Blue Collar Booms and American Mortality: Evidence from the Fracking Revolution*

Vivek S. Moorthy† and Paul Shaloka‡

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Abstract

We exploit the positive labor demand shocks driven by the fracking boom to investigate whether improvements in economic opportunity reduce mortality. Using variation in geological characteristics amenable to fracking within a difference-in-differences design, we find that the boom reduces overall mortality for working aged adults. We find no robust evidence of reductions in external forms of death, such as suicide. Rather, the reductions are concentrated among more medically treatable causes, such as cardiovascular deaths. Finally, we find evidence of increased health insurance coverage following the boom. Our results suggest that increased access to medical care serves as an important mediator in the relationship between labor market conditions and mortality.

JEL: I12, I15, J23, Q40, R12, R58

Keywords: Mortality, Suicides, Labor Demand, Fracking, Regional Development

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†College of the Holy Cross, email: vmoorthy@holycross.edu

‡US EEOC, email: paul.shaloka@eeoc.gov

I Introduction

A growing body of research shows that the negative impacts of job loss permeate beyond the labor market. The most pernicious of these effects center around health, as job loss increases BMI, alcohol consumption (Deb et al., 2011), depression (Schaller and Stevens, 2015), and even overall mortality (Eliason and Storrie, 2009). Much of this literature focuses on short-term job losses, while a smaller literature looks at the mortality consequences of larger long-term negative shocks. Further, there is even less evidence on how large and persistent *increases* in labor demand impact mortality. Whether these shocks necessarily lower mortality is not ex ante obvious, as short-term income receipt has been found to increase certain causes of mortality (Ruhm, 2000; Moore and Evans, 2012). The lack of evidence is partly due to the relative difficulty of finding quasi-exogenous variation that drives large changes in labor demand.

This paper overcomes that challenge and considers the effect of large, sustained, localized labor demand shocks on mortality by exploiting variation in the intensity and location of the hydraulic fracturing (fracking) boom. Feyrer et al. (2017) found that fracking led to the creation of more than half a million jobs, with positive spillovers beyond the mining industry, suggesting that the boom was transformative for local communities. To measure the mortality effects of the fracking boom, we use restricted data from the National Vital Statistics System (NVSS) to construct mortality rates at the county-level from 1990 to 2018. The setting and these data give us the rare opportunity to consider the effects of large-scale improvements in economic opportunity on an important health outcome.

To estimate the relationship between labor demand shocks and mortality, we have to overcome the endogeneity of fracking production. This can manifest in several ways: local regulations on drilling operations can limit or even outright ban fracking, and these decisions may be directly related to factors which could influence mortality, such as the strength of local labor markets and investments in public health. Further, places which benefited from the boom may differ from areas of the country with no fracking potential; for example, increasing opioid mortality was initially more of a rural phenomenon (Rigg et al., 2018).

To address this issue, we use a county-level measure of the potential profitability of fracking operations provided by Rystad, a private energy company. Crucially, this profitability measure is based on detailed geographic surveys, rather than the potentially endogenous realized level of extraction.[4] Specifically, we employ a difference-in-differences (DD) strategy that compares counties with higher geological potential for fracking to similar geographically adjacent counties that were less likely to benefit as much from the fracking boom. We do this in practice by comparing counties in the top-quartile of our profitability measure to others with lower potential within the same shale formation, referred to as shale plays, which are the geological formations that are amenable to fracking.[5] We also use the differential timing of the adoption of modern fracking technologies across shale plays, which enabled producers to construct wells over under-surveyed and previously inaccessible fossil fuel deposits. We find that while counties had similar levels of production and economic activity before fracking adoption, there is a sizable separation in economic activity between our treatment and control counties after the boom begins. Overall, employment and earnings increase by 2-3% over the 6 years following the start of fracking, and the effects increase over time. Although men are more likely to be employed in the mining and transportation sectors, we show that women also experience earnings and employment gains, likely through local equilibrium effects such as agglomeration (Allcott and Keniston, 2018).

We then show that overall mortality declines in boom counties for working age individuals (25-64) who are the direct beneficiaries of the earnings and employment increases. This group experiences a 2.5% reduction in all-cause mortality. The effects are strongest for older working age men and women (45-64) who experience 2.4% and 3.3% reductions, respectively.[6] We further show that our mortality results are not driven by differential trends in mortality before the fracking boom and are consistent across different functional forms and ways of measuring mortality. Using age-adjusted death rates as our outcome, we find a reduction of about 9.4 deaths per 100k, which translates to 14% of the overall decline in mortality between 1990-2000.[7] We also demonstrate that migratory responses are not driving our

[4] Bartik et al. (2019) pioneer this identification strategy.

[5] There 16 Shale Plays in our sample, which constitute contiguous counties across different regions of the U.S.

[6] While larger mortality reductions for women are consistent with work such as Dow et al. (2020), we fail to reject the null hypothesis that the effects are the same across genders.

[7] The age-adjusted death rate fell from 938 to 869 per 100k between 1990 and 2000: https://www.cdc.gov/nchs/data-visualization/mortality-trends/index.htm.

results. First, we control for the contemporaneous population in our baseline specifications. Second, our mortality results are robust when excluding counties within the Bakken shale play, which experienced a particularly large in-migration of male workers (Wilson, 2020). Next, we find only modest changes in overall population or age distributions between our treatment and control counties. And finally, our findings are robust to directly controlling for any compositional changes in the population.

To better understand the mechanisms underlying the reduction in mortality, we explore changes by more specific causes of death. We show that the fall in mortality attributable to the fracking boom is driven by reductions among treatable, internal causes of death, with the largest declines concentrated in the latest treatment years. This is consistent with Browning and Heinesen (2012), who find that job loss *increases* the risk of internal mortality using administrative data on workers and plant closures from Denmark. Similar to that study, we find that circulatory/cardiovascular mortality drives the reductions in internal causes of death. However, unlike previous studies on plant closures (Browning and Heinesen, 2012; Venkataramani et al., 2020), macroeconomic downturns (Hollingsworth et al., 2017), or large persistent negative shocks (Pierce and Schott, 2020), we do not find evidence of reductions in external causes of death like suicides or drug-overdoses, although our point estimates are negative.

There are many potential mechanisms through which improved labor market opportunities could reduce internal causes of death. Additional income is associated with better health (Chetty et al., 2016), and there are non-pecuniary benefits of employment such as increased self-worth (Noordt et al., 2014). Our findings also point to a health insurance mechanism. Increases in health insurance coverage have led to sizable mortality declines, concentrated in the same internal causes of death and among the same 45-64 year old age groups that we find here (Borgschulte and Vogler, 2020; Goldin et al., 2021), and Schaller and Stevens (2015) find that workers who lose a job that was their primary source of insurance reduce doctor's visits and prescription drug usage.[8] We do find suggestive evidence that health insurance coverage increases in boom counties by matching our fracking data to county-level coverage

[8] Einav and Finkelstein (2023) also show that the duration of lack of insurance spells following a loss in coverage are remarkably persistent.

estimates constructed from the American Community Survey (ACS).[9] Sommers (2017) finds that a 1 percentage point increase in coverage from state-level Medicaid (a public means-tested insurance program for the poor) expansions reduced overall mortality for working-age adults by 1.3%, whereas Goldin et al. (2021) finds a 1 percentage point increase in coverage led to a 5.7% reduction in mortality. With the restrictive assumption that increased coverage drives our findings, our results would imply a 1 percentage point increase in coverage leads to a 2% overall mortality decline, falling between these reductions induced solely by health insurance expansions, supporting this as a plausible mechanism. Moore and Evans (2012) find that increased income receipt leads to short-run mortality spikes over the following several days; the alternative mechanisms discussed here suggest that our results are driven by very different factors. Additionally, our results are over a longer time period and are based around a sharp, discontinuous, and unexpected change in employment and earnings rather than receipt of expected payments. These suggest increased access to medical care may serve as an important mediator between labor market conditions and mortality, particularly in a US context where insurance coverage is tightly linked to employment.

Our paper contributes to work on the effects of labor market outcomes on health and mortality outcomes. While the existing literature has exploited plant closures to generate quasi-experimental variation in labor market opportunities, we consider the effects of plant (fracking well) *openings* on labor demand and mortality. It is not obvious ex-ante whether the size of the effects we observe would be of similar magnitude to these studies. The shock and stress of job loss are likely to have consequential, immediate health impacts, which may lead to important non-linearities in the effect of employment changes on health outcomes. Iizuka and Shigeoka (2021) finds that demand responses to price increases for child healthcare are twice that of the change induced by price decreases, suggesting increases in income and coverage may not induce as dramatic changes in behavior as decreases along those dimensions.

We can compare our results to the closest papers in this literature to our own study. Sullivan and Von Wachter (2009) exploit plant closings in Pennsylvania and find that sustained

[9] Anecdotally, fracking jobs provided fairly robust health insurance. Surveys from Rigzone, a large online oil and gas industry job posting site and career network platform, show that "Oil and gas professionals have become quite accustomed to rich health benefits offerings". An industry health consultant even bemoans the fact that generous health packages have become expected, and simply providing good coverage does not grant a competitive advantage in attracting employees. https://www.rigzone.com/news/survey_shows_oil_gas_workers_want_rich_health_benefits-19-sep-2019-159825-article/

employment and earnings losses of around 10% after a decade leads to a 17% increase in mortality, with the effects being larger for displaced workers under 55. Using Danish administrative data, Browning and Heinesen (2012) finds that job displacement leads to slightly smaller earnings declines over a 20-year window following the initial job loss, and that overall mortality increases by almost half the amount found by Sullivan and Von Wachter (2009). Similar to us, Browning and Heinesen (2012) find that changes in mortality from circulatory disease are an important dimension for explaining the overall mortality results. The reductions in mortality we observe relative to the change in earnings and employment are similar in magnitude, suggesting a symmetric response.

Our second contribution is to the literature on "deaths of despair" by providing some of the first evidence of the effects of a large positive shock to local labor markets. The Case and Deaton (2017) hypothesis that labor market conditions matter, especially over the long-run and at the time of entry into the labor market, suggests that the fracking boom may lead to reductions in "deaths of despair," and implies that this overall decline may be driven by reductions in external causes of death. However, we do not find any robust evidence of reductions in external causes broadly or deaths of despair specifically, albeit with imprecise estimates for overdoses. Several papers find that increased opioid mortality is largely driven by supply-side changes in opioid availability (Currie and Schwandt, 2020; Alpert et al., 2022), suggesting that there is less of a role for increased economic opportunity to play in reducing deaths of despair.

Our paper is also related to the literature within and outside of economics that directly assesses the health effects of hydraulic fracturing. Literature here has found adverse health impacts on infant and adult health from fracking-induced air and water pollution (Denham et al., 2021; Hill and Ma, 2022), and Jemielita et al. (2015) and Denham et al. (2019) show that increased fracking correlates with higher hospitalization rates. Closest to our work, Boslett and Hill (2022) uses two-way fixed effects panel regressions to find that deteriorating economic conditions from declining coal mining are associated with increases in mortality, but fracking is associated with higher suicides and otherwise has limited impacts on mortality. Our findings of decreased mortality do not directly contradict this literature. Rather, our focus is to estimate

the mortality response due to the economic improvements generated by the boom, rather than to measure the direct negative health impacts from fracking production directly. Our empirical strategy compares only those counties with higher to lower fracking potential within the same shale play, meaning that both treatment and control groups experience fracking production. This minimizes the potential of capturing the negative impacts of fracking production itself in our estimates. We confirm this by not finding any mortality increases among groups that are more susceptible to heightened mortality and morbidity from air and water pollution, such as infants or adults over 65 years of age.[10] In this way, our findings are more closely related work that exploits the fracking boom to test how economic opportunity impacts other behavior such as human capital investment (Cascio and Narayan, 2015), family formation (Kearney and Wilson, 2018), and crime (Street, 2018). Our results show that labor market gains from positive economic shocks can lower certain forms of mortality through similar mechanisms as job loss, such as health insurance access and psychological well-being.

II Background on the Fracking Boom

Oil and natural gas firms drill traditional wells vertically above large concentrated fossil fuel reservoirs. By contrast, unconventional fracking wells exploit far more dispersed fossil fuel reserves that remain trapped within sedimentary, organic-rich rock formations called shale plays. Companies began limited drilling of these shale plays as early as the 1960s, but the low permeability of the shale prevents oil and gas from pooling into the reservoirs conventional wells are typically drilled over, rendering traditional production techniques unprofitable.

New advancements in horizontal drilling and hydraulic fracturing enabled the fracking boom. Horizontally drilled well bores can access large areas of shale at once, obviating the need to drill many vertical wells. Fracking also involves injecting a highly pressurized slurry into the well bore, which fractures the surrounding shale and allows the encased oil and natural gas to flow freely. While the presence of a shale play is a necessary condition for fracking, actual production is sensitive to several geological factors, including the permeability of the

[10] We also further complement work such as Black et al. (2021) that discuss the difficulties in establishing causal relationships from fracking by demonstrating that when we alternatively compare fracking regions to non-fracking regions, we find strong evidence of confounding pre-trends in mortality.

rock, as well as the size and density of the hydrocarbon deposits.

Oil and gas firms did not immediately adopt the new technologies that enabled widespread, profitable fracking, partially because private and academic researchers were initially unaware of the true magnitude of the hydrocarbon reserves. For example, the US Geological Survey estimated in 2002 that the Marcellus Shale (covering WV, PA and NY) held two trillion cubic feet of recoverable natural gas. By 2011, these estimates had risen to 84 trillion cubic feet, based on new surveys. This large correction highlights how little understood the shale deposits were before they became exploitable.[11] Figure 1 Panel B plots the dramatic increase in fracking production over time from 2000, where it accounted for barely any of total US oil and natural gas production, to 2014, when it overtook the output of more traditional methods.

Both academic researchers and the popular press have linked the "fracking revolution" to labor market opportunities. Maniloff and Mastromonaco (2017) review various studies of both the local and national earnings gains attributable to fracking, and document estimates of wage growth which range from 2.6% to 16.75%. While the initial job growth is concentrated in the mining industry, the operation of even a single fracking well involves over 6,000 one-way trucking trips (Xu and Xu, 2020) to haul the water and sand needed for the hydraulic fracturing process. Finally, Allcott and Keniston (2018) find that the manufacturing sector actually grows overall in the wake of natural resource booms in the US (driven by upstream and locally-traded sub-sectors), and so there is little evidence of negative spillovers caused by a "Natural Resource Curse".

III Data

We aggregate all our data to the county-year level. We use county definitions as of the 2000 decennial census,[12] and our main sample includes data from 1990 to 2018. As we discuss below, our empirical strategy only compares counties under the same shale play; thus we omit

[11] Source: https://www.usgs.gov/news/usgs-estimates-214-trillion-cubic-feet-natural-gas-appalachian-basin-formations.

[12] If county boundaries change over time, we aggregate to the 2000 boundary definitions using initial population weights. For example, in 2001, Broomfield, Colorado is created from parts of Adams, Boulder, Jefferson, and Weld counties, and the Census Bureau reports the resulting population loss for each of the original counties. Source: https://www.ddorn.net/data.htm

counties that do not intersect with a shale play from our main sample. We further omit two Texas counties with several years of missing mortality data, including Loving Texas, which has fewer than 100 residents as of the 2020 Census. This leaves us with 484 counties (104 of which are in the top-quartile of the within-play RPI) and 29 years of data.

