studies that demonstrate the interaction between drug abuse and suicidal tendencies or alcohol abuse. Our analysis exploits three well-established opioid medication supply shifters shortly before the opioid epidemic broke out that are used in recent economics studies: triplicate laws, cancer mortality, and Social Security disability insurance benefits. Results suggest that the opioid epidemic contributed 25-40% to increases in NDS and ARM through 2020. This sheds light on additional adverse secondary effects of the opioid epidemic.

Keywords: Opioid Epidemic, Suicide, Alcohol-Related Mortality

JEL Classification: I12, I18, I31

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1 Introduction

The U.S. has concerningly experienced rising mortality rates due to non-drug suicide and alcohol-related disease for the past 25 years (Garnett and Curtin, 2024; Ilyas et al., 2023). Figure 1 presents these rising trends. The causes though are not well-understood. Case and Deaton (2015) identified rising trends in suicide more broadly (not just non-drug) and alcohol-liver disease mortality that coincided with rising trends in deaths from drug overdoses. They argued that these are the result of adverse changes in social and economic factors for specific population segments in the US, and named them 'deaths of despair'. This demand-side approach to explaining these mortality trends has come under scrutiny, particularly since increasing trends in drug overdose deaths have been shown to be largely driven by supply-side factors related to the pharmaceutical industry and the availability of opioid medication (Kolodny et al., 2015; Evans et al., 2019; Alpert et al., 2022; Evans and Lieber, 2023; Arteaga and Barone, 2025) or physician and insurance incentives and practices (Barnett et al., 2017; Buchmueller and Carey, 2018; Schnell and Currie, 2018; Khan et al., 2019; Powell et al., 2020; Eichmeyer and Zhang, 2022). For rising suicide and alcohol-related mortality rates, explanations have been limited and have focused more on demand-side factors, including declines in religiosity (Giles et al., 2023), a decline in the social safety net available to disadvantaged families (Lichtman-Sadot and Slusky, 2024), or a depreciation of the minimum wage (Dow et al., 2020).

The effects of supply-side factors on rising suicide rates have been minimally investigated. For rising alcohol-related mortality rates, we are not aware of any study that argues that supply-side factors played a role. Our project fills this gap by asking whether the opioid epidemic has contributed to rising non-drug suicide (NDS) and alcohol-related mortality (ARM) rates over the last 25 years, while specifically evaluating supply-side factors that have been shown to contribute to the opioid epidemic. Our underlying hypothesis is that opioid and drug abuse, which have been shown to be driven by supply shifters in opioid medication, increase suicidal behavior and alcohol abuse, and these result in increased NDS and ARM rates. Our hypothesis on the interaction between drug abuse and suicidal tendencies or alcohol abuse is supported by studies in the psychiatry and public health literature, as described in Section 2.

Our three supply shifters are taken from three studies. First, we exploit state-level variation at the dawn of the opioid epidemic in the mid-1990's in triplicate prescription programs, which posed a major obstacle to sales of opioid drugs, and as such, resulted in reduced opioid drug marketing

efforts among pharmaceutical companies. We mostly follow the empirical strategy in Alpert et al. (2022) which demonstrated how non-triplicate states experienced substantially greater drug mortality than triplicate states during the opioid epidemic. Our second supply shifter is taken from Arteaga and Barone (2025): commuting-zone level 1996 cancer mortality rates. Arteaga and Barone (2025) argue that pharmaceutical companies targeted these markets for opioid medication advertising efforts, under the assumption that the wide use of opioid medication among cancer patients will facilitate the penetration of opioid medication treatment to other patients. Their results indeed show that commuting zones with greater 1996 cancer mortality rates experienced higher drug mortality rates in later years. Lastly, our third supply shifter is based on Evans and Lieber (2023). Evans and Lieber (2023) assume that higher shares of adults treated for pain resulted in greater opioid medication penetration at the local level. They proxy for higher shares of adults in pain with the share of the adult population with social security disability insurance (SSDI). They show that counties with higher shares in 1990 experienced greater drug mortality rates during the opioid epidemic.

Our mortality rates are for the population ages 25-64 at the relevant geographic level (state, commuting zone, or county). Our focus on this age range is in line with previous literature showing that the main population segments affected by the opioid epidemic were prime-age adults (Case and Deaton, 2015; Arteaga and Barone, 2025; Giles et al., 2023; Ruhm, 2024). Our empirical strategy follows the three main studies from which our supply shifters originated, an event study design, with regional variation in the supply shifters at the dawn of the opioid epidemic interacted with annual indicators that show the rising mortality rates after 1996, when OxyContin, a leading opioid prescription drug, was approved by the FDA. We also reinforce our event-study results with an instrumental variable approach that uses our supply shifters as instruments and evaluates opioid use for the first stage.

Our project helps explain the causes of a major public health concern that has taken the lives of hundreds of thousands of Americans over the past 25 years. We find that the supply shifters we evaluate increased NDS and ARM rates by as much as 4 deaths per 100,000 population ages 25-64, which translates into contributing 25-40 percent to the increase in NDS and ARM rates over the last two decades. This represents a shift in the common notion in the existing literature that NDS or ARM are purely demand-driven. Our paper argues otherwise, at least for the period during the opioid epidemic. Our project also contributes to a growing literature that shows the downstream

effects of the opioid epidemic on the labor market (Cohle and Ortega, 2023; Mukherjee et al., 2025), crime (Wen et al., 2017; Bondurant et al., 2018; Dave et al., 2021; Smart and Reuter, 2022), family (Bullinger et al., 2022; Buckles et al., 2023), and voting patterns (Arteaga and Barone, 2025). We focus on mortality, an essential factor for assessing the overall adverse effects of the opioid epidemic.

2 Background

In the last few years, deaths resulting from drug overdoses have exceeded 100,000 annually, and death rates are nearly ten times the rates that presided in the 1990's. Scholarly attempts to explain this surge are based on demand and supply-side hypotheses. Demand-side explanations conjecture that adverse changes in social and economic factors have led certain segments of the population to drug use (Case and Deaton, 2015, 2017; Pierce and Schott, 2020; Giles et al., 2023). Supply-side hypotheses focus on changes in the availability of opioid prescriptions as driving forces for increased drug mortality.

The introduction of the prescription opioid OxyContin in 1996 has been established as a major supply factor that increased drug mortality rates. Alpert et al. (2022) use variation in state-based triplicate prescription programs. These posed a major obstacle to sales of opioid drugs and resulted in reduced marketing efforts for opioid pain medication among pharmaceutical companies. Alpert et al. (2022) show that non-triplicate states had substantially greater increases in drug overdose deaths following the introduction of OxyContin in 1996, as opposed to triplicate states. Arteaga and Barone (2025) similarly argue that greater marketing efforts on behalf of pharmaceutical companies increased drug mortality post-1996. Their proxy for the extent of marketing efforts is the cancer mortality rate as of 1996 at the commuting zone level, based on pharmaceutical companies' internal records documenting that these markets were easier to penetrate due to wider usage of opioid medication among cancer patients. Evans and Lieber (2023) argue that the increased supply of opioid medication did not only result from greater marketing efforts but also from changes in prescribers' behaviors. The authors show that this is strongly correlated with SSDI coverage, as this implies higher concentrations of individuals with pain, thus facilitating the shift toward increased prescription of pain medication. They therefore exploit county-level variation in SSDI coverage as of 1990. Taken as a whole, all three studies exploit regional variation in opioid medication supply shifters at the dawn of or shortly before the opioid epidemic. Using these shifters within a difference-in-differences framework, all three studies demonstrate how supply factors contributed

substantially to rising drug mortality rates.

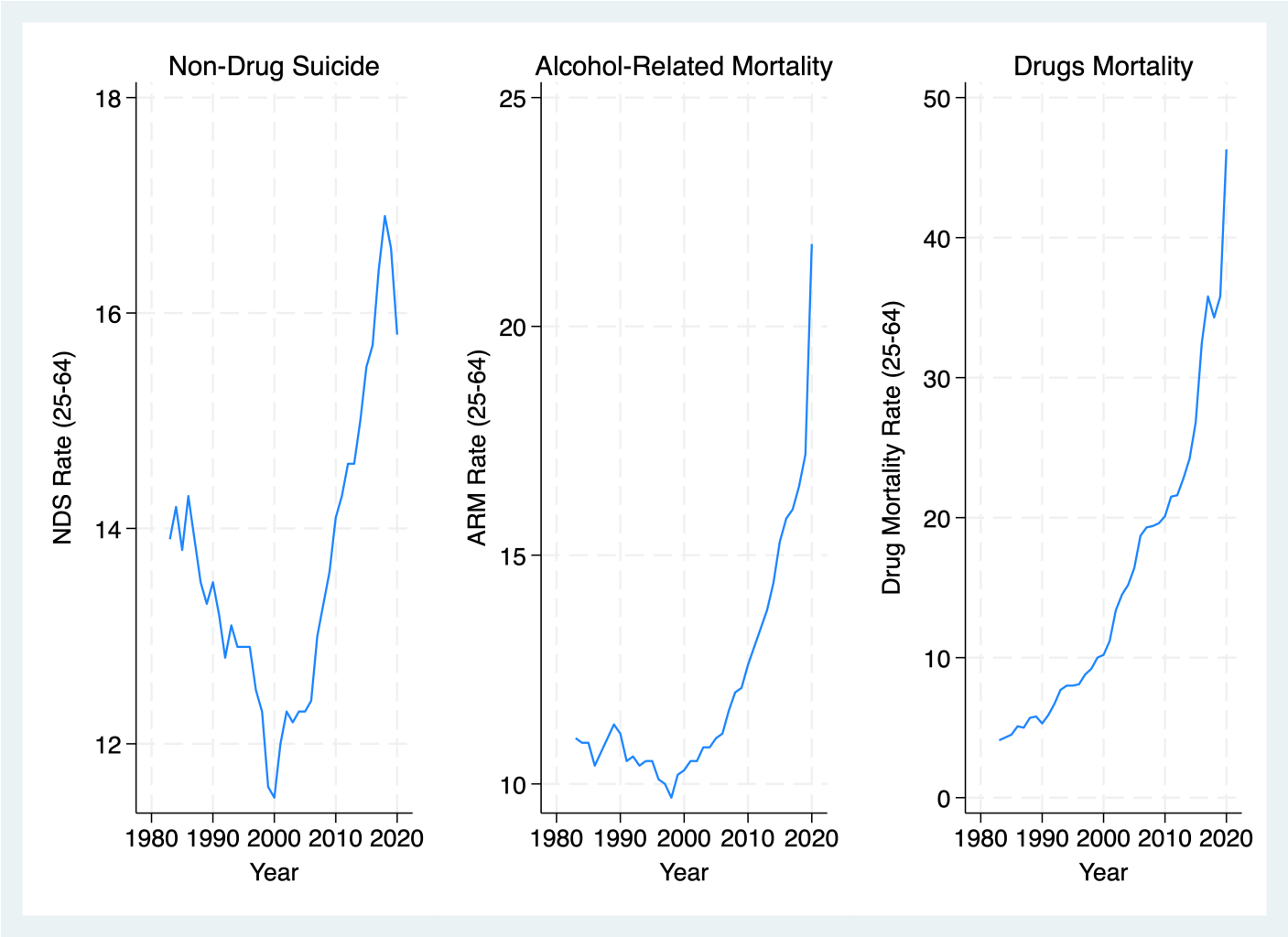
Additional studies have evaluated other supply-side factors that could have affected opioid or drug abuse. These include prescription drug monitoring programs (PDMP) (Buchmueller and Carey, 2018), or physician and insurance incentives and practices (Barnett et al., 2017; Schnell and Currie, 2018; Khan et al., 2019; Powell et al., 2020; Eichmeyer and Zhang, 2022, 2023).

Increases in non-drug suicide (NDS) or alcohol-related mortality (ARM) evolved simultaneously with increased drug mortality (Case and Deaton, 2015). Figure 1 demonstrates this. NDS and ARM rates began to increase sharply in 2000 or 1999, respectively, and by 2020 this represented a nearly 50% increase for NDS or more than a doubling for ARM. While these increases were substantial, drug-mortality increases were much more dramatic.

The NDS and ARM increases have been associated with demand-side factors related to institutional declines of social or economic factors, such as religiosity (Giles et al., 2023), the social safety net provided to disadvantaged families (Lichtman-Sadot and Slusky, 2024), or minimum-wage depreciation (Dow et al., 2020). The notion that NDS and ARM are driven by demand-side factors is so ingrained that studies wishing to rule out a correlation between potential opioid supply shifters and demand factors evaluated NDS or ARM rates as a placebo/robustness check (Alpert et al., 2022; Arteaga and Barone, 2025; Evans and Lieber, 2023). However, a correlation between opioid supply and NDS or ARM rates can exist due to several channels. First, public health and psychiatric studies suggest that drug abuse can intensify suicidal tendencies or alcohol abuse, which could result in a positive correlation between opioid or drug supply factors and NDS or ARM. Second, drug overdose may be a competing risk to NDS or ARM, and if there is an increase in one, this may be at the expense of the other. Our study focuses on the former of these channels, the interaction and potential reinforcement between drug abuse and suicidal behavior or alcohol abuse, which has already been documented in the public health and psychiatric literature outlined below. The latter channel may exist, but given the positive relationship between opioid supply shifters and NDS and ARM rates that we find, this would only imply that our results may be biased downward.

A major risk factor for suicidal behavior is drug misuse. Hesse et al. (2020) find a higher suicide risk among people treated for drug use disorder. There is also evidence of increased suicidal behavior among individuals with substance use disorder (Poorolajal et al., 2016; Yuodelis-Flores and Ries, 2015) or prescription opioid misuse (Ashrafioun et al., 2017). Lastly, Rizk et al. (2021) provide a theoretical framework for the link between chronic opioid use and neurobiological changes

Figure 1: National Mortality Rates for Ages 25-64



Notes: The figure plots the national annual death rates for non-drug suicides (NDS), alcohol-related mortality (ARM), and drug overdose for ages 25-64 during 1983-2020. See footnote 2 for ICD codes used for each cause of death.

that elevate the risk of suicidal behavior.

Witkiewitz and Vowles (2018) provide a review on the evidence of interactions between alcohol and opioid abuse arguing that this is common although understudied. Among a cohort of 5,307 adult patients (18+) with opioid use disorder in an electronic health record database, 23.4% also had a diagnosis of alcohol use disorder (Hser et al., 2017). Furthermore, among adults in treatment for alcohol or drug use disorder approximately 68% had used prescription opioids non-medically in the 30 days prior to intake (Price et al., 2011).