III.A Fracking Data

The U.S. Energy Information Administration (EIA) provides shape files defining every known shale play, which we use to identify counties that have any fracking potential. We also take data on the monthly prices of oil and natural gas from the EIA.[13] We obtained well-level production data from Enverus, a private oil and gas software company, through their academic outreach initiative. These data include information on both production and the orientation of the well bore, which we use to identify fracking wells.[14]

To capture variation in fracking suitability *within* shale plays, we purchased the NASMaps product from Rystad Energy, a private energy research company. The company produces a Rystad "prospectivity index" (hereafter referred to as RPI), a continuous, non-linear measure of how amenable a specific location within a shale play is to fracking production. Importantly, this measure is not based on realized/actual fracking production, but only on the underlying geological potential of an area. The index ranges from zero to five, with larger numbers representing increased potential fracking yields. We aggregate this measure to the county level, and we show which counties have any fracking potential (RPI greater than zero) in Figure 1 Panel A. Since the methodology used to calculate the RPI is unique to each play, the measure is not directly comparable across broad geographic areas.[15] We therefore follow Bartik et al. (2019) and identify counties which are in the top-quartile of the prospectivity index within each shale play, and these counties (which are more likely to be the most productive: our treatment counties) are shaded darker in Figure 1 Panel A.

[13] For oil prices we use the Cushing, Oklahoma spot price for West Texas Intermediate crude oil, and for natural gas we use the city-gate price.

[14] We identify fracking wells as any well with a non-vertical well bore orientation. DrillingInfo, the production database provided by Enverus, is also used by the EIA for their official releases concerning US production.

[15] After forming a zero to five sub-score index based on the parameters available for a given play, the final index is a weighted average of each sub-score. As an example, these parameters include lime thickness and lime depth for the Mississippian Lime shale play, and thickness, depth, and thermal maturity for the Utica shale play.

While Bartik et al. (2019) has shown that counties within the same shale play are more comparable along many economic dimensions, our analysis requires that these counties be comparable along dimensions that are relevant to our mortality outcomes. We confirm whether our control counties provide a good counterfactual to our top-quartile counties by comparing them along various county-level characteristics from 1990 Census (well before the technology that enabled fracking was first applied), including median household income, share of the population that are veterans, and other demographic information. Different demographic groups have varying propensities for succumbing to deaths of despair, and may also be differentially located across shale play counties. Controlling for these baseline characteristics can improve precision and ameliorate worries that our estimates are being driven by factors besides the fracking boom. Table 1 shows baseline 1990 summary statistics for top-quartile and other shale play counties, and shows that there are no statistically or economically significant differences (in terms of 1990 characteristics) between treatment and control counties before the boom. We show these same baseline differences across all shale play counties and the rest of the lower 48 states in Appendix Table 1. Shale play counties are poorer and more white (91%) than the rest of the country, although residents are more likely to be married. Shale play counties also have a lower age-adjusted death rate per 100,000 residents in 1990.

In addition to the cross-sectional variation in fracking potential, the timing of fracking adoption varied across shale plays. The gray bars in Figure 1 Panel B indicate the number of shale plays for which fracking potential became public knowledge in that year, which we take from Bartik et al. (2019). While firms begin exploratory adoption of new fracking technologies in the Barnett shale play in Texas as early as 2001, more well-known fracking hot spots like the Barnett shale play in North Dakota and the Marcellus Shale plays in the Mid-Atlantic do not begin widespread fracking production until 2007 and 2008, respectively. Despite an initial lag, top-quartile RPI counties produce substantially more than other three quarters of shale play counties combined.

III.B Employment and Earnings Data

We use county-level data on earnings and employment from the Quarterly Workforce Indicators (QWI) database, which is an aggregation of micro-level records from the Longitudinal Employer-Household Dynamics (LHED). These data are primarily based on unemployment insurance earnings data from participating states[16] available for a limited number of two-way group tabulations, including sex-age and sex-education. We focus on aggregate changes to employment and earnings instead of restricting attention to the natural resource extraction industry. Previous work on agglomeration such as Greenstone et al. (2010) suggests that the opening of large work sites may create positive spillovers for other industries, and Feyrer et al. (2017) finds evidence for such spillovers in response to the fracking boom. We aggregate our main variables of interest, average quarterly earnings and total quarterly employment to the yearly level. Specifically, we take the simple average of employment, and the employment-weighted average of earnings across all 4 quarters in a year.

III.C Mortality Data

We use a restricted-access version of the National Vital Statistics System (NVSS) mortality files from 1990-2018, which represents a census of all deaths in the United States. These data identify basic demographic information, primary/additional causes of death, and contain identifiers for the county of residence and occurrence. We follow Stevens et al. (2015) by separating all causes of death into mutually exclusive categories,[17] further separated into whether the causes of death are internal (cancer, cardiovascular, etc.) or external (homicides, motor vehicle accidents, etc.). For external causes of death, we also include "deaths of despair": suicides, drug-related deaths, and alcohol-related deaths, using the definitions provided by the US Congress' Joint Economic Committee. Since our data span across the use of ICD-9 and ICD-10 codes for reporting causes of death, this use of consistent, broad categories ensures comparability across time.

[16] In the earlier years of our sample, The QWI has limited coverage, which leaves very few observations before 2002, after which we have full coverage of every county in our main sample. The main earnings and employment results are robust to limiting the sample to years when we have data on all shale play counties, as shown in Appendix Table B.4 and Appendix Table B.5.

[17] We do not consider two of the broad death categories used in Stevens et al. (2015): other/unspecified/ill-defined and miscellaneous, in part because this latter category includes drug abuses which we look at separately.

Our primary outcome is the inverse hyperbolic sine (IHS) of the number of deaths among working age (25-64) individuals, where the contemporaneous population of the relevant demographic group is included as a control. We take these population data from estimates constructed by the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program. The IHS transformation allows us to retain county-year observations with zero deaths, which occur often for some of the more uncommon causes of death. We also use death rates per 100k (crude death rates) and age-adjusted mortality rates per 100,000 residents. While the crude death rate is just the total number of deaths for a specific demographic group divided by the current relevant population, the age-adjusted death rate is a weighted average of crude death rates across standard age categories, where the national population shares in those age categories in 2000 are the weights.[18]

IV Empirical Strategy

Advancements in horizontal drilling and slick water fracturing enabled the extraction of previously inaccessible reserves of oil and natural gas from shale plays. However, since the level of production is endogenous with respect to local labor market characteristics and the regulatory environment, simple comparisons using this measure may introduce bias. For instance, high-productivity areas may have had upward trending economic growth which enabled more widespread and earlier adoption of fracking technologies. In addition, areas that expanded fracking may have had different levels of pre-existing environmental conditions and/or zoning regulations that may be correlated with factors that influence mortality like pollutants such as radon (Black et al., 2019) or the level of public investment. Appendix Table 1 reports baseline comparisons across all shale play counties and the rest of the lower 48 states. Shale play counties are poorer and more white than the rest of the country, and have a lower age-adjusted death rate per 100,000 residents in 1990. These highlight the necessity of carefully constructing the appropriate counterfactual.

Following the approach pioneered by Bartik et al. (2019), we use variation in the RPI to account for these issues, which provides a straightforward approximation of the exogenous

[18]This adjustment is standard in the literature, and accounts for changing demographic patterns over time.

variation within a play that determines the extraction potential of fracking wells, and therefore the intensity of the positive labor demand shock. Combining this with temporal variation in the initiation of fracking in each play leads to the following DD specification:

$$y_{cpt} = \beta(\textit{Top-Quartile}_{cp} \times \textit{Post}_{pt}) + \sum_{t} \Psi_t(\mathbf{I}_{year=t} \times \mathbf{X}_{c,1990}) + \lambda_c + \gamma_{pt} + \epsilon_{cpt} \quad (1)$$

where y_{cpt} is the outcome of interest. $\textit{Post}_{pt}$ is an indicator for whether shale play p had adopted fracking by year t. $\textit{Top-Quartile}_{cp}$ indicates whether county c is in the top-quartile of the RPI for shale play p. Ψ_t captures the potentially time-varying effects of $\mathbf{X}_{c,1990}$, a vector of initial county-level characteristics.[19] Our baseline specification uses the IHS of mortality as the outcome variable and controls for the relevant contemporaneous population. We also control for time-invariant county characteristics with county fixed effects, λ_c. Regressions are weighted by the 2000 population [20] and all standard errors are clustered at the county level.

Including play by year fixed effects, γ_{pt}, captures play-year shocks and ensures our results are based on variation between counties *within* shale plays. These effectively aggregate estimates from each shale play. Since the timing of fracking adoption varies at the play level, adding these fixed effects safeguard our results from issues associated with staggered treatment timing (e.g. Goodman-Bacon, 2018). Due to this design, we drop any county that is not over a shale play. This leaves us with 407 shale play (control) counties and 112 top-quartile RPI (treatment) counties.

Since the timing of fracking adoption varies across shale plays (Figure 1 Panel B), the simple difference-in-differences coefficient is subject to composition bias as the number of years post-treatment varies across the sample. We restrict the data to a balanced sample, where the balanced sample is defined by whether every shale play experiences the same number of lags and leads in event years. In the main mortality sample, we have data for each play 11 years prior to treatment and 7 treated years (including the year of initiation of fracking), or 18 event-years of data for each observation.[21]

[19] All the variables shown in Table 1 are included as controls aside from the initial age-adjusted death rate.
[20] We show robustness to these weights in the Appendix.
[21] We show that our results are robust to using an unrestricted unbalanced sample in Appendix Figure A.8.

The identifying assumption of our DD model is that the control counties within plays provide an estimate of the counterfactual time-path of mortality and labor market outcomes had fracking intensity been lower in boom counties. While this assumption can never be directly tested we examine whether our treatment and control counties have the same pre-treatment trends by running the following event study specifications where we replace the $Post_{pt}$ indicator with a vector of event year indicators, omitting the event year prior to fracking's introduction:

$$y_{cpt} = \sum_{n \neq -1} \beta_n (\textit{Top-Quartile}_{cp} * \mathbf{I}_{year-\tau_p=n}) + \sum_t \Psi_t (\mathbf{I}_{year=t} * \mathbf{X}_{c,1990}) + \lambda_c + \gamma_{pt} + \epsilon_{cpt} \quad (2)$$

where τ_p represents the time of initiation of fracking in a given play. The coefficients β_n trace out the difference in outcomes between top quartile and other counties within a play, in a given event year n relative to omitted year. Given the concerns with using the IHS for count outcomes (e.g. Cohn et al., 2022), we also use crude and age adjusted death rates as the outcome, and estimate exponential models of (2) using Poisson regressions.

A key feature of our identification strategy is that the RPI accurately predicts the highest intensity boom counties in terms of actual production. Figure A.1 shows a flat, almost nonexistent pre-trend in production followed by an immediate increase after the boom begins, although production does not really begin to take off until the second and third year after the adoption of fracking technologies.[22] We also show that production increases in a similar, albeit attenuated, manner whether we define treatment using our standard top-quartile definition or whether we expand treated counties to include counties above the median play-level RPI measure or simply look at a standard deviation shift in the actual underlying RPI values. We can see that fracking production is nearly $400 million greater in top-quartile counties six years after the boom begins, indicating a meaningful separation between treatment and control counties.

[22]Since treatment timing is determined by when fracking became public knowledge within a play, and because hydrocarbon deposits within shale plays were relatively under-surveyed, an initial lag in production is not surprising.

V Results

V.A Earnings and Employment

To demonstrate the first order labor market effects of fracking, Figure 2 shows the gender-specific results of the fracking boom for earnings and employment using Equation (2). Panel A and Panel B report estimates for the log of average earnings for all employees, while Panel C and Panel D show results for the log of the average employment to population ratio. The dark and lighter shaded bars represent the associated 90% and 95% confidence intervals, respectively. Results from estimating Equation (1) are shown above each event study.

Overall, Figure 2 shows that earnings and employment increased for both men and women following the fracking boom, and continued to do so for up to six years after the adoption of fracking technologies. While the average effects show a roughly 3% increase in earnings and employment for men, the coefficients for later event-years are larger and seem to finally begin settling closer to 3-4%. Since our specification only uses within-play variation, and because fracking production is also increasing in our control counties (Figure 1 Panel B), our results do not represent fracking's *overall* impact, but instead leverage variation in plausibly exogenous production ability. Thus these labor market effects are likely smaller than the overall impact of fracking.

Despite the anecdotal evidence that fracking is an almost entirely male-dominated field, we find wage and employment growth for women.[23] For women, both earnings and employment increase by around 2%. However, both Bartik et al. (2019) and Feyrer et al. (2017) show that the boom led to substantial positive spillovers to other industries, with Feyrer et al. (2017) finding that in 2012, half of the overall employment increases attributable to the boom were actually sectors not directly related to extraction, while 30% were concentrated in the transportation sector and only 20% of the overall increase in employment came from the mining sector.

Kearney and Wilson (2018) also find differential sizes of the male and female labor demand

[23] According to the US Bureau of Labor Statistics, men made up over 84% of the workforce in mining, quarrying, and oil and gas extraction industries as of 2019.

shocks in response to the boom, and they find slightly larger effects for male earnings (4%) and the employment-to-population ratio (5%) than our main results suggest. We show in Appendix Table B.4 and Appendix Table B.5 that when we do not include county-level population weights our results are roughly similar to Kearney and Wilson (2018), suggesting that some more sparsely populated counties experience the largest relative production booms which men were differentially able to benefit from.[24]

V.B Mortality Results

Having confirmed that fracking adoption led to a sustained increase in labor demand, we now consider the reduced-form effects of fracking amenability on mortality. Primarily this is because we are unable to disentangle whether the increase in earnings or the increase in employment and the associated non-pecuniary benefits (increased optimism, access to health insurance, etc.) are leading to changes in mortality.

Table 2 looks at the effects of the fracking boom on overall mortality. The dependent variable is the inverse hyperbolic sine of overall mortality. In addition to all controls mentioned earlier, these specifications also include the log of the contemporaneous population of the relevant group as an independent variable. Column (1) shows that overall mortality fell by 2.4% in top-quartile counties relative to their shale play counterparts.[25] Columns (3)-(6) examine the mortality effects by gender. These reveal a 2% decline in overall mortality for men, and an even larger magnitude 3% decline in mortality for women.[26]

We also check whether our results are driven by migration and changes in population composition. While it is not obvious ex ante that migrants select based on higher or lower mortality risk, particular among older cohorts (Fletcher et al., 2022), Wilson (2020) found a sizable migration response to the fracking boom, particular in the Bakken Shale play, intersecting North Dakota and Montana.[27] First, recall that all base specifications control for

[24] Additionally, the Kearney and Wilson (2018) measure boom intensity using a simulated instrument for actual production, comparing fracking counties to others within the same state. The RPI is not constructed from contemporaneous production, and we only compare counties within the same shale formation.

[25] Similar to log-linear models with dummy variables, the semi-elasticities for in an arcsinh-linear specification with a dummy variable can be approximated as $\exp(\beta)$-1. See Bellemare and Wichman (2020) for a direct derivation.

[26] However, we fail to reject a Wald test of whether the effects for men and women are statistically indistinguishable from each other.

[27] Further, Arthi et al. (2022) highlights the importance of migration in impacting measured mortality.

the contemptuous population on the right hand side. Next, we follow Kearney and Wilson (2018) and omit both North Dakota and Montana from our sample. Columns (2), (4), and (6) of Table 2 show that our mortality results remain of similar magnitude without the inclusion of these high in-migration regions. We examine migratory influences in more depth in Section *V.D*.

Figure 3 Panel A plots the estimates from equation (2) for overall mortality.[28] Panels B and C separately examine overall mortality for men and women, respectively. The shaded bars represent the associated 95% and 90% confidence intervals. Overall, there is a reassuring absence of differential trends in mortality between treatment and control counties before the initiation of fracking. After fracking, there begins a decline in overall mortality. While imprecisely estimated, there is a 1-2% decline in mortality starting 2-3 years following the initiation of fracking. This effect grows to a statistically significant 4.4% reduction in mortality six years after fracking.

Panel B looks at men. Here, while the point estimates do suggest up to 4% decline in overall mortality six years after fracking, the individual point estimates are not statistically significant. For women, Panel C shows that starting 4 years after the initiation of fracking, there is a statistically significant decline in overall mortality, with a 6.5% and 5.2% decline after five and six years, respectively.