Two economic studies also explore opioid supply-side factors and their effects on suicide. Eichmeyer and Zhang (2023) use administrative veterans' health data and find that assignment to a primary care provider with a greater tendency to prescribe opioid medication results in higher risks of long-term opioid use or opioid abuse disorder, increased rates of depression diagnosis, and suggestive evidence of increased suicide attempts. The latter analysis is presented as secondary to the main analysis concerning opioid abuse outcomes, and no further discussion is provided on the potential implications of these findings with regard to the interaction between increased access to opioid medication and suicidal behavior. Borgschulte et al. (2018) evaluate the effects of PDMP, a policy tool that established databases that track individual drug prescriptions and has been shown to be effective in decreasing opioid abuse (Buchmueller and Carey, 2018). The authors find that PDMP implementation decreased suicides when there were more drug treatment centers available locally to enable a path out of drug dependency once supply was disrupted. While Borgschulte et al. (2018) tie a supply-side factor (PDMP) with suicide rates, the dependency of this result on the extent of drug treatment centers available implies that this channel is still not entirely supply-driven. In both Borgschulte et al. (2018) and Eichmeyer and Zhang (2023) the suicide mortality rates include drug-related suicide, so one cannot rule out the possibility that some of the effects observed are mechanical - i.e. limiting the supply or access to drugs also results in reduced drug-related suicidal attempts. Our study focuses on non-drug-related causes of death (NDS and ARM), as we wish to isolate the effects of increased drug supply on mortality as a secondary outcome that cannot be argued to be mechanical.

As drug mortality grew more severe over the years, its sources changed as well. At the start of the opioid epidemic, most drug deaths were due to prescription opioid abuse. However, by 2010, the vast majority of drug overdose deaths were due to illicit drugs, such as heroin and fentanyl (Alpert et al., 2018; Evans et al., 2019; Ruhm, 2019; Cutler and Glaeser, 2021). Nevertheless, prescription

opioid supply shifters that prevailed at the dawn of the opioid epidemic still affected drug mortality even 20 years later (Alpert et al., 2022; Arteaga and Barone, 2025), thereby demonstrating the long-lasting effects these factors had on drug mortality even after opioid medication was no longer the major contributor to drug mortality. These early supply factors were instrumental in shaping drug mortality for many years thereafter. Consequently, our analysis also focuses on this time span.[1]

3 Data

3.1 Mortality Data

Our analyses use restricted-use mortality data that identifies causes of death using ICD-9 or ICD-10 codes and includes geographic identifiers at the state or county level. The data are from the Center for Disease Control and Prevention (CDC) National Center for Health Statistics (NCHS). For alcohol-related mortality (ARM), we choose all ICD codes that relate the cause of death to alcohol, rather than concentrating on chronic liver disease conditions. This matches our underlying hypothesis more precisely - that opioid and drug abuse led to greater alcohol abuse, and as such, any cause of death related to alcohol could have increased as a result of greater supply of opioid medication. We therefore follow the ICD codes used in Arteaga and Barone (2025) and Evans and Lieber (2023), rather than those used in Alpert et al. (2022) and Case and Deaton (2015).[2] Our deceased span ages 25-64, as in other research examining morbidity related to these causes of death (Case and Deaton, 2022). We also utilize restricted-use mortality data to follow Arteaga and Barone (2025) and construct cancer mortality rates for the entire population at the commuting zone (CZ) level as of 1996.[3]

Our time span for the analysis is 1983-2020 for the triplicate analysis and 1989-2017 for the cancer and SSDI analysis. This is because the triplicate analysis is at the state level and we can use public-use mortality data for this analysis, whereas for the cancer and SSDI analysis, we are

[1]This draft uses restricted mortality data from the CDC for the period 1989-2017 for the cancer mortality and SSDI analysis. For the triplicate analysis we use public-use mortality data for the period 1983-2020. Subsequent drafts will expand the time span for the restricted data to 1983-2020, once data access has been approved.

[2]Causes of death are based on ICD-9 codes for years prior to 1999 and on ICD-10 for years from 1999 onward. Non-Drug Suicide ICD-9 codes: E950.6-E959. Non-Drug Suicide ICD-10 codes: X65-X84. Alcohol liver disease ICD-9 codes: 2971.3, 303, 350.0, 357.5, 425.5, 535.3, 571.0-571.3, 790.3, E860. Alcohol liver disease ICD-10 codes: E24.4, F10, G31.2, G72.1, I42.6, K29.2, K70, K85.2, K86.0, O35.4, Q86.0, X45, X65, Y15.

[3]Cancer ICD-9 codes are: 140-208 and 210-239, excluding 162 as specified in Table B1 in Arteaga and Barone (2025).

awaiting approval to use restricted-use data that extends beyond 1989-2017.

3.2 Population Counts

The data on population counts come from the Survey of Epidemiology and End Results (SEER). We use these data for the following purposes: (i) to construct the denominators at the state, CZ, or county level for adult mortality rate measures, SSDI recipient rates, or opioid pill usage rates; (ii) to construct control variables at the state, CZ, or county level for the share of the population of a particular age or demographic group.

3.3 Social Security Disability Insurance (SSDI) Data

We follow Evans and Lieber (2023) here. Specifically, we define for each county the share of those 18-64 on SSDI as the ratio of the number of individuals on SSDI in December 1989 from Moore (2020)'s compiled data and the corresponding 18-64 population from the SEER population for 1990.

3.4 Opioid Use Data

We use data on the legal supply of opioids from the Drug Enforcement Agency's (DEA) Automation of Reports and Consolidated Orders System (ARCOS) from Arteaga and Barone (2025). This contains distribution records of all Schedule II drugs by active ingredient (e.g. morphine, oxycodone) at the 3-digit ZIP code from 1997-2020. The primary variable we use is the per capita prescription opioids, per year. THis is calculated as the sum of oxycodone, codeine, morphine, fentanyl, hydrocodone, hydromorphone, and meperidine in morphine-equivalent doses (60 morphine-equivalent mg).

3.5 Summary Statistics

Table 1 presents summary statistics for each analysis - Triplicate, Cancer Mortality, and SSDI - by column. Populations-weighted mean mortality rates per 100,000 residents ages 25-64 are across states, CZ's, or counties for the Triplicate, Cancer Mortality, and SSDI analysis, respectively. The mortality rates presented are Non-Drug Suicide, Alcohol-Related Mortality, and Drug Overdose, as discussed in Section 3.1. In addition, the means and minimum and maximum values of the explanatory variable are presented for each analysis.

The "Number of Clusters" listed at the bottom of each column is the number of states, CZ's, or counties, in the Triplicate, Cancer Mortality, and SSDI analysis, respectively. Alaska and Hawaii counties are excluded from the Cancer Mortality and SSDI analyses, as county-level population measures were not available for these states prior to 1990 or 2000, respectively. Furthermore, the Cancer Mortality analysis excludes CZ's with a population under 20,000, and the SSDI analysis excludes counties with a population under 10,000.[4]

Table 1: Summary Statistics

	Triplicate Analysis	Cancer Mortality Analysis	SSDI Analysis
Non-Drug Suicide	13.36	13.37	13.35
	(3.49)	(4.81)	(7.29)
Pre-1996 Non-Drug	13.04	13.08	13.05
Suicide	(2.80)	(4.06)	(6.81)
Alcohol-Related Mortality	11.75	11.76	11.72
	(4.49)	(5.95)	(8.23)
Pre-1996 Alcohol-Related	10.39	10.44	10.37
Mortality	(3.36)	(4.91)	(7.62)
Drug Overdose	16.08	16.05	16.08
	(10.35)	(11.97)	(14.04)
Pre-1996 Drug Overdose	6.95	6.93	6.96
	(3.39)	(4.57)	(6.41)
Mean Explanatory Variable	0.69	201.13	1.82
	(0.47)	(41.67)	(0.74)
Minimum Exp. Variable	0.00	84.32	0.00
Maximum Exp. Variable	1.00	378.66	8.88
Number of Observations	1,479	17,864	89,059
Number of Clusters	51	616	3071

Notes: All means are populations-weighted across states, CZ's, or counties for the Triplicate, Cancer Mortality, and SSDI analysis, respectively, with standard deviations in parentheses. Mortality rates are per 100,000 residents ages 25-64. The Triplicate Analysis explanatory variable is an indicator for non-triplicate states - all states except for California, Idaho, Illinois, New York, and Texas. The Cancer Mortality Analysis explanatory variable is the CZ-level cancer mortality rate per 100,000 population (for all ages) as of 1996. The SSDI Analyses explanatory variable is the rate per 100,000 population ages 18-64 receiving SSDI benefits at the county level as of 1990. The Cancer Mortality and SSDI analyses exclude Alaska and Hawaii counties. Number of Clusters refers to the number of states, CZ's, or counties for each respective analysis. Data is for 1989-2017.

[4]Arteaga and Barone (2025) similarly exclude CZ's with populations under 20,000. Although Evans and Lieber (2023) do not apply a sample restriction based on county population levels, we apply this restriction to maintain consistency across the three analyses. Our SSDI analysis is not sensitive to this population restriction (recall that our regression specifications are population-weighted).

4 Regression Specifications

All three analyses explore changes in NDS and ARM rates after 1996, when OxyContin was introduced, as a function of exogenous determinants for local opioid supply: non-triplicate status at the state level (as of 1996), the cancer mortality rate at the CZ level (as of 1996) and SSDI receipt rate at the county level (as of 1990). Our specifications are event-study analyses with mortality rates as the dependent variables and the unit of observations is at the year and respective local level (state, CZ, or county).

The following outlines the state-level regression specification for the Triplicate event study analysis:

$$y_{sy} = \alpha_s + \gamma_y + \Sigma_{k \neq 1995} \beta_k \times (Non - Triplicate)_s \times 1(y = k) + X_{sy} + \epsilon_{sy} \quad (1)$$

where y_{sy} represents the mortality rate per 100,000 residents ages 25-64 in state s in year y. The specification includes state (α_s) and year (γ_y) fixed effects. The $(Non - Triplicate)_s$ indicator is equal to one for states that had a non-triplicate status as of 1996 and it is interacted with a full set of year fixed effects, excluding 1995. Equation 1 also includes time-varying state-level control variables (X_{sy}): white and female population shares, and the shares of the populations ages 18-29, 30-49, 50-64, above 65 years, and newborns.[5] Standard errors are clustered at the state level and regressions are weighted by state-level populations for ages 25-64.

The following outlines the CZ or county-level regression specification for the Cancer Mortality or SSDI event study analysis:

$$y_{cy} = \mu_c + \theta_{sy} + \Sigma_{k \neq 1995} \pi_k \times (ExpVariable)_c \times 1(y = k) + X_{cy} + \epsilon_{cy} \quad (2)$$

where y_{cy} represents the mortality rate per 100,000 residents ages 25-64 in CZ or county c in year y. The specification includes CZ or county fixed effect (μ_c) and state-year fixed effects (θ_{sy})[6] that control for time-varying state-level policies. The $(ExpVariable)_c$ is either the 1996 cancer

[5] Alpert et al. (2022) do not include any control variables in their triplicate analysis regression specification. However, for the sake of consistency across our three analyses, we included in all our regression specifications the same control variables that are utilized in Arteaga and Barone (2025). Results are not sensitive to this.

[6] For the state-year fixed effects in the cancer mortality analysis, we follow Arteaga and Barone (2025) and associate with each CZ the state that has the largest population share within the CZ.

mortality rate at the CZ level or the 1990 SSDI receipt rate among ages 18-64 at the county level. It is interacted with a full set of year fixed effects, excluding 1995. $X_c y$ are time-varying CZ or county-level demographics, as specified for equation 1. Standard errors are clustered at the CZ or county level and regressions are weighted by CZ or county populations for ages 25-64.

5 Results

5.1 Event Study Results

We present the results of event study analyses for all three sources of exogenous variation in the supply of opioid medication in Figure 2. Columns 1, 2, and 3 present results for the Triplicate, Cancer Mortality, and SSDI analysis, respectively. In the top panel, the dependent variable is the NDS rate for ages 25-64, while in the bottom panel, it is the ARM rate for ages 25-64. All regression results are graphical representations of all β_k or π_k estimates from equations 1 or 2, respectively, along with vertical lines representing their 95 percent confidence intervals.

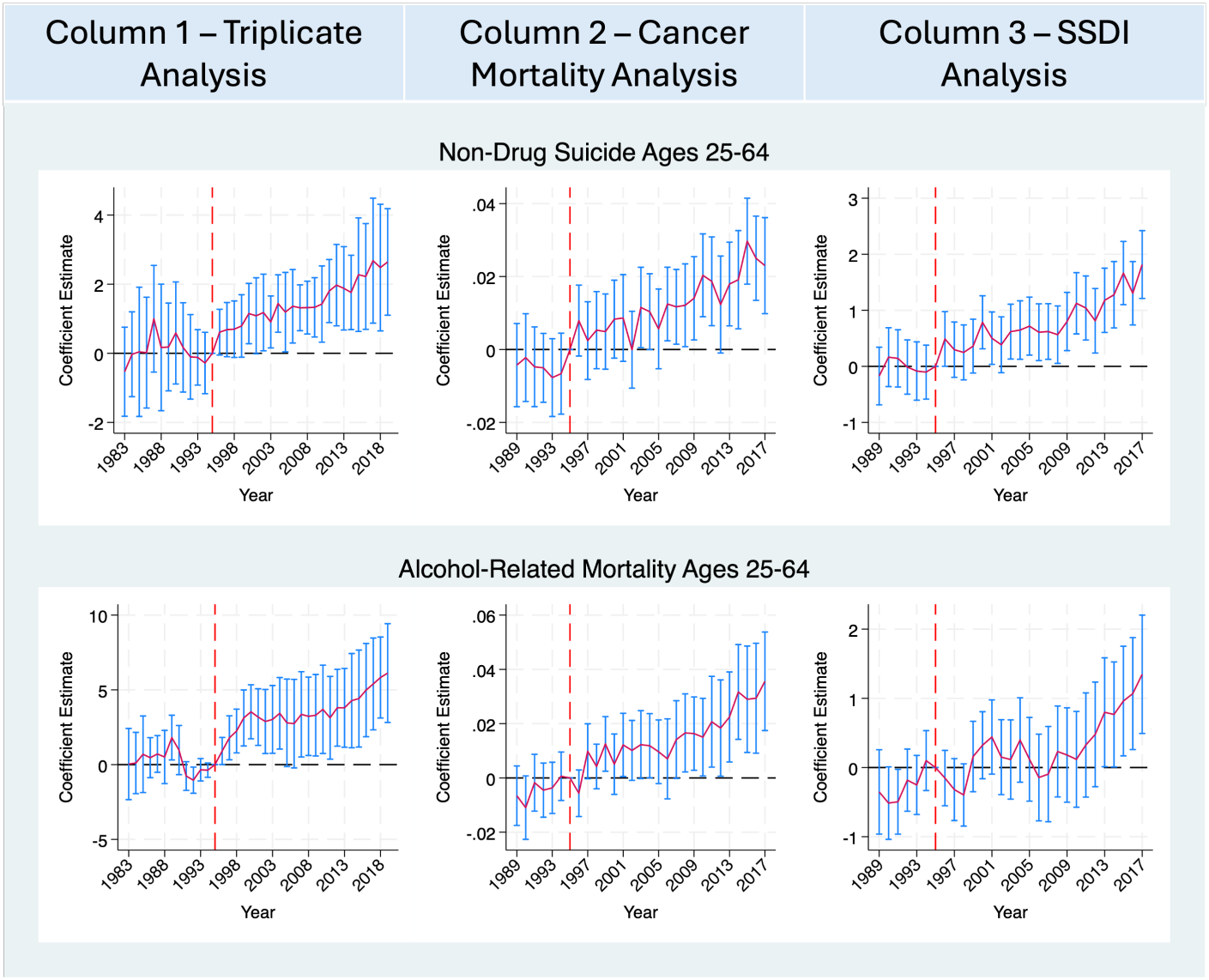
The results exhibit increases in NDS and ARM mortality rates beginning in 1996, or several years thereafter, in localities that experienced a greater supply of opioid medication due to one of our three exogenous supply shifters - non-triplicate status, greater cancer mortality, or greater SSDI recipient rates. The effects also appear to persist 20 years after the introduction of OxyContin and increase in magnitude. One slight outlier is the ARM mortality effect resulting from larger SSDI recipient rates, which only materializes post-2010.