We now examine whether the results are robust to an alternative measure of mortality, specifically the age-adjusted mortality rate, discussed in Section *III.C*. Column (1) of Appendix Table B.6 shows that the overall age-adjusted mortality rate fell in top-quartile counties relative to their shale play counterparts by 8.9 deaths per 100,000 people, which is a 1% decline in terms of the sample period mean. This effect remains largely the same when excluding Montana and North Dakota from the sample in Column (2). Columns (3) and (4) show that the coefficients for men are negative and of similar magnitude to the combined death rate, and the remaining coefficients show larger in magnitude declines for women, in line with the IHS specification. Appendix Figure A.2 then shows the event study estimates from (2) using the age-adjusted mortality rate, and Figure A.3 alternatively uses crude death rates

[28] These figures continue to drop North Dakota and Montana given the unique nature of the Bakken Shale play, however, as in Table 2 the estimates are largely the same with these states included.

per 100k with the current population as the denominator. Both show similar results as our main specification. We next estimate a nonlinear Poisson model of (2) in Figure A.4, which further reaffirms that the absence of pre-trends and decline in mortality from the main results are robust to different functional forms, lending support to the parallel trends assumption (Wooldridge, 2023). Finally, A.8 shows that our findings are robust to whether we include or exclude the Bakken play as well as to running unweighted specifications.

While we do not explicitly instrument overall earnings or employment, we can consider what our estimates imply about the elasticity of mortality with respect to the observed change in either variable. However, we caution that these comparisons may be misleading because changes in both income and employment are occurring at the same time, so simply scaling our mortality results by the magnitude of one of these changes does not consider all the pecuniary and non-pecuniary changes as a result of a labor demand shock at once. If we take the 2.4% decline in overall working-age mortality from Column (2) of Table 2 and the 2.4% increase in overall wages from Column (4) of Table B.4, our estimates suggest that a 1% increase in wages leads to a unit-elastic 1% decline in overall mortality.

We now further break down the age ranges to examine which groups specifically may be experiencing lower mortality. We follow Stevens et al. (2015) and examine mortality by ages under 25, 25-44, 45-64, and 65 and older to create broadly defined aged groups corresponding to different parts of an individual's working life. These age ranges also fall along common mortality delineations that offer interpretative value. For example, the literature on health insurance and mortality often focuses on the middle age range of 45-64 since they are particularly susceptible to higher mortality from lack of insurance coverage (e.g. Goldin et al., 2021). Further, cardiovascular mortality is particularly sharp among those 45 and older (Benjamin et al., 2017), whereas drug overdoses have risen sharply for 25-44 year old men (CDC, 2023) and suicides among youth under 25 (Marcotte and Hansen, 2024). Figures A.5, A.6, and A.7 plot estimates from (2) using these age ranges for all, male, and female respectively. These show that mortality declines are concentrated among working age populations. While there is some movement among certain age groups, such as 25-44 year old men, the figures more strongly show that the overall mortality impacts are driven by those aged 45-64. There is a

2.4% and 3.3% decline in mortality for 45-64 year old men and women, respectively.[29] These suggest the mortality reductions may be driven by the types of deaths found to decrease in response to increased access to medical care. In the next section, we examine this directly.

V.C Heterogeneity by Cause of Death

Internal causes death, such as circulatory and respiratory illnesses, have been found to increase from job displacement, likely due to stress and lack of health insurance/health care utilization to manage chronic conditions (Browning and Heinesen, 2012; Schaller and Stevens, 2015), whereas external causes such homicides, traffic accidents, and deaths of despair may have a different data generating process. Here, we consider whether the declines in mortality are driven by internal causes of mortality, such as cardiovascular mortality, or in external causes, such as suicides and homicides. Panels A and B of Figure 4 present estimates of equation (2) for the IHS of internal and external causes of death, respectively, with the DD estimates from equation (1) presented above. The decline in mortality is driven by declines in internal causes of mortality. Top quartile counties experience a 3% decline in internal causes of death relative to other counties. Figure A.9 breaks down these deaths by gender, again confirming that the main mortality effects are driven by internal causes of death, with a 2.4% decline and a 3.2% decline for men and women, respectively.[30] These findings are similar when using the mortality rates per 100k contemporaneous population (Figure A.10) or nonlinear Poisson regressions (Figures A.13 and A.14).

Next, we look directly at more granular causes of death, focusing on the types of mortality with a stronger ex-ante reason to expect changes from the previous literature. Starting with internal causes of death, we separately examine cardiovascular and non-cardiovascular internal mortality (e.g. Cancer, Kidney Disease, etc.). Figure 5 plots event study estimates using the IHS of these two types of deaths as the dependent variable. There is a 1.9% reduction in non-cardiovascular type internal mortality (significant at the 10% level), however there is a clearer and stronger reduction in cardiovascular mortality. The difference in differences

[29] Note, Denham et al. (2021) finds an increase in hospitalizations for those 65-99 (particularly for women) with additional fracking production. Our null findings (but negative coefficients) here are likely due to both treatment and control counties experiencing some degree of fracking, exposing this susceptible age group to the same pollutants and hence not influencing our results.

[30] As earlier, performing a Wald test across equations, we fail to reject that the effects are the same across genders.

estimate shows a mortality decline of ((exp(-.052)-1)×100 =) 5.1% in top quartile counties, and the event study plots show reductions reaching near 10% in the years following the initiation of fracking.

Figure 6 then looks at external causes of death, specifically the "deaths of despair" identified by Case and Deaton (2015). We note however that the precision and noisiness in the event studies for drug overdoses in the pre-period (Panel A) make it difficult to make strong claims about this type of mortality. However the event study estimates do support no reduction in overdoses. On the other hand, we can more clearly see an absence of any mortality response for both suicides (Panel B) and alcohol-related mortality (Panel C).

One potential issue is the large number of zero observations inherent to these external causes of mortality. For instance, 38% of county-year observations for drug overdoses are 0 in our estimating sample, raising concern with the inverse hyperbolic sine transformation over interpreting these as semi-elasticities. In light of this, we, as before, also re-estimate these event studies for each death of despair using both crude death rates as the dependent variable (Figure A.12) as well as Poisson regressions (Figure A.16). Both show similar patterns. Overall, while Pierce and Schott (2020) find that reductions in labor demand lead to increased death of despair, we ultimately do not find robust evidence to conclude that even relatively sizeable and sustained *increases* in earnings and employment reduce these causes of mortality. On the other hand, the specifications using crude death rates (Figure A.11) and Poisson regressions (Figure A.15) further support our findings of internal, and specifically cardiovascular, mortality driving the results.

Finally, we explore other more specific causes of death. We interpret this exercise as exploratory and suggestive, as increasing the number of outcomes raises concerns over multiple hypothesis testing. Appendix Figure A.17 looks at specific types of death within each category for men and women separately, and Appendix Figure A.18 repeats this exercise using age-adjusted death rates. Each coefficient is estimated from separate regressions, with the bands showing both 90 and 95% confidence intervals. Panel A looks at internal causes. While most coefficients are negatively signed, there are statistically significant declines in cardiovascular related deaths, infection related deaths (tuberculosis, whooping cough, etc.) and

kidney/urethra related deaths (renal failure, kidney infections, etc) for men. For women, there are large in magnitude but imprecisely estimated declines in other internal causes of death, such as respiratory and nutrition-based deaths. Panel B examines external causes of death. Overall, we find no statistically significant change in external causes of death, with the exception of traffic accidents (Figure A.18). This is consistent with Moore and Evans (2012), who find that traffic accidents are pro-cyclical. Additionally, transportation jobs are an important driver of the employment growth in response to the fracking boom (Bartik et al., 2019; Feyrer et al., 2017); the operations of just a single well can involve hundreds of commercial truck trips (Goodman et al., 2016) to haul the water and particulate matter needed for hydraulic fracturing.[31]

V.D Additional Results and Mechanisms

Our findings suggest symmetries in the relationship between long-term job loss and long-term job gains in terms of internal causes of deaths, while external causes of deaths that may be exacerbated by long-term declines in economic conditions (e.g. Pierce and Schott, 2020) may not be easily reversed. In this section, we examine potential mechanisms behind these effects.

*V.D.*1 Mechanisms: Health Insurance Results

Why do we observe reductions in internal causes of death? While greater income has been closely linked to life expectancy in the US (Chetty et al., 2016), fracking boom counties experienced increases in employment in addition to changes in income. While it is challenging to measure the non-pecuniary benefits of employment such as reduced stress that have been linked to employment opportunities (Marcus, 2013), we can look at one relevant mechanism: increased health insurance coverage in the wake of the boom.[32] Wherry and Miller (2016)

[31] Blair et al. (2018) and Graham et al. (2015) find positive associations of traffic accidents with shale drilling activity. Further, several law firms in Texas (https://www.daxgarzalaw.com/blog/fracking-and-oilfield-trucking-dangers/) and Pennsylvania (https://www.rosenbauminjuryfirm.com/practice-areas/fracking-accidents-damages/fracking-related-truck-and-transportation-accidents/) even specialize in fracking related vehicle accidents. We explore this further in Appendix Table B.10 using data on the number of accidents by vehicle type from the Fatality Analysis Reporting System (FARS). The outcome for each column is the IHS of the number of accidents. These support increases in truck traffic accidents, however the results are not robust to excluding the Bakken play.

[32] Bartik et al. (2019) find, using the same source of variation as we do, that local government's increased welfare and hospital expenditures by approximately 24% after the boom. Although this result was not statistically significant, it suggests that changes

finds substantial increases in high cholesterol diagnosis following Medicaid expansion, and cardiovascular drugs are known to reduce mortality within months of treatment (Aronow et al., 2001; Cannon et al., 2004).[33] Likewise, Medicaid expansion has been linked to reduced cardiovascular mortality (Khatana et al., 2019), increased access to vaccinations and antibiotics that can reduce death from infectious diseases (Lu et al., 2015), and lower indices of kidney failure among non-elderly adults (Thorsness et al., 2021).

We explore the possibility of increased insurance coverage by using data from the Small Area Health Insurance Estimates (SAHIE) Program. The SAHIE is the only source for single-year estimates of health insurance coverage status for all counties in the US, and we use coverage data from 2008-2020. The SAHIE uses data from the American Community Survey (ACS) on whether a person is currently covered by health insurance or health coverage plans to form model-based estimates of coverage.[34] Coverage is estimated based on the proportion of a demographic-group within a specific income category and the proportion insured within that income category. While imperfect, these estimates can provide some evidence of changing insurance coverage.

We regress the share of individuals ages 18 to 64 in a county with health insurance on our measure of fracking potential in Table 3. We find evidence that health insurance coverage increased by 1.2 percentage points, or a 1.5% increase off the baseline mean, following the fracking boom. Goldin et al. (2021) show that inducing middle-age adults to enroll in health insurance led to moderate to large declines in subsequent mortality. A 1.9% relative increase in coverage led to a 6% reduction in mortality, which is larger than the magnitudes we find here. Sommers (2017) finds that each percentage point increase in insurance led to a reduction of 3-4 deaths per 100k, which is smaller than our reductions of 16 deaths per 100k (Figure A.3). Although we refrain from conducting an IV analysis due to exclusion restriction concerns, this crude comparison suggests the magnitude of our mortality results fall in between previous estimates of mortality given the insurance coverage increase we observe, suggesting that this is a plausible mechanism.

in public health investments may also be a contributing factor to the observed mortality declines.

[33] Increased income, in addition to expansions in health insurance through increased employment, could also lead to increased access to these treatments. In other words, gaining employment could increase access to market-based health care inputs in a health production function as in Grossman (1972).

[34] Although estimates are available for 2005-2007 as well, these prior years use Current Population Survey data with different insurance definitions, and so the results are not comparable across time periods.

*V.D.*2 Migration

All baseline regressions controlled for the contemporaneous population and we have demonstrated that our results are robust to dropping the Bakken shale play, which experienced high levels of in migration. We now test for migratory responses more directly by estimating (1) with the log number of men and women as the dependent variables, using the same age ranges as before (under 25, 25-44, 45-64, and 65 onward). Table B.2 shows these results of these regressions. We find statistically insignificant and modestly sized coefficients, however the point estimates are positive for the working age groups[35] Table B.3 then looks at the shares of the populations by gender that belong to each age group as the dependent variable. Again, while positively signed for the working age groups, they are statistically and economically small. As a final test, we revisit our mortality results, but control for changes in the age composition of the population. Figures A.19-A.22 re-estimate Figures 3 - 6, controlling for the contemporaneous shares of the relevant population (male or female) used in Table B.3 as independent variables. Overall, the coefficients and patterns of mortality declines are consistent as before, with mortality reductions concentrated in internal (cardiovascular) mortality, with little movements in external mortality and deaths of despair specifically.[36]

*V.D.*3 Direct Effects of Fracking

Our empirical strategy compares counties within the same shale play, meaning both treatment and control counties experience increases in fracking production (as confirmed in Figure 1). Spillovers from more productive top quartile countries into their adjacent control counties may potentially attenuate the impacts of economic opportunity driven by the fracking boom. However, our empirical strategy also offers the benefit of minimizing any direct health impacts that may result due to the nature of fracking production itself, such as those that may arise from water or air pollution. For example, Hill and Ma (2022) finds that the adverse effects of fracking on water quality, and subsequently infant health, is concentrated with drilling within one kilometer of a well. In that sense, both treatment and control counties would be

[35] Recall for our identification strategy, in or out migration would have to systematically vary between top quartile and other counties within the same shale play to be driving our results.

[36] Bartik et al. (2019) also confirm modest net migration changes, but that the increases are not robust across specifications.

susceptible to such pollutants, potentially biasing against our results as treatment counties experience more drilling. We find no evidence of increased mortality for those 65-99 who may be more susceptible to increased morbidity from fracking induced air pollution (Denham et al., 2021), and also Tables B.8 and B.9 show no changes in infant mortality. In total, these suggest that any direct negative impacts directly from fracking production are not influencing our results.

Further, Bartik et al. (2019) highlight significant imbalances between shale and non-shale counties, and stress even further the difficulty of balancing covariates between shale play counties and others in a propensity-score-matching strategy. We confirmed this issue in our context when considering the mortality related imbalances in Table B.1. Here, we demonstrate this further by performing an exercise modifying our identification strategy to include comparisons with counties that do not lie over a shale play. First, we keep all counties and redefine our treatment as an indicator equal to one for all counties that lie over a shale play. Thus we still refrain from using the endogenous actual level of extraction as our treatment. Second, we replace our play-by-year fixed effects with state-by-year fixed effects to further strengthen this strategy by only comparing shale play counties to other non-shale play counties within the same state.[37] For timing, we define the initiation of fracking for each state as the earliest initiation date among the plays that fall within that state. This difference-in-differences strategy compares the changes in mortality outcomes for shale play counties to that of non-shale play counties within the same state, after the initiation of fracking relative to before.

Figure A.23 shows event study estimates of this alternative strategy for overall, internal, and external mortality, and reports the main difference-in-differences estimate and standard error above. Notably, there are trends in mortality before the initiation of fracking among these groups, with internal mortality *increasing* in shale counties relative to non-shale countries in the years prior. The difference in differences estimation finds a statistically significant *increase* (at the 10% level) in overall and internal mortality by about 1%. This finding may be driven by these differential pre-trends or partly by additional determinants of mortality driven by fracking production itself (e.g., pollution). We do not aim to distinguish this here,

[37] Note, this effectively identifies the estimate only off of counties that reside in a state that overlaps with a shale play.

but this exercise complements Bartik et al. (2019) by stressing the importance of making the correct comparisons when identifying the mortality impacts from the economic opportunity generated by the fracking boom.

VI Conclusion

While a growing body of evidence finds negative mental and physical health consequences of unemployment, we know less about the role that increased earnings and employment play in terms of mortality. This question has become even more policy salient in recent years, as Case and Deaton (2017) have linked declining labor market opportunities to rising suicide, drug-related and alcohol mortality, and the subsequent decline in life expectancy in the US. We show that the positive labor demand shocks driven by the fracking boom led to decreased mortality. While we do not find robust evidence that "deaths of despair" decline in response to these positive labor demand shifts, we do find that treatable, internal causes of death decline. Along with suggestive evidence that health insurance increased, our findings suggest a potential channel behind the positive income and life expectancy gradient (Chetty et al., 2016).