In the Triplicate Analysis, the results suggest that non-triplicate states experienced roughly two additional NDS and ARM death rates per 100,000 population post-1996, in comparison to triplicate states. This is in comparison to an estimated increase of roughly 10 in the drug overdose mortality rate for the same age group and regression specification, as presented in Appendix Figure A1.

In the Cancer Mortality Analysis, the estimates in Figure 2 suggest that for each additional unit increase in the CZ 1996 cancer mortality rate, NDS and ARM rates increased by roughly 0.1. With a mean 1996 CZ cancer mortality rate of slightly over 200 (see Table 1), this implies an increase of roughly 2 deaths from each mortality measure per 100,000 residents. In Appendix Figure A1, the corresponding point estimates for drug mortality are five times larger.

Lastly, the SSDI Analysis estimates in Figure 2 suggest an increase of roughly 0.7 NDS deaths per 100,000 residents at the county level for each additional 1990 SSDI recipient per 100,000 residents

Figure 2: Event Study Analyses - Opioid Supply Shifters and Non-Drug Suicide / Alcohol-Related Mortality



Notes: The figure plots for each analysis the coefficient estimates β_k or π_k from equations 1 or 2, respectively, along with the 95 percent confidence intervals. In column 1, the coefficient estimates represent the increase in the mortality rate among non-triplicate states relative to triplicate states and the base year, 1995. In column 2, each coefficient estimate measures the change in the mortality rate for each unit increase in the local 1996 cancer mortality rate, relative to the base year, 1995. In column 3, each coefficient estimate measures the change in the mortality rate for each unit increase in the local 1990 SSDI recipient rate, relative to the base year, 1995.

ages 18-64. The estimated effect of each 1990 SSDI recipient per 100,000 residents on drug mortality post-1995 in Appendix Figure A1 is around five times larger. The ARM SSDI analysis in Figure 2 is less conclusive, although post-2010, there appears to be an increase of roughly 0.5 deaths per 100,000 residents for each additional 1990 SSDI recipient per 100,000 residents. With a mean SSDI rate of 1.82 (see Table 1), this suggests that larger opioid medication supply resulting from greater SSDI coverage increased NDS and ARM rates by an average of 1.27 and 0.91, respectively.

Overall, all three analyses suggest that the estimated effect of greater opioid medication supply at the dawn of the opioid epidemic on non-drug suicide and alcohol liver disease mortality is roughly 40% of the estimated effects on drug overdoses (the point estimates for each NDS or ARM analysis are roughly 1/5 of those found for drug overdose mortality and these causes of death are mutually exclusive). Interestingly, the magnitudes of the effects are similar for both NDS and ARM.

By taking the quantitative estimates from all three analyses and dividing these by the increase in NDS and ARM rates observed in Figure 1, we calculate the potential contribution of opioid medication supply shifters to rising mortality rates. The triplicate and cancer mortality analysis suggest that supply shifters contributed on average roughly 25 and 40 percent to the increase in NDS and ARM rates during the first two decades of the 21^{st} century. The SSDI analysis produces smaller estimates of 12 and 25 percent for NDS and ARM rates, respectively.

5.2 Instrumental Variable Results

Our analyses exploit three exogenous opioid medication supply shifters. The underlying hypothesis is that these shifters increased the local supply of opioid medication, thereby increasing opioid and drug abuse. The relationship between 1996 cancer mortality rates or 1990 SSDI recipient rates and opioid medication consumption has already been demonstrated in Arteaga and Barone (2025) and Evans and Lieber (2023), respectively, using Drug Enforcement Administration's (DEA) Automation of Reports and Consolidated Orders System (ARCOS) data (see Section 3.4 for data description). We use the ARCOS data to further establish our hypothesis: that the increase in opioid medication consumption, which led to increased opioid and drug abuse, also led to increased suicidal tendencies and alcohol abuse, thereby increasing mortality from these causes. We can thus apply an instrumental variable (IV) approach. Our dependent variable is the NDS and ARM rate. Our endogenous variable is the annual morphine equivalent doses prescribed per capita, and our instrument is one of the three opioid supply shifters - non-triplicate status at the state level, 1996

CZ cancer mortality rate, or 1990 county SSDI recipient rate.

We note that ARCOS data is only available beginning in 1997. As such, our empirical specification for the IV framework treats 1997 as the base year and assesses changes in opioid medication consumption or mortality relative to that base year, as a function of the explanatory variable (i.e. the opioid medication supply shifter). Our regression specifications follow closely equations 1 and 2 from Section 4, with population weights at the relevant locality level, standard errors clustered at the relevant locality level, and the fixed effects and control variables that are outlined in the equations for each analysis.

We present our IV results in Table 2 for each of the three instruments we use in their respective column. The first panel presents the first-stage regressions. The coefficient estimates are positive and statistically significant with F-statistics exceeding the minimal value required for the explanatory variable to be a valid instrument. The second-stage regression results are presented in the second column. They all suggest that for each additional morphine equivalent dose perscribed for each person would cause 1 more NDS/ARM death per 100,000 in the population.

The third panel of Table 2 presents second-stage regression results when the drug mortality rate, rather than the NDS and ARM mortality rate, is the dependent variable. This allows us to compare the magnitude of the effect of opioid medication usage between NDS and ARM mortality and drug mortality. We expect the drug mortality effect to be greater in magnitude than the NDS and ARM mortality effect because this is a more direct effect, whereas the NDS and ARM mortality is a secondary effect. Indeed, panel 3 of Table 2 presents effects on drug mortality that are 2.5-5 times greater than the effects on NDS and ARM mortality.