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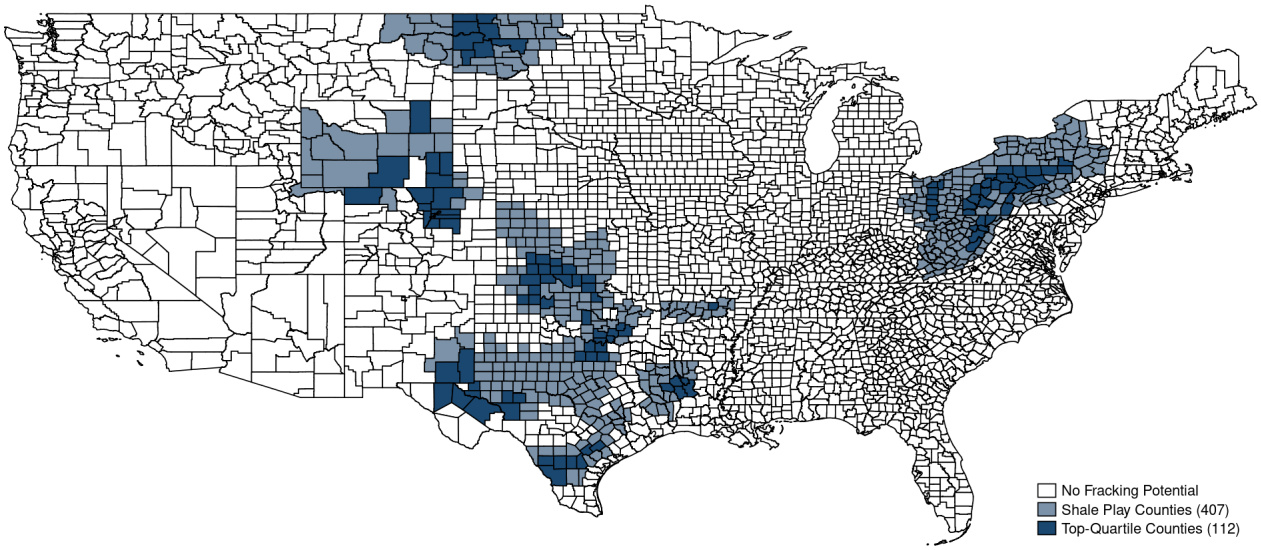
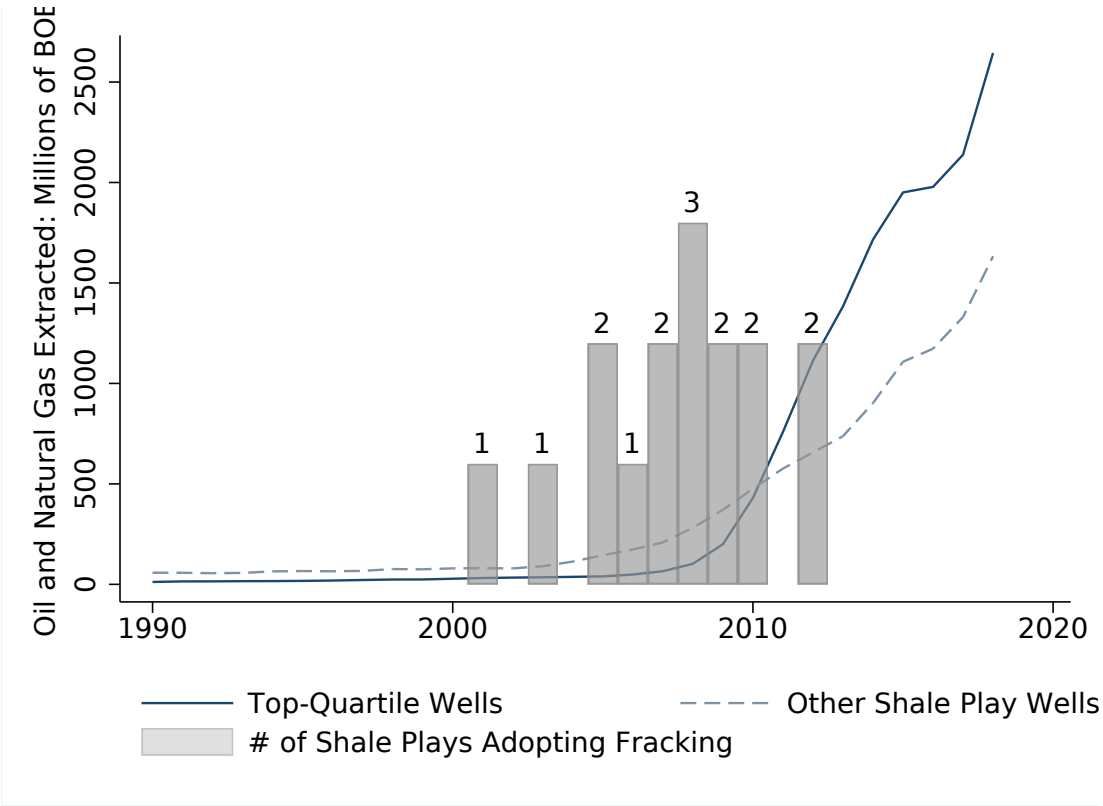
VII Figures

Figure 1: Hydraulic Fracturing Potential and Production - Rystad Prospectivity Index (RPI)

Panel A: Mapping RPI by US Counties



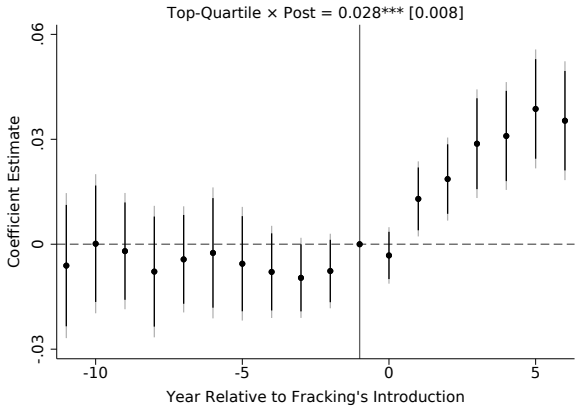
Panel B: County-Level Fracking Production by RPI



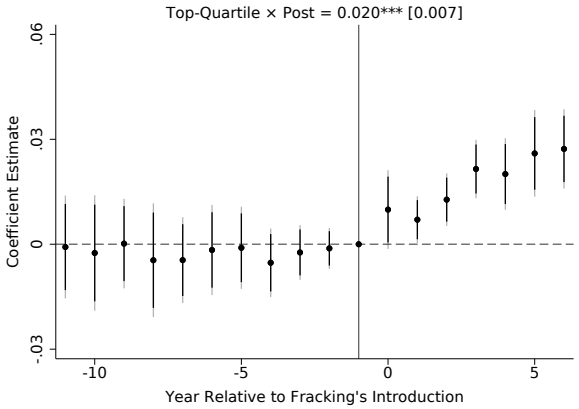
Notes: Panel A plots all US counties from the lower 48 states using 2000 census boundaries. White counties do not intersect with a shale play and are unable to benefit from the fracking boom. Lightly shaded counties (control) intersect with a shale play and are in the bottom three quartiles of the RPI, our measure of fracking potential discussed in Section III.A. Darkly shaded counties (treated) intersect with a shale play and are in the top-quartile of the RPI within a specific shale play. Shale play borders are not shown here for visual clarity. Panel B plots oil and natural gas production measured in millions of barrels of oil equivalent units (BOE) produced by horizontally-drilled wells. These aggregate amounts are calculated from monthly, well-level production data from Enverus. The number of shale plays adopting fracking technology in a specific year (as identified by Bartik et al. (2019), are shown using the shaded gray bars.

Figure 2: Earnings and Employment Effects by Gender

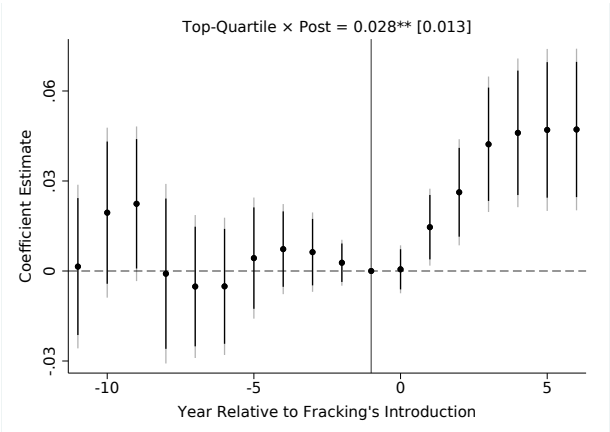
Panel A: Log of Male Earnings



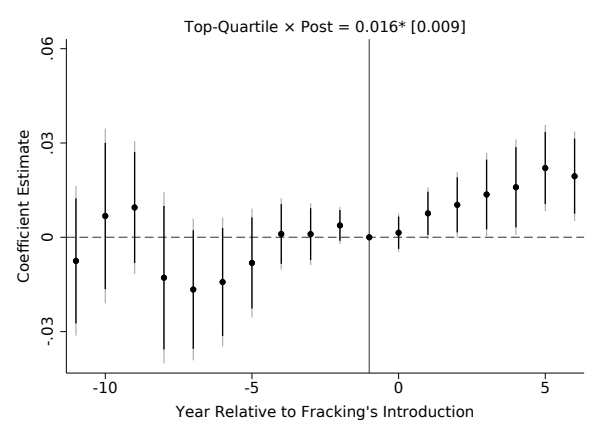
Panel B: Log of Female Earnings



Panel C: Log of Male Employment to Pop. Ratio



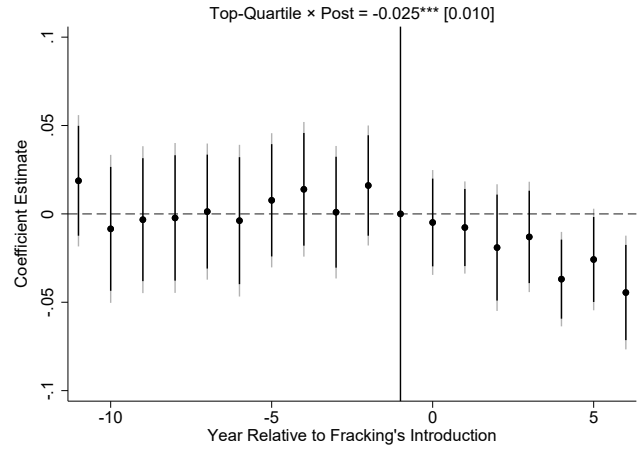
Panel D: Log of Female Employment to Pop. Ratio



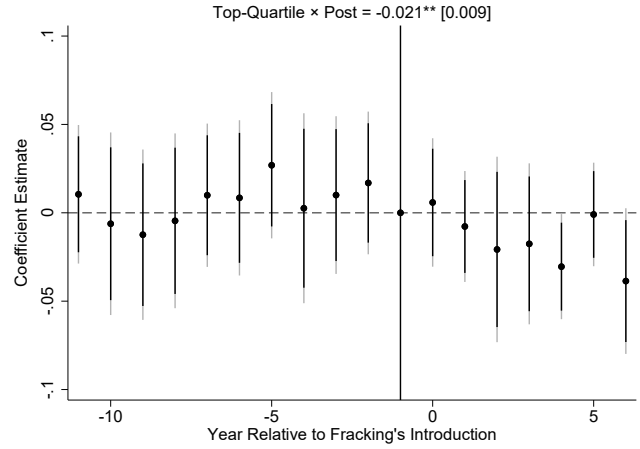
*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. We take earnings measures (adjusted to real 2010 $ amounts) and employment counts from the QWI database. We take population counts from SEER. All values are calculated for 14-99 year old individuals in each county. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Each panel omits North Dakota and Montana from the sample. Standard errors are adjusted for clustering at the county level. The difference-in-differences coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

Figure 3: Overall Mortality by Gender

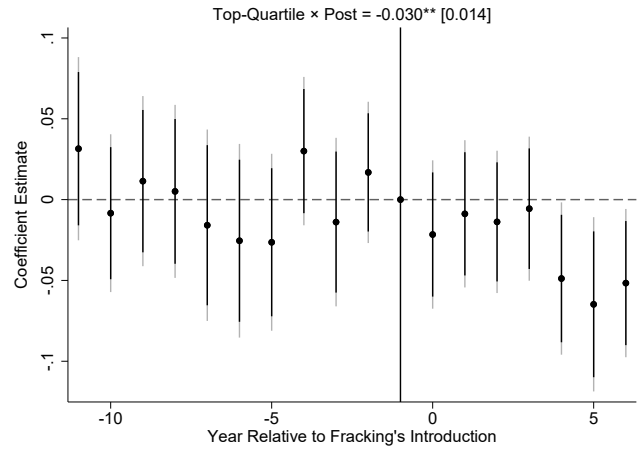
Panel A: Men and Women



Panel B: Men



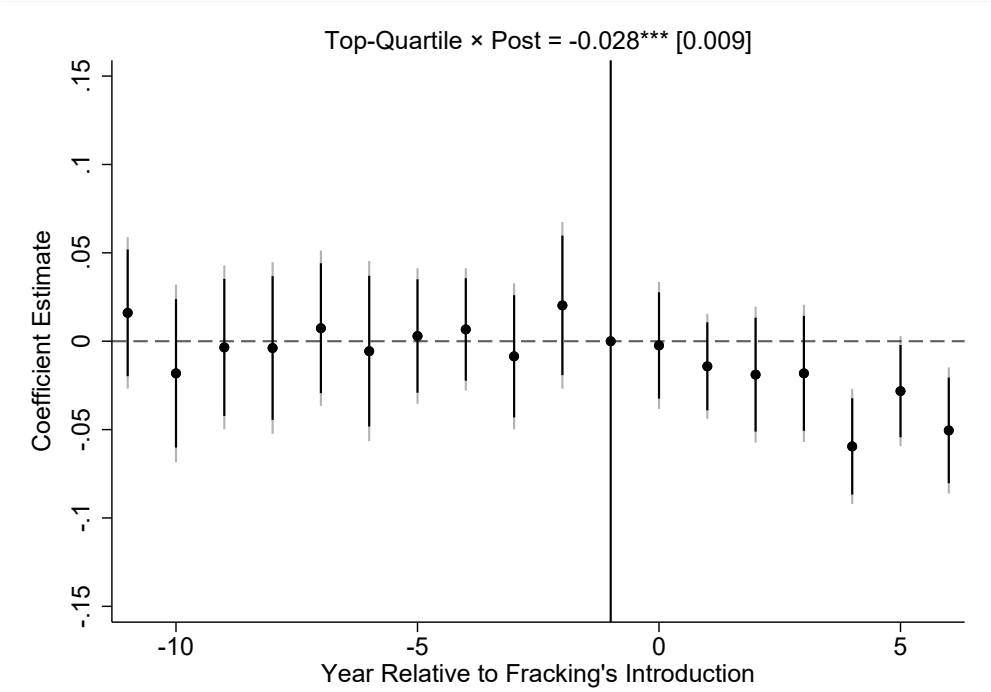
Panel C: Women



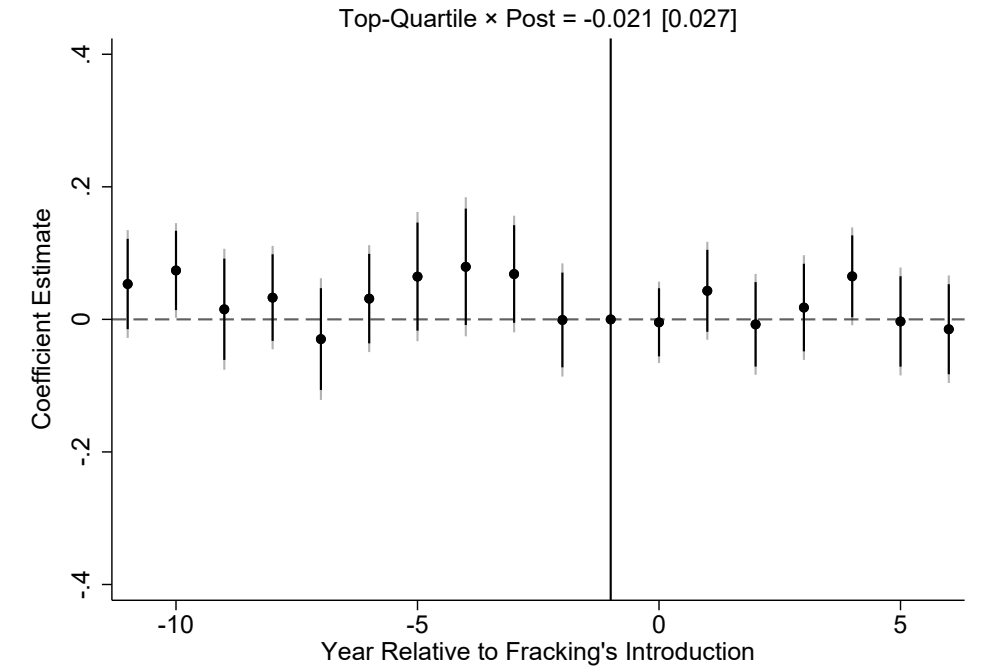
*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the inverse hyperbolic sine of overall mortality, and we control for the (IHS of) contemporaneous population. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Standard errors are adjusted for clustering at the county level. The difference-in-differences coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

Figure 4: Internal vs. External Causes of Death

Panel A: IHS of Internal Causes of Death



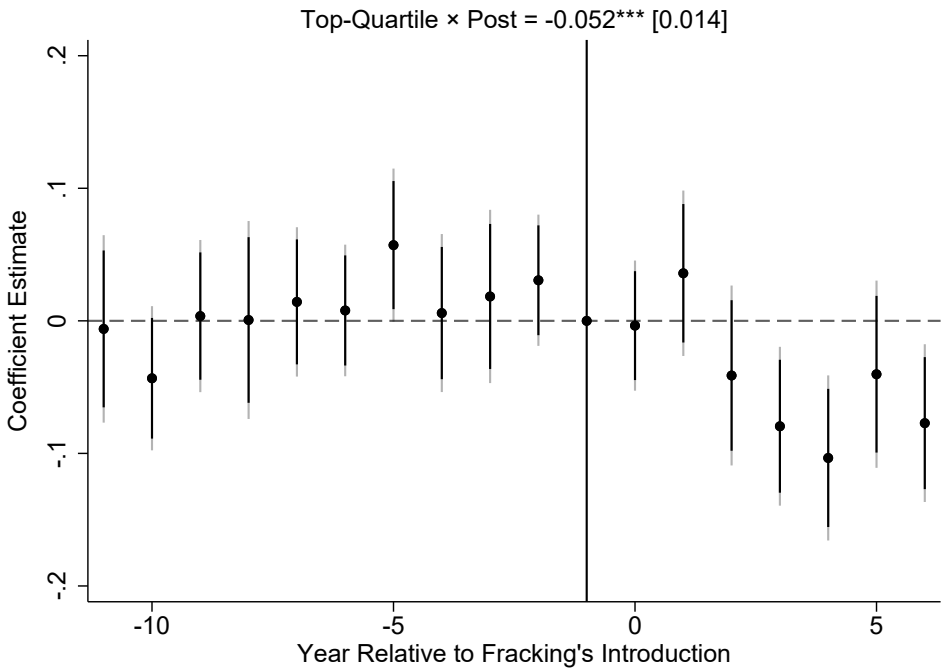
Panel B: IHS of External Causes of Death



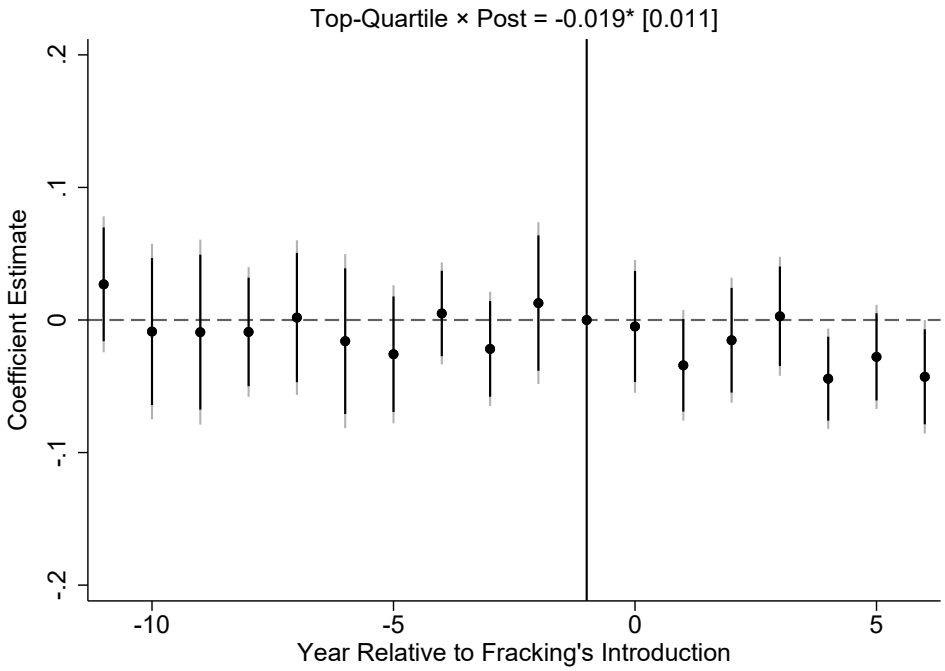
Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the inverse hyperbolic sine of Internal (Panel A) and External (Panel B) mortality, and we control for the (IHS of) contemporaneous population.We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Death categories are taken from Stevens et al. (2015), and represent consistent definitions across ICD-9 and ICD-10 cause of death codes. All regressions were estimated using interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights, and control for contemporaneous population. Standard errors are adjusted for clustering at the county level.

Figure 5: Internal Causes: Cardiovascular vs Other Internal Mortality

Panel A: Cardiovascular



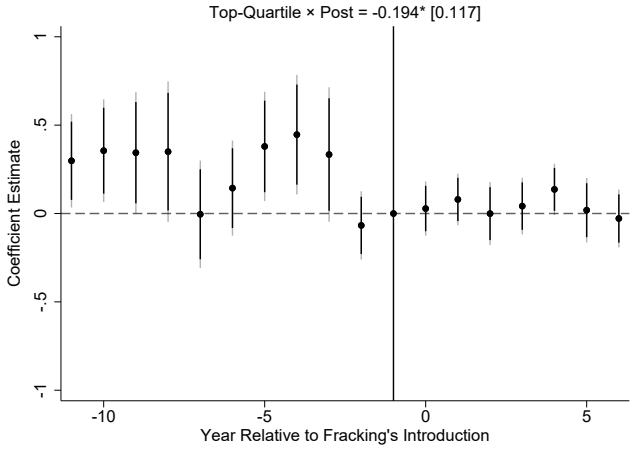
Panel B: Non-Cardiovascular



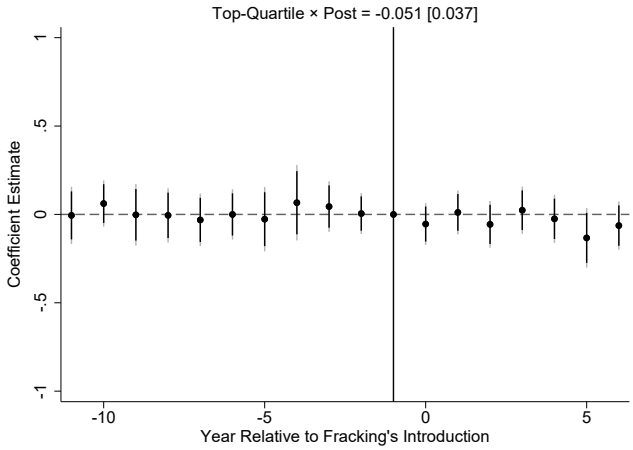
Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the inverse hyperbolic sine of Cardiovascular (Panel A) and Non-Cardiovascular Internal (Panel B) mortality, and we control for the (IHS of) contemporaneous population. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. All regressions were estimated using interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights, and control for contemporaneous population. Standard errors are adjusted for clustering at the county level.

Figure 6: External Causes of Death: Deaths of Despair

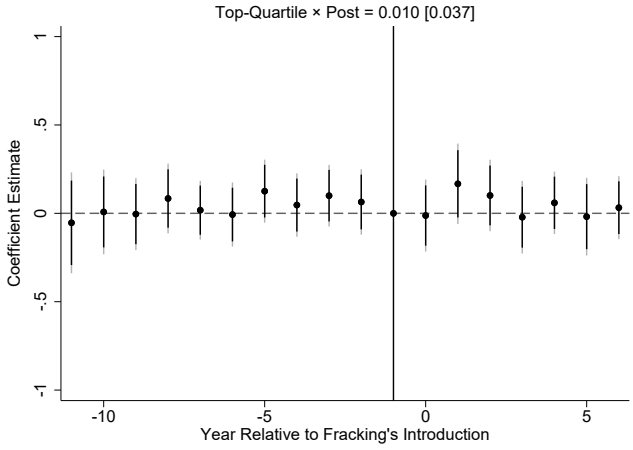
Panel A: Drug Overdoses



Panel B: Suicides



Panel C: Alcohol Related



Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the inverse hyperbolic sine of drug overdose (Panel A), suicide (Panel B), and alcohol-related (Panel C) mortality, and we control for the (IHS of) contemporaneous population. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. All regressions were estimated using interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights, and control for contemporaneous population. Standard errors are adjusted for clustering at the county level.

VIII Tables

Table 1: Summary Statistics - Treatment and Control Comparisons (1990 Variables)

	Top-Quartile County	Other Shale Play County	Within Play Difference
Age-Adjusted Death Rate	906.23	916.07	-2.60
	(146.75)	(124.18)	[15.17]
Median Household Income	30532.81	29815.35	-111.46
	(7878.33)	(6442.70)	[597.67]
% High School Graduates	34.90	34.83	-0.87
	(7.94)	(6.25)	[0.56]
% in Manufacturing	5.22	5.89	0.42
	(3.88)	(4.54)	[0.39]
% Married	60.76	60.16	-0.38
	(5.66)	(5.40)	[0.50]
% Rural	63.74	61.71	-4.48
	(32.11)	(28.68)	[3.01]
% Veterans	14.59	14.66	-0.03
	(2.38)	(2.14)	[0.23]
% White	91.04	90.88	-0.56
	(10.16)	(10.17)	[0.72]
% Foreign Born	2.80	2.33	-0.32
	(3.82)	(2.97)	[0.32]
% w/ a Bachelors Degree	9.57	8.77	-0.18
	(4.55)	(3.54)	[0.34]
Observations	112	407	519

Notes: All variables are measured at the county-level in 1990. Aside from the age-adjusted death rate, all variables are taken from the 1990 Decennial Census. The age-adjusted death rate is calculated using mortality data from the CDC's National Center for Health Statistics, and all the population data come from SEER. Column (3) reports the regression-adjusted difference between top-quartile counties and other counties in the same shale play (only play-specific fixed effects are controlled for), with standard errors in parenthesis.

Table 2: Working-Age Overall Mortality Rates by Gender

	Men and Women		Men		Women	
	(1)	(2)	(3)	(4)	(5)	(6)
Top-Quartile × Post	-0.024**	-0.025***	-0.019**	-0.021**	-0.030**	-0.030**
	[.0099]	[.0096]	[.0096]	[.0093]	[.0138]	[.0137]
Omits ND & MT?	No	Yes	No	Yes	No	Yes
Outcome Mean	151.27	160.94	93.70	99.68	57.56	61.25
Observations	9,342	8,712	9,342	8,712	9,342	8,712

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. All regressions include a time-varying control for the inverse hyperbolic sine of the relevant population group. All columns include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census, and we control for the (IHS of) contemporaneous population. Some columns also omits all observations from North Dakota and Montana, and include 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.*

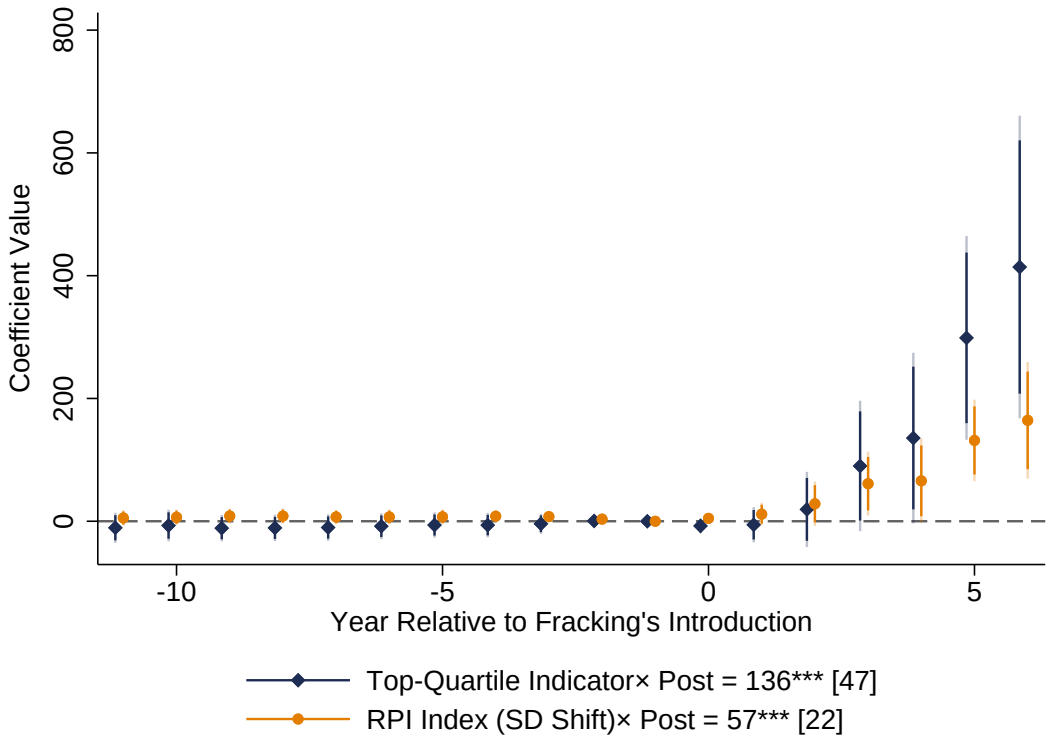
Table 3: Health Insurance Coverage by Gender: Ages 18-64

	men/women	men	women
	(1)	(2)	(3)
Top-Quartile × Post	0.012***	0.009*	0.014***
	[0.004]	[0.005]	[0.004]
1990 Controls?	Yes	Yes	Yes
Outcome Mean	0.81	0.79	0.82
Observations	5,731	5,731	5,731

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. The sample is restricted to years after 2008 due to comparibility of the insurance estimates. We take all insurance estimates from the Small Area Health Insurance Estimates (SAHIE) Program, which are calculated using data from the 2008-2018 ACS. All columns include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Standard errors are adjusted for clustering at the county level.*