The fourth and fifth panels of Table 2 present OLS regression estimates for the relationship between opioid medication usage and NDS and ARM or drug mortality, respectively. For NDS and ARM rates, the estimates are small and statistically insignificant. This is in contrast to the much larger and statistically significant IV estimates in the second panel. While the OLS drug mortality estimates are positive and statistically significant, they are substantially smaller than the IV estimates for drug mortality. The sharp difference between the IV and OLS estimates suggests that opioid medication usage is also correlated with factors that negatively affect mortality. For example, opioid medication usage may be higher in more advantageous localities, and drug, suicide and ARM rates are greater among disadvantaged low-educated populations (Case and Deaton, 2017). This highlights the value of the IV approach in estimating a causal relationship between

opioid medication usage and mortality.

5.3 Reconciling Our Results with Other NDS and ARM Analyses

Our three explanatory variables are used in three separate studies evaluating the effects of opioid medication supply shifters on drug mortality (Alpert et al., 2022; Arteaga and Barone, 2022; Evans and Lieber, 2023). These studies perform supplementary analyses that evaluate the effects of their supply shifters on other causes of death, including NDS or ARM rates. The effects of opioid medication supply shifters on NDS or ARM are either ruled out or deemed inconclusive in these studies. This is in contrast to our results. However, a comparison of our analysis with the analysis in these studies reveals that the main (although not only) reason for this is that these other studies evaluate mortality rates for much wider population bases, in terms of age ranges. Below, we discuss the implications of these wider population bases separately for each study, along with other factors that generate the contrasting results.

In a supplementary analysis, Alpert et al. (2022) perform the triplicate analysis for NDS and chronic liver condition (CLD) mortality. Their results demonstrate a statistically significant increase in NDS and CLD mortality in non-triplicate states beginning shortly after 1996, similar to column 1 in Figure 2 above. However, their analysis exhibits pre-existing upward trends among non-triplicate states during the 1980's. This could imply that increases in NDS and CLD mortality among non-triplicate states post-1996 are due to varying trends prior to the introduction of OxyContin in 1996.

Our triplicate event study analysis - as presented in column 1 of Figure 2 - differs from the Alpert et al. (2022) NDS and CLD event study analysis in three aspects: first, our mortality rates are for ages 25-64, rather than the entire population; second, we include time-varying control variables in our regression specification - namely, demographic and age-group population shares at the state level - whereas the Alpert et al. (2022) event study specification does not include control variables; third, we use a broader set of alcohol-related causes of death and limit the liver conditions to just those related to alcohol.[7] It turns out that the first two above-mentioned factors are important for whether pre-existing trends arise in the triplicate event study analysis.

In Appendix Figure B1 we present a replication of the Alpert et al. (2022) event study analysis

[7] More specifically, Alpert et al. (2022) focus on ICD-9 codes K70 and K73-74, whereas we do not include ICD-9 codes K73-74. Their ICD-10 codes are 571.0-571.9, whereas ours do not include 571.4-571.9. We also have ICD codes that are alcohol-related causes of death that are not included in Alpert et al. (2022) - see footnote 2 for details.

Table 2: Opioid Pill Usage and Non-Drug Suicide & Alcohol-Related Mortality - IV Approach

	Triplicate Analysis	Cancer Mortality Analysis	SSDI Analysis
Stage 1 Regressions - Opioid Use Per Person as Dependent Variable			
Explanatory Variables X Post-1997	2.782***	0.0146***	0.837***
	(0.544)	(0.00357)	(0.188)
F-Statistic	26.17	16.76	19.86
Stage 2 Regressions - Non-Drug Suicide & Alcohol-Related Mortality as Dependent Variable			
Opioid Use Per Person	1.020**	1.042*	1.083***
	(0.428)	(0.533)	(0.380)
Stage 2 Regressions - Drug Mortality as Dependent Variable			
Opioid Use Per Person	2.724***	3.154***	5.101***
	(0.819)	(0.834)	(1.044)
OLS Regressions - Non-Drug Suicide & Alcohol-Related Mortality as Dependent Variable			
Opioid Use Per Person	0.149	0.00503	-0.0112
	(0.0980)	(0.0447)	(0.0127)
OLS Regressions - Drug Mortality as Dependent Variable			
Opioid Use Per Person	0.967***	0.526***	0.159***
	(0.267)	(0.103)	(0.0587)
Mean NDS & ARM Rate	25.63	25.62	25.49
Mean Opioid Use Per Person	7.789	7.775	7.788
Mean Drug Mortality Rate	18.99	18.94	19.06
Observations	1,068	12,936	50,064

Notes: All regression are population-weighted. Point estimates are presented with standard errors clustered at the state, CZ or county level in parentheses. Mortality rates are per 100,000 residents ages 25-64. The Triplicate Analysis explanatory variable is an indicator for non-triplicate states - all states except for California, Idaho, Illinois, New York, and Texas. The Cancer Mortality Analysis explanatory variable is the CZ-level cancer mortality rate per 100,000 population (for all ages) as of 1996. The SSDI Analyses explanatory variable is the rate per 100,000 population ages 18-64 receiving SSDI benefits at the county level as of 1990. The Cancer Mortality and SSDI analyses exclude Alaska and Hawaii counties. Number of Clusters refers to the number of states, CZ's, or counties for each respective analysis. Data is for 1997-2017 (drug use data only available beginning 1997).

for NDS and CLD mortality with and without control variables in the top and bottom panels, respectively. The results demonstrate that the inclusion of control variables eliminates all pre-existing trends. In Appendix Figure A2, we present our own triplicate analysis (for age group 25-64, rather than the entire population). When comparing the results in Appendix Figure A2 with the results in column 1 of Figure 2, it is clear that for age 25-64 mortality rates, the triplicate event study analysis is not sensitive to the inclusion of demographic or age-group population share control variables. Taken as a whole, we conclude that the pre-existing trends found in the Alpert et al. (2022) triplicate NDS and CLD analysis are due to shifts in the population shares of either the young (< 25) or elderly (65+) in triplicate vs. non-triplicate states, and these age groups likely have different NDS or CLD rates. An analysis using age 25-64 mortality rates should better reflect our underlying hypothesis regarding the interaction between opioid and drug abuse and suicidal tendencies or alcohol abuse, as this age group was more prone to experiencing opioid or drug abuse due to increased opioid medication supply. As such, the pre-existing trends found in the Alpert et al. (2022) triplicate NDS and CLD analysis are not relevant to our analysis.

Arteaga and Barone (2025) exploit 1996 cancer mortality rates at the CZ level to establish how the opioid epidemic shaped voting patterns. In a supplementary event study analysis, using a very similar specification to our equation 2, they show no relationship between 1996 cancer mortality rates and NDS mortality (Figure D1 in Arteaga and Barone (2025)). This is at odds with our results for NDS mortality in column 2 of Figure 2. However, the age range for the mortality rates in the Arteaga and Barone (2025) analysis is 20+, whereas our age range is 25-64. An expansion of the age group potentially affected by the opioid epidemic to elderly and younger adults can distort the true effects that opioid and drug abuse had on the more relevant target population - prime-age adults who are 25-64.