A Appendix Figures

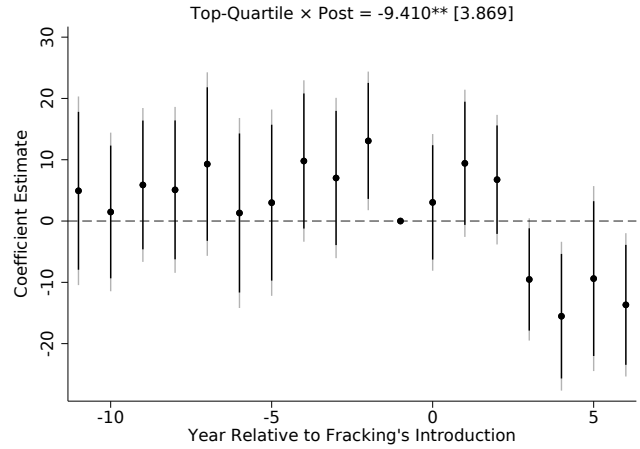
Figure A.1: Horizontal Well Production: Millions of $ of BOE



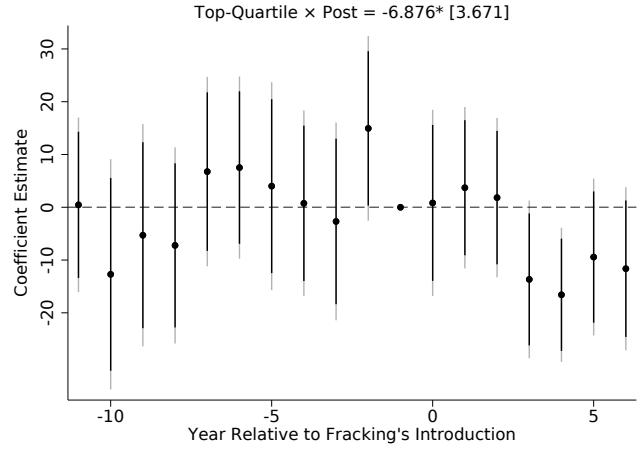
*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. Here, we show coefficients from 2 separate regressions where the coefficient of interest is a different transformation of the RPI. Monthly, well-level production of oil and natural gas data from Enverus, and we aggregate these amounts to the county-level using the latitude and longitude of each well. We use yearly price data from the EIA to calculate the value of fracking production in millions of dollars, transformed into real, 2010 $ using the PCEPI. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Standard errors are adjusted for clustering at the county level. The difference-in-differences coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

Figure A.2: Age-Adjusted Overall Mortality per 100K

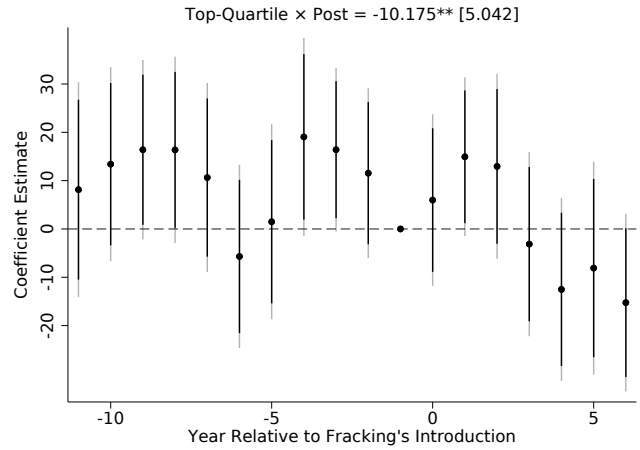
Panel A: Men and Women



Panel B: Men



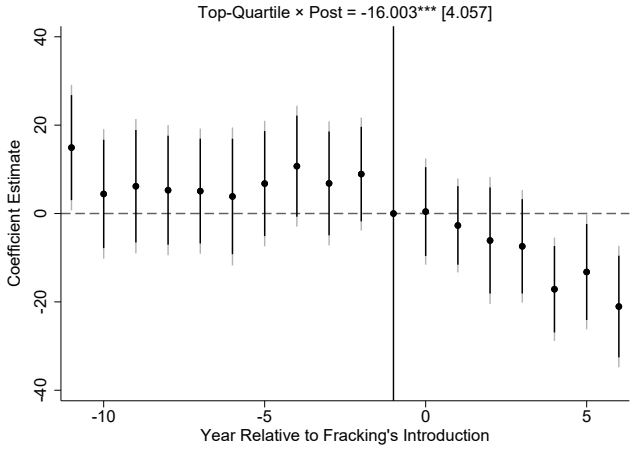
Panel C: Women



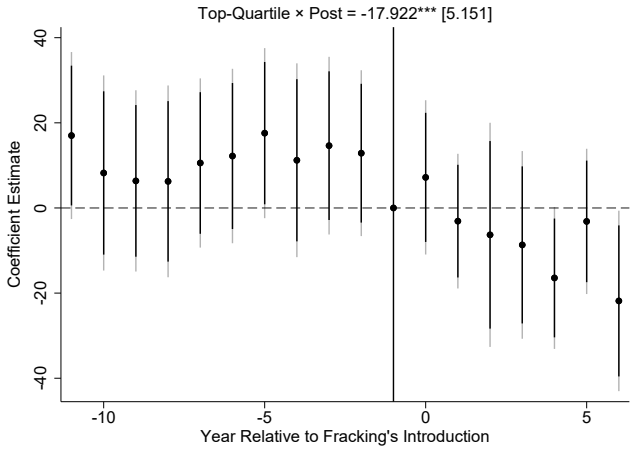
*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. We use the standard method for age-adjustment by taking a weighted average of the crude death rates for different age categories within a county, where the national population shares in those age categories in 2000 are the weights. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Standard errors are adjusted for clustering at the county level. The difference-in-differences coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

Figure A.3: Crude Overall Mortality Rate per 100K

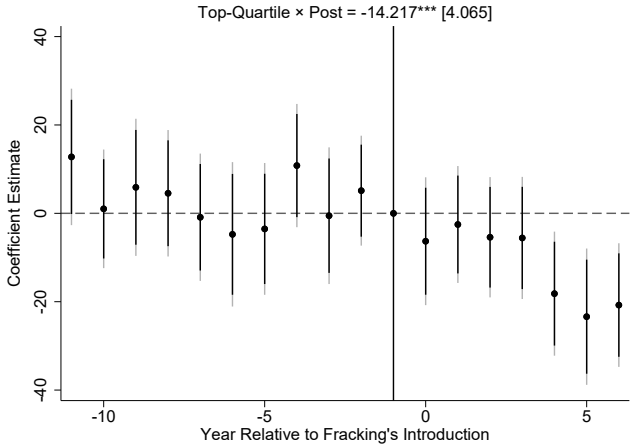
Panel A: Men and Women



Panel B: Men



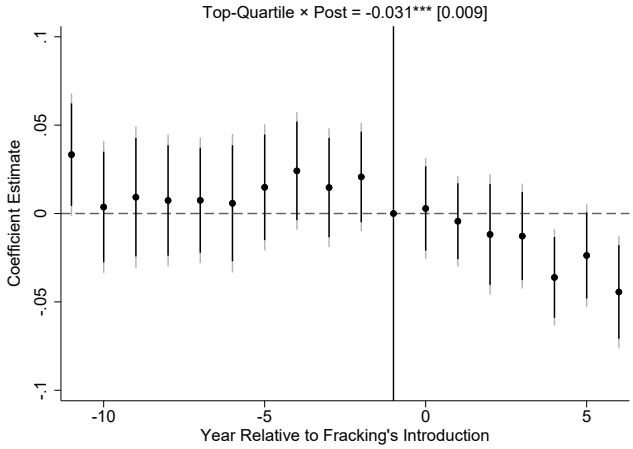
Panel C: Women



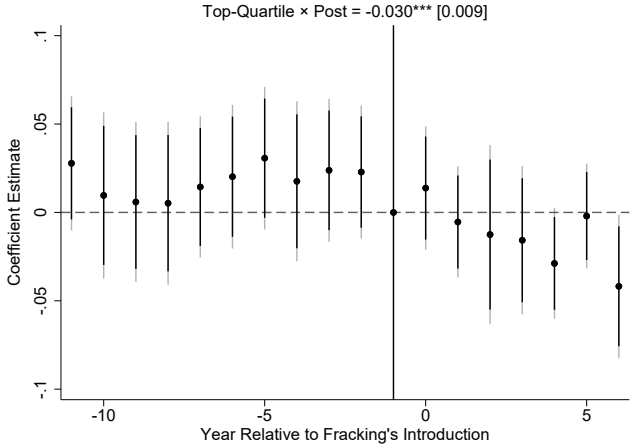
*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. The dependent variable is the death rate per 100k people, using contemporaneous populations. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Standard errors are adjusted for clustering at the county level. The difference-in-differences coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

Figure A.4: Poisson Regressions: Overall Mortality

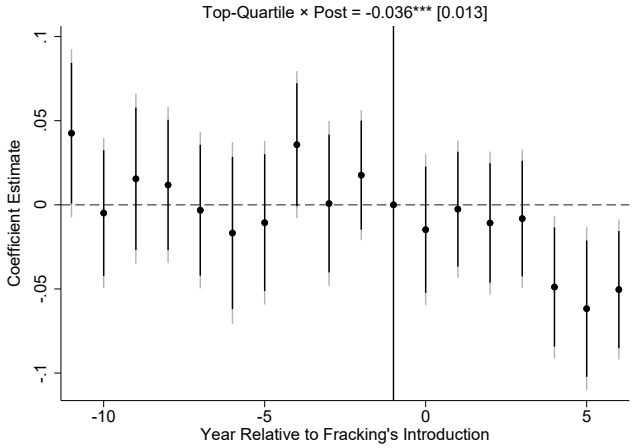
Panel A: Men and Women



Panel B: Men



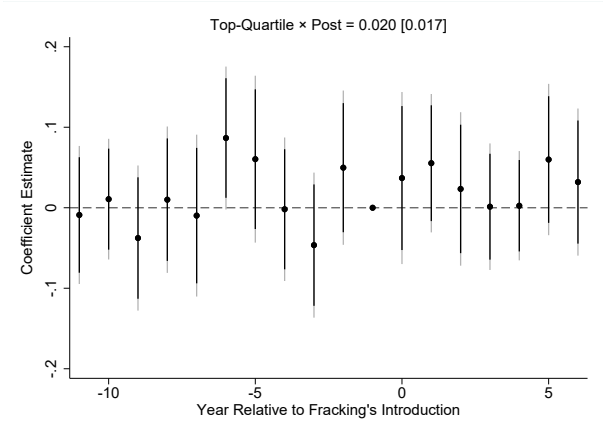
Panel C: Women



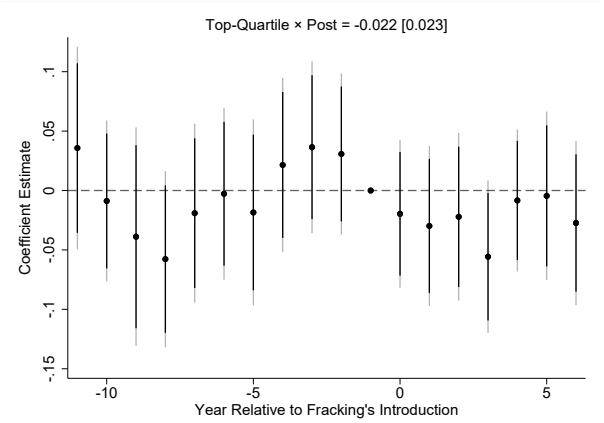
*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from a Poisson regression based off of Equation (2) for the balanced set of event-years, using the relevant gender and age population as the exposure variable. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Standard errors are adjusted for clustering at the county level. The difference-in-differences Poisson coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

Figure A.5: Overall Mortality Effects by Age

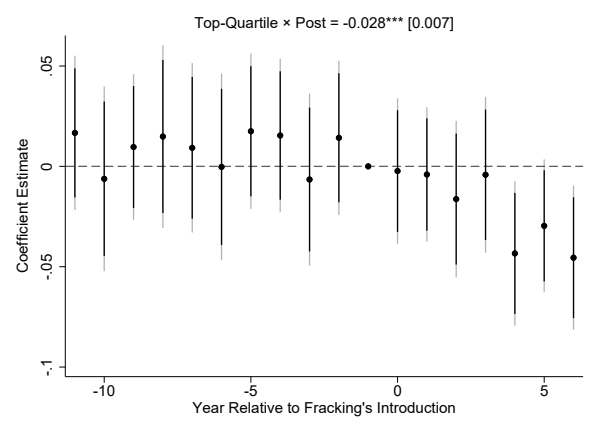
Panel A: IHS of Overall Mortality Ages : Under 25



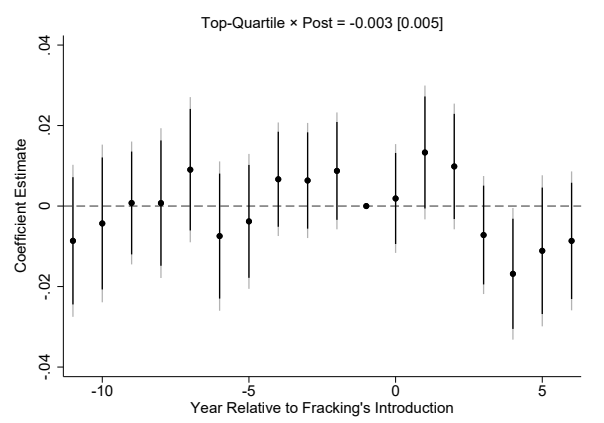
Panel B: IHS of Overall Mortality Ages: 25-44



Panel C: IHS of Overall Mortality Ages: 45-64



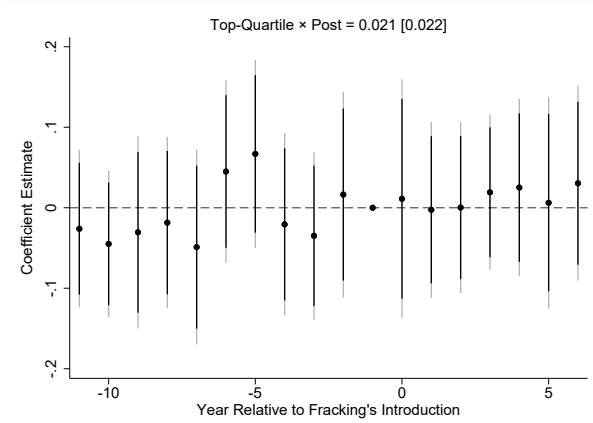
Panel D: IHS of Overall Mortality Ages: 65-99



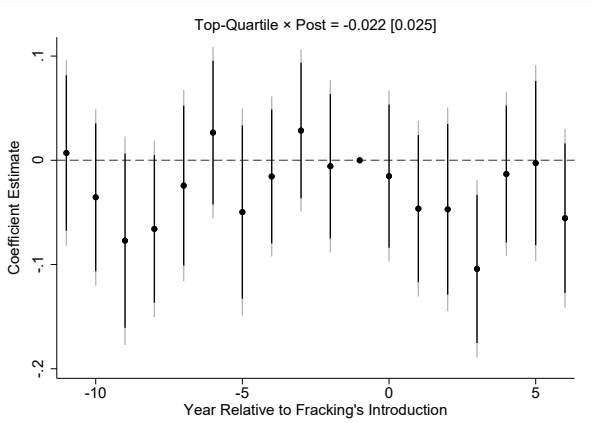
*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the inverse hyperbolic sine of overall mortality, and we control for the (IHS of the) relevant contemporaneous population. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Standard errors are adjusted for clustering at the county level. The difference-in-differences coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

Figure A.6: Overall Mortality Effects by Age: Male

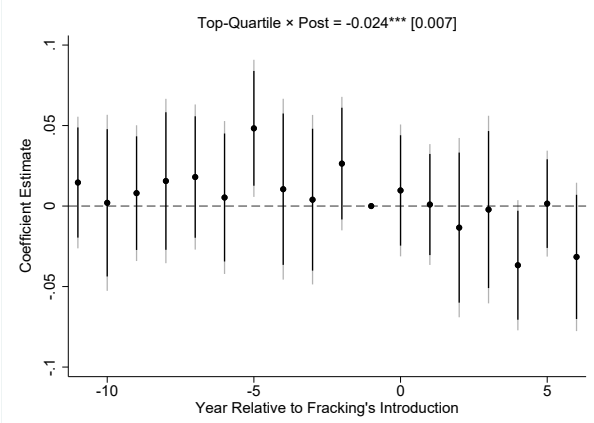
Panel A: IHS of Overall Mortality Ages : Under 25