Evans and Lieber (2023) exploit variation across counties in SSDI receipt rates as their opioid medication supply shifter and demonstrate how this affected drug mortality. Here too the authors perform a supplementary analysis with NDS or ARM rates as dependent variables within an event study framework. Their results demonstrate some positive and statistically significant effects of 1990 SSDI coverage on NDS mortality, and the magnitude of these effects are only slightly smaller than the magnitudes observed in column 3 of Figure 2. Furthermore, the magnitudes of the estimates in the Evans and Lieber (2023) analysis do not increase over time and are statistically significant for less years, in comparison to the results in column 3 of Figure 2. However, the age range for the

NDS and ARM measures in Evans and Lieber (2023) is 18+, so as in Arteaga and Barone (2022), we believe a more expansive age range that includes the elderly and younger adult population produced less precise estimates for the effects of SSDI recipient rates on NDS and ARM rates.

6 Concluding Remarks

We have demonstrated the substantial role of supply factors in rising NDS and ARM rates during the first two decades of the 21^{st} century. This is a major shift in the prevailing notion that NDS and ARM are entirely demand-driven. We note that while our analysis solely captures mortality, mortality is representative of the wider prevalence of alcohol abuse and mental health issues. Our results therefore help us understand the devastating social and economic consequences of greater drug abuse, whether demand or supply-driven.

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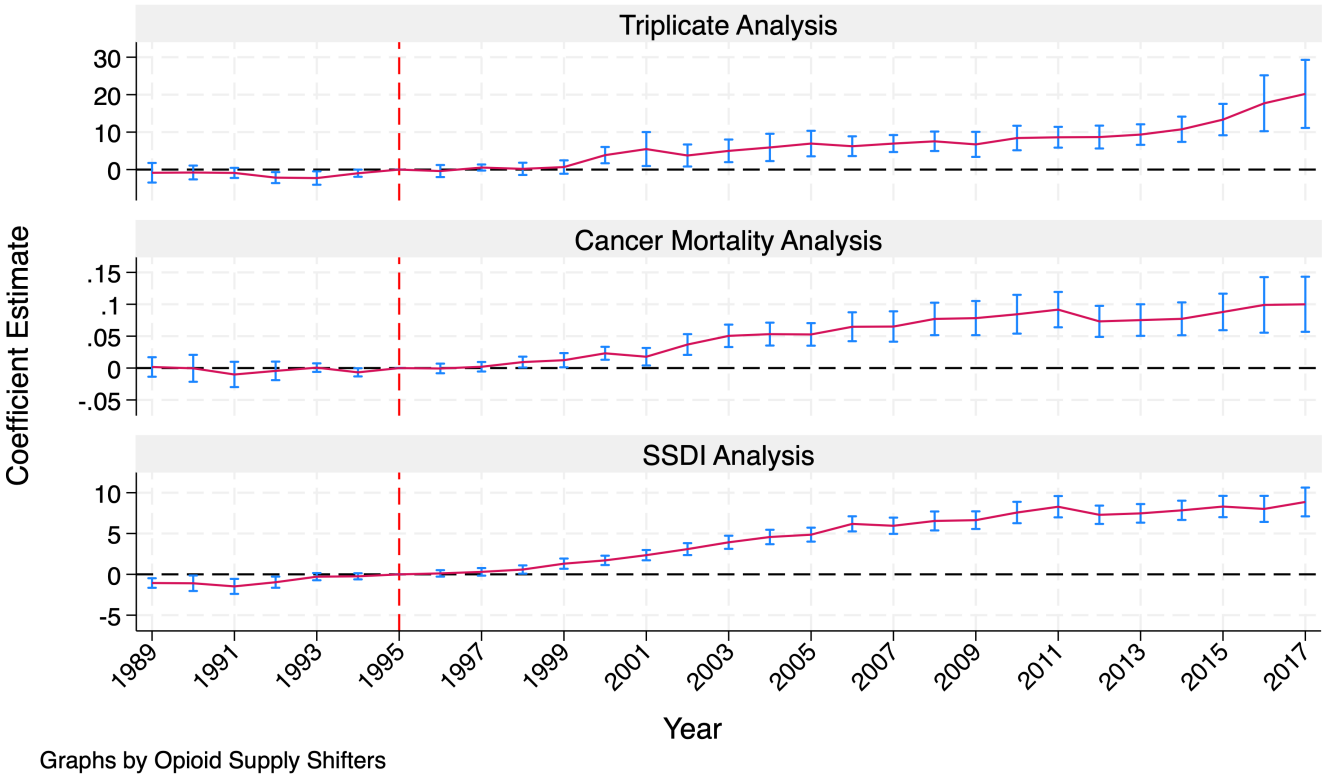
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Appendices

Appendix A Drug Mortality Event Study Results

Appendix Figure A1: Event Study Analysis - Opioid Supply Shifters and Drug Mortality



Notes: The figure plots for each analysis the coefficient estimates β_k or π_k from equations 1 or 2, respectively, along with the 95 percent confidence intervals. The dependent variable is the drug mortality rate for ages 25-64 at the state, CZ, or county level in the top, middle, or bottom panel, respectively. Standard errors are clustered at the state, CZ, or county level in the top, middle, or bottom panel, respectively.

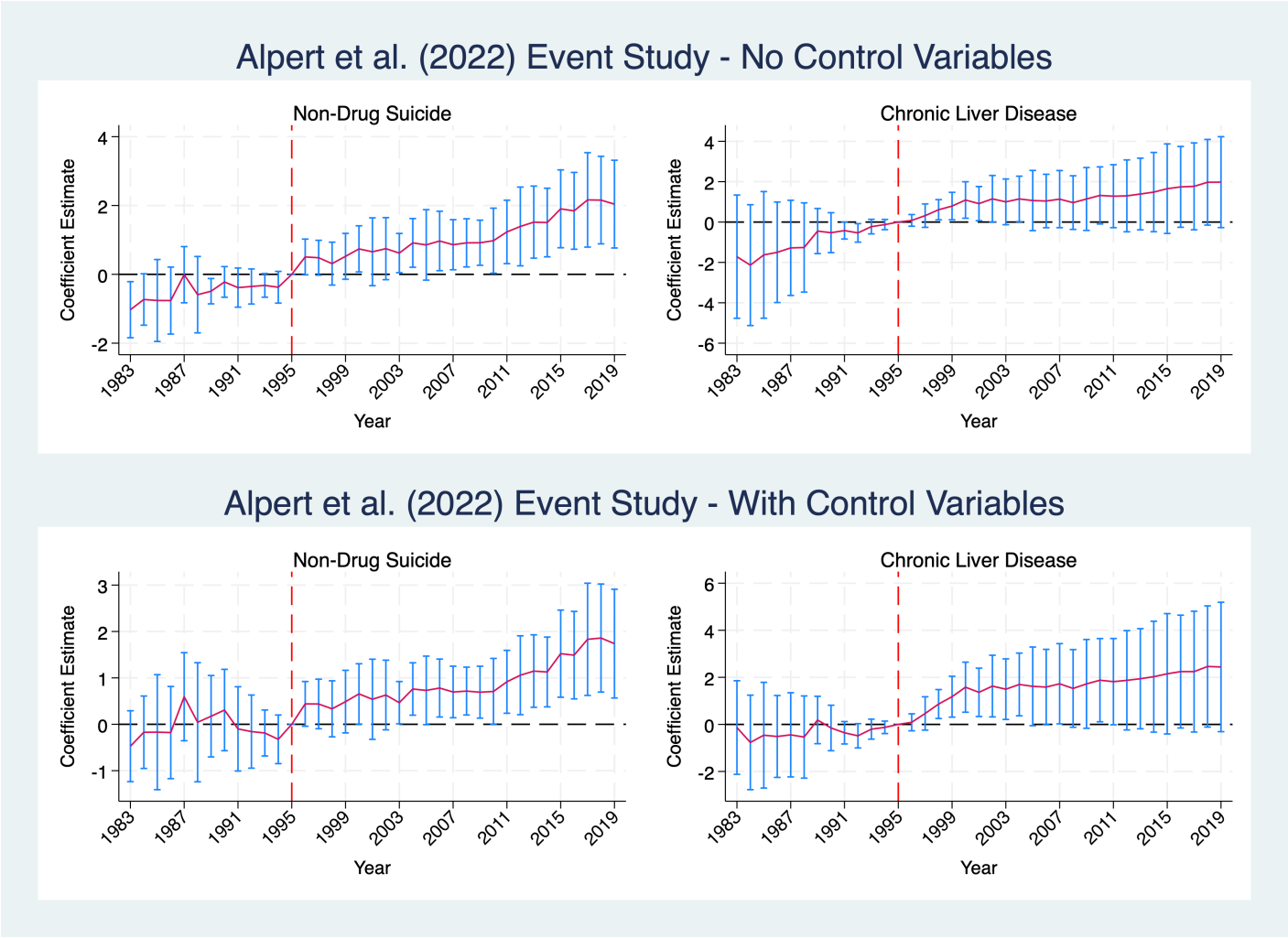
Appendix B Concern for Pre-Trends in the Triplicate Event Study Analysis