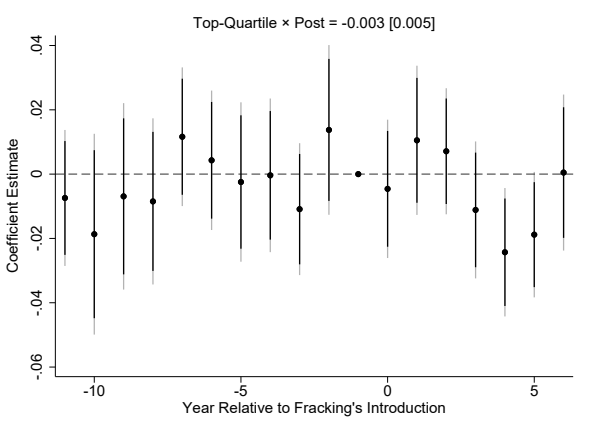
Panel B: IHS of Overall Mortality Ages: 25-44



Panel C: IHS of Overall Mortality Ages: 45-64



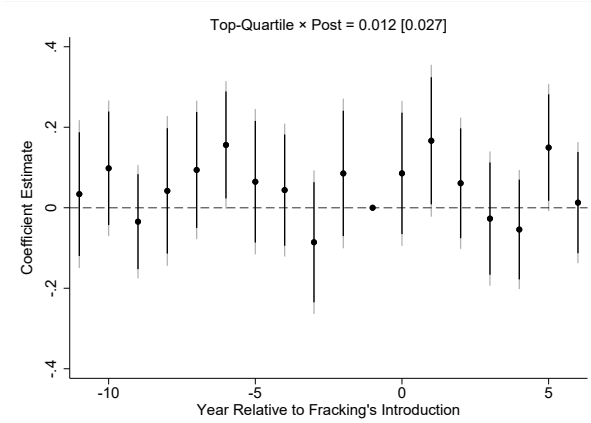
Panel D: IHS of Overall Mortality Ages: 65-99



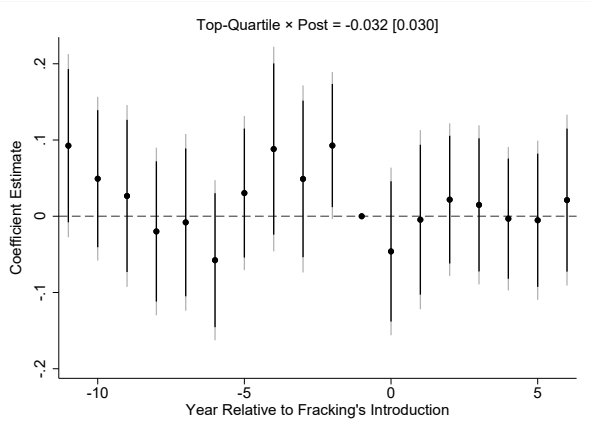
*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the inverse hyperbolic sine of overall mortality, and we control for the (IHS of the) relevant contemporaneous population. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Standard errors are adjusted for clustering at the county level. The difference-in-differences coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

Figure A.7: Overall Mortality Effects by Age: Female

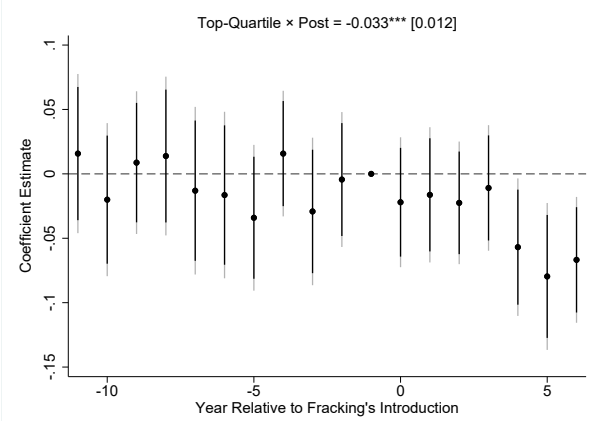
Panel A: IHS of Overall Mortality Ages : under 25



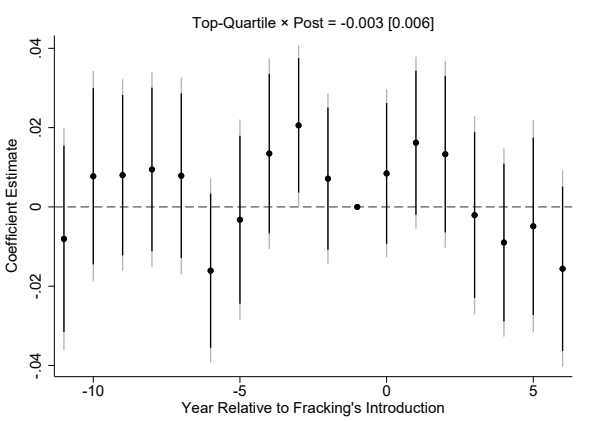
Panel B: IHS of Overall Mortality Ages: 25-44



Panel C: IHS of Overall Mortality Ages: 45-64

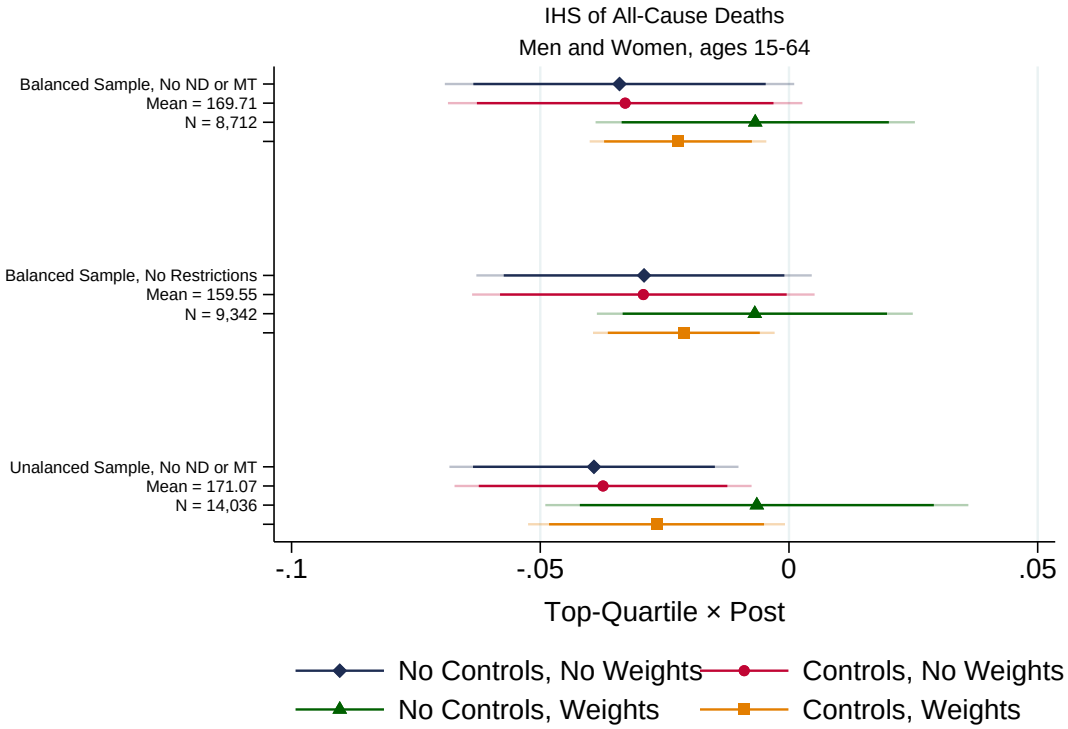


Panel D: IHS of Overall Mortality Ages: 65-99



*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the inverse hyperbolic sine of overall mortality, and we control for the (IHS of the) relevant contemporaneous population. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Standard errors are adjusted for clustering at the county level. The difference-in-differences coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

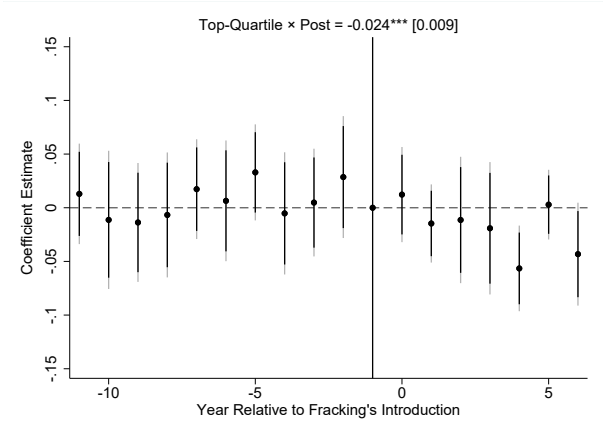
Figure A.8: Men/Women Working-Age Mortality Robustness



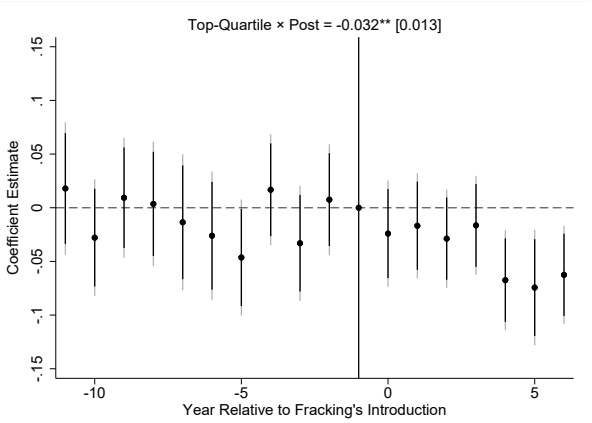
Notes: We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Death categories are taken from Stevens et al. (2015), and represent consistent definitions across ICD-9 and ICD-10 cause of death codes. Each point represents the outcome from a separate regression (Equation (1)), and the dark and lighter shaded bars represent the associated 90% and 95% confidence intervals, respectively. All regressions include 13,746 observations, (except the specification which omits North Dakota and Montana, which has 12,371 observations).

Figure A.9: Internal and External Causes of Death by Gender

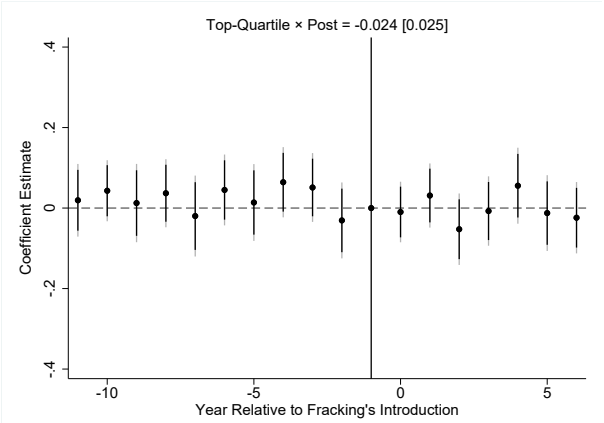
Panel A: IHS of Internal Deaths (Male)



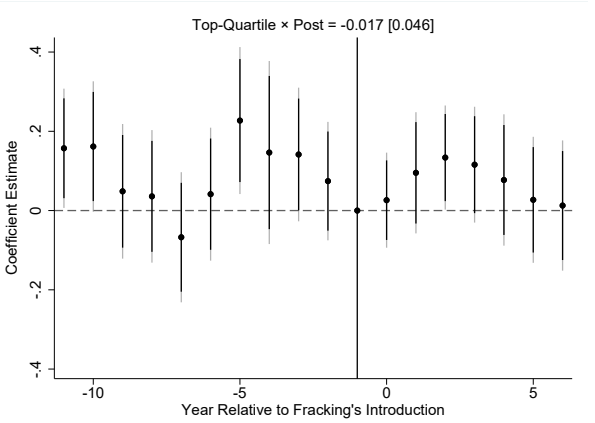
Panel B: IHS of Internal Deaths (Female)



Panel C: IHS of External Deaths (Male)



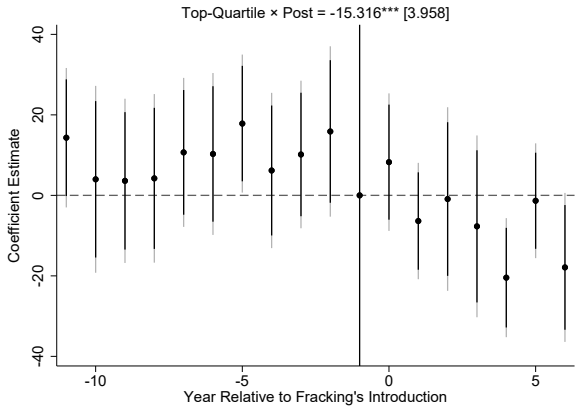
Panel D: IHS of External Deaths (Female)



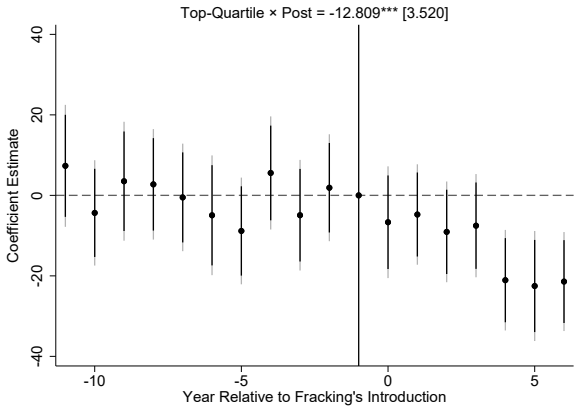
*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Our primary outcome is the inverse hyperbolic sine (IHS) of the number of deaths, where the contemporaneous population of the relevant demographic group is included as a control. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Standard errors are adjusted for clustering at the county level. The difference-in-differences coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

Figure A.10: Crude Death Rates: Internal and External Causes of Death by Gender

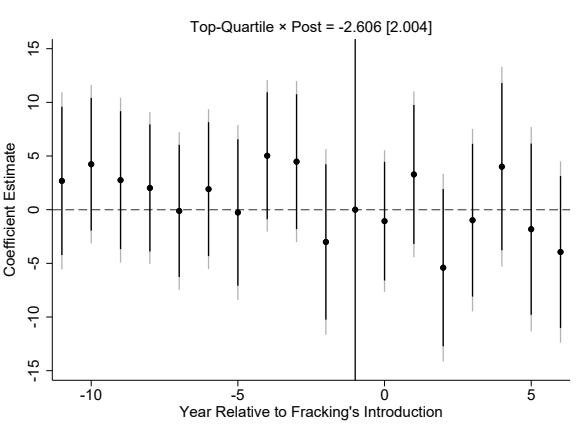
Panel A: Internal Death Rates per 100k: Male



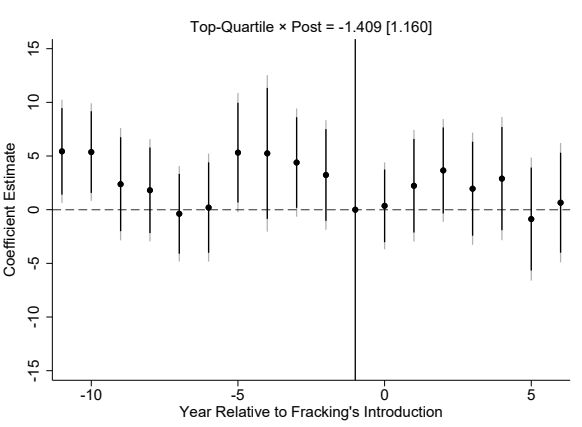
Panel B:Internal Death Rates per 100k: Female