Appendix Figure B1 presents the triplicate event study analysis in Alpert et al. (2022) - using mortality rates for the entire population rather than ages 25-64, as in our analysis, and for chronic liver disease (CLD) conditions, rather than alcohol-related (ARM) mortality, as in our analysis - with and without control variables. The control variables are age group and demographic population shares (at the state level), as specified for equation 1. The top panel - without the control variables - is supposed to be an exact replication of the results presented in Figure A17 in Alpert et al. (2022). The bottom panel repeats this analysis but adds to the regression specification the control variables.

The results presented in Appendix Figure B1 suggest that once population shares for age groups and other demographics are accounted for, there is no evidence of pre-existing trends in the 1980's. This suggests that the pre-existing trends are due to changes in the demographic composition of triplicate vs. non-triplicate states during the 1908's, and that these demographic groups likely differ in their NDS or CLD mortality rates.

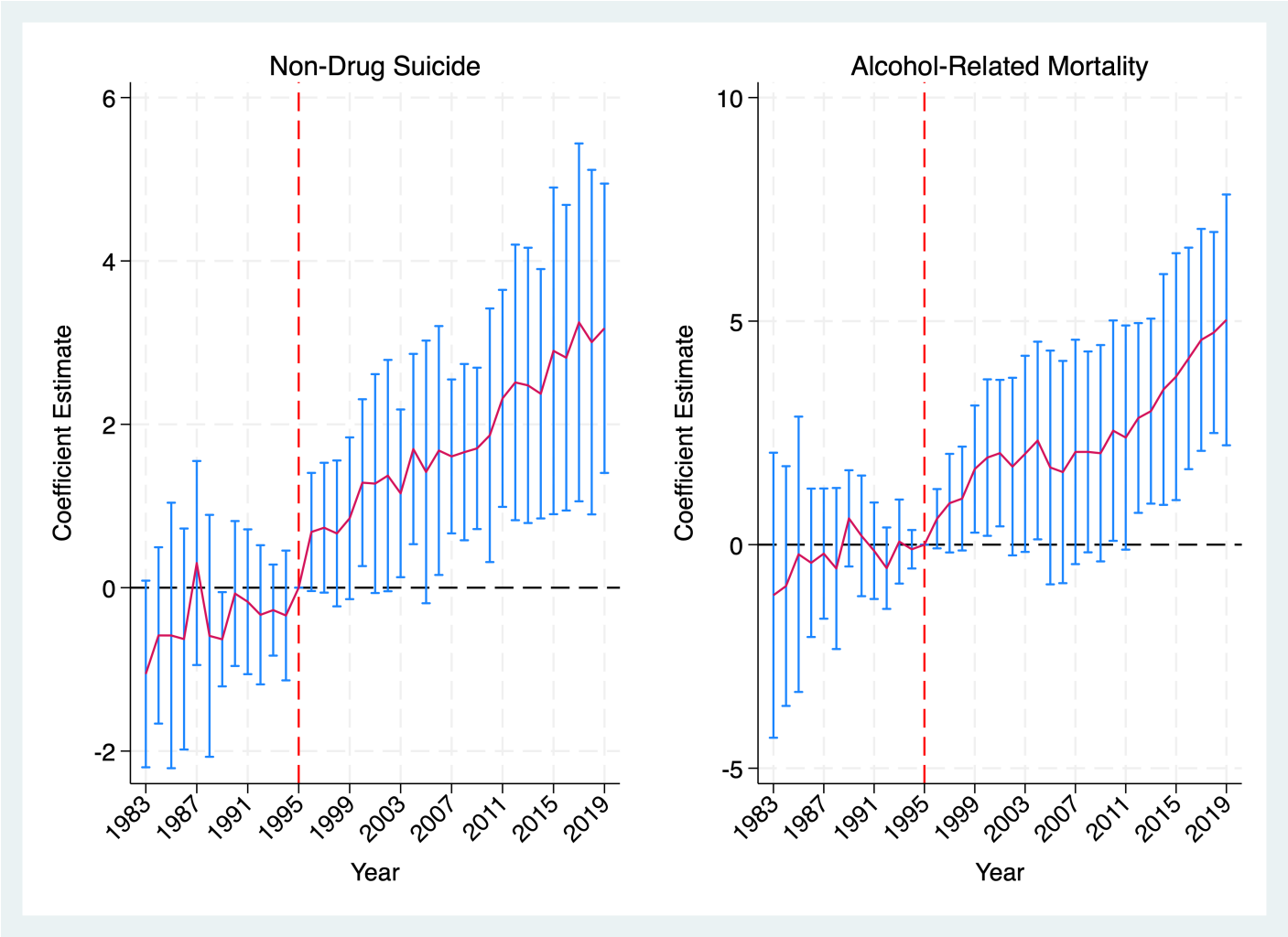
We evaluate whether our event study analysis - which uses mortality rates for ages 25-64, rather than for the entire population - is also sensitive to the inclusion of control variables. Appendix Figure B2 presents the triplicate event study results without control variables, and these should be compared to the results in column 1 of Figure 2. In both cases, there is no evidence of pre-existing trends. As such, we conclude that the pre-existing trends in the NDS and CLD analysis in Alpert et al. (2022) are driven by compositional demographic changes in the younger (< 25) and elderly (65+) populations that are included in the Alpert et al. (2022) analysis but excluded from our analysis.

Appendix Figure B1: - Triplicate Event Study Analyses in Alpert et al. (2022) with or without Control Variables



Notes: The figure plots for each analysis the coefficient estimates β_k from equation 1, along with the 95 percent confidence intervals. The dependent variables are NDS or CLD mortality rates for the entire population at the state level. Standard errors are clustered at the state level. Control variables are population shares by age group and demographics, as specified for equation 1.

Appendix Figure B2: - Triplicate Event Study Analyses without Control Variables



Notes: The figure plots for each analysis the coefficient estimates β_k from equation 1, along with the 95 percent confidence intervals, but control variables (X_{sy}) are not included in the regression specification. The dependent variables are NDS and ARM rates for ages 25-64 the state level. Standard errors are clustered at the state level.