Panel C: External Death Rates per 100k: Male



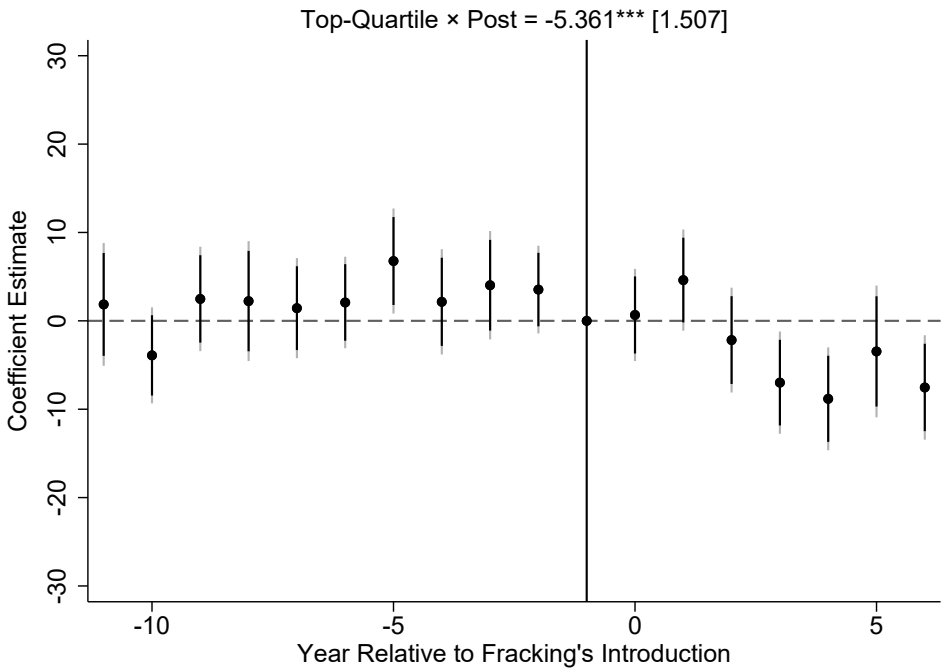
Panel D: External Death Rates per 100k: Female



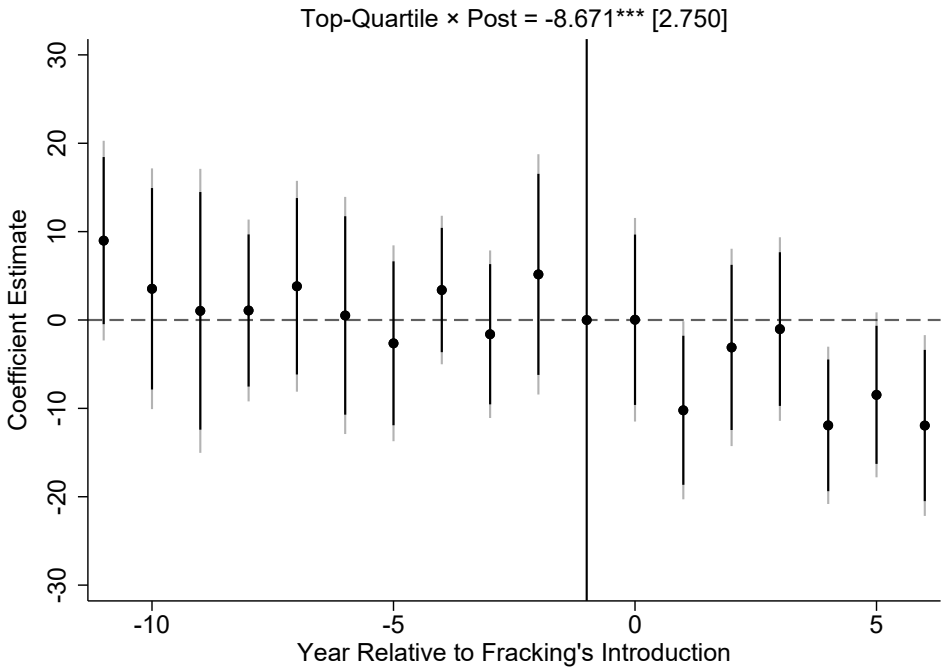
*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Dependent variables are the crude death rate per 100k individuals of working age. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Standard errors are adjusted for clustering at the county level. The difference-in-differences coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

Figure A.11: Crude Death Rates: Cardiovascular vs Other Internal Mortality

Panel A: Cardiovascular



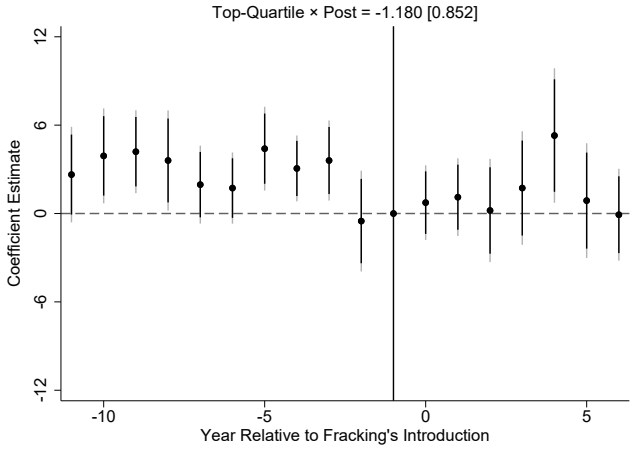
Panel B: Non-Cardiovascular



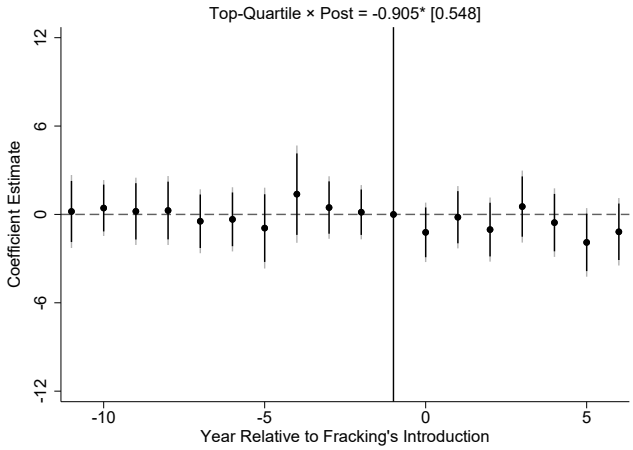
Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the death rate per 100k of Cardiovascular (Panel A) and Non-Cardiovascular Internal (Panel B) mortality, and we control for the (IHS of) contemporaneous population. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. All regressions were estimated using interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights, and control for contemporaneous population. Standard errors are adjusted for clustering at the county level.

Figure A.12: Crude Death Rates: External Causes of Death: Deaths of Despair

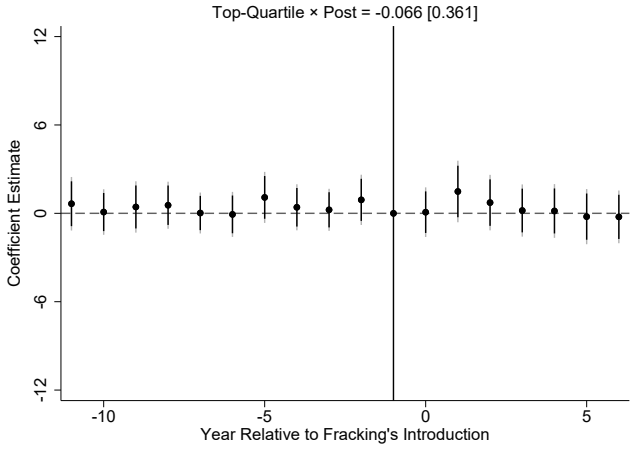
Panel A: Drug Overdoses



Panel B: Suicides



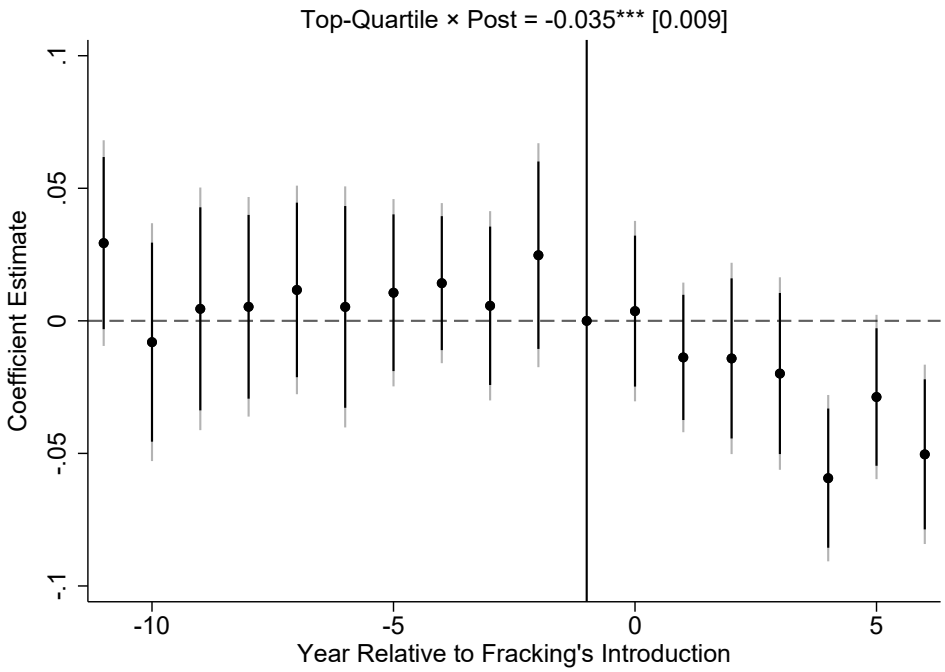
Panel C: Alcohol Related



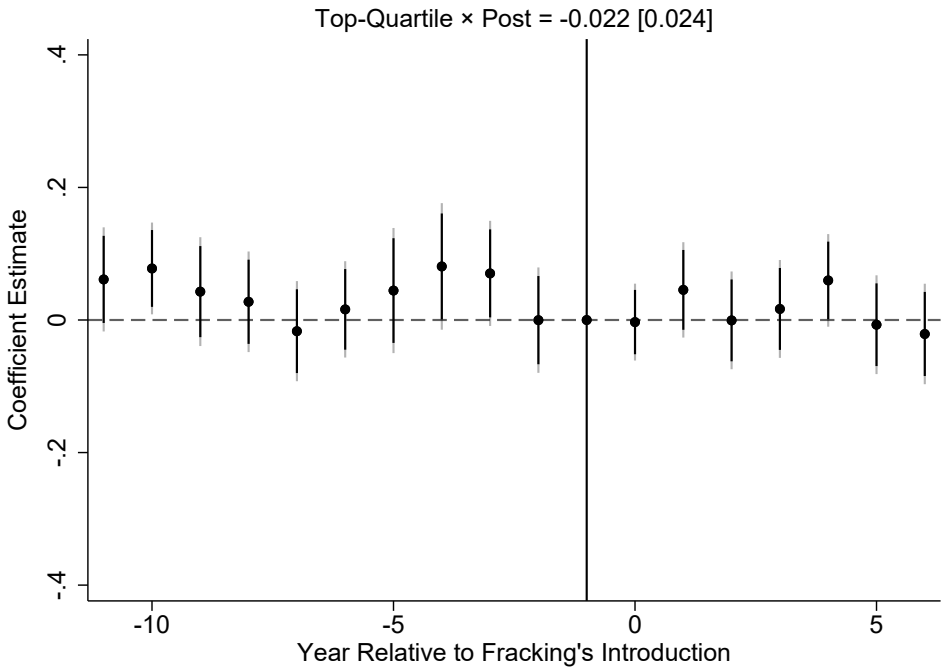
Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the death rate per 100k people of drug overdose (Panel A), suicide (Panel B), and alcohol-related (Panel C) mortality, and we control for the (IHS of) contemporaneous population. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. All regressions were estimated using interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights, and control for contemporaneous population. Standard errors are adjusted for clustering at the county level.

Figure A.13: Poisson Regression: Internal vs. External Causes of Death (Ages 25-64)

Panel A: Poisson Regression: Internal Causes of Death



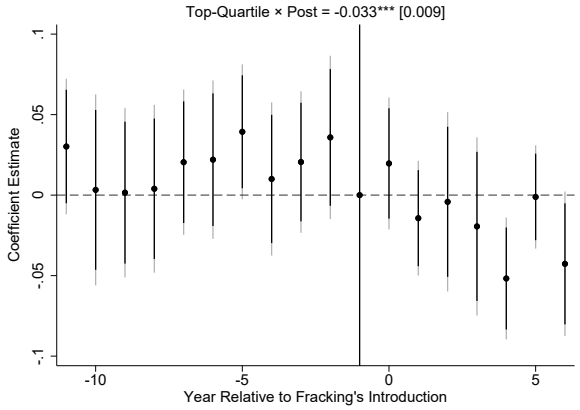
Panel B: Poisson Regression: External Causes of Death



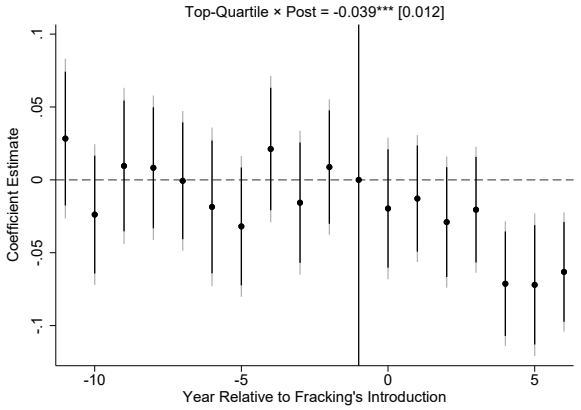
Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from a Poisson specification of Equation (2) for the balanced set of event-years using the relevant age and gender population as the exposure variable. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Death categories are taken from Stevens et al. (2015), and represent consistent definitions across ICD-9 and ICD-10 cause of death codes. All regressions were estimated using interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Standard errors are adjusted for clustering at the county level.

Figure A.14: Poisson Regressions: Internal and External Causes of Death by Gender

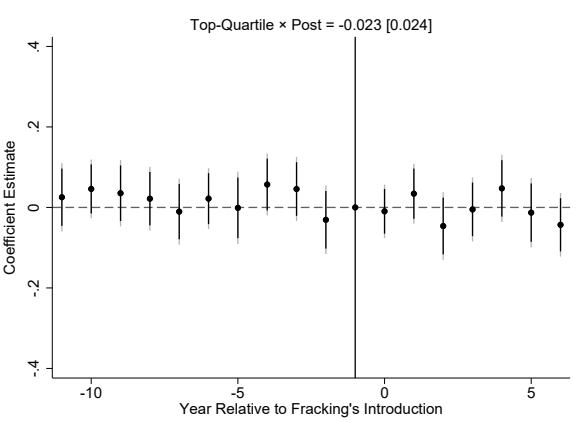
Panel A: Internal Deaths (Male)



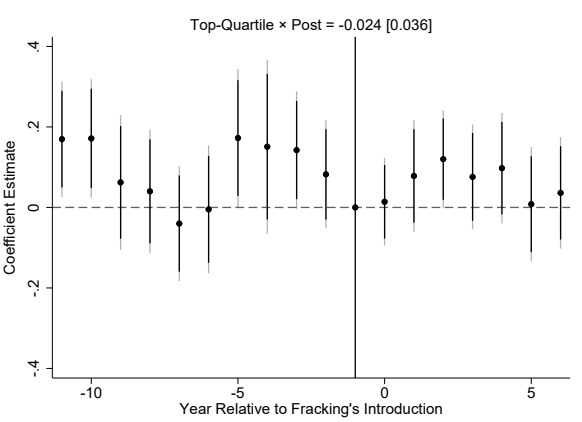
Panel B: Internal Deaths (Female)



Panel C: External Deaths (Male)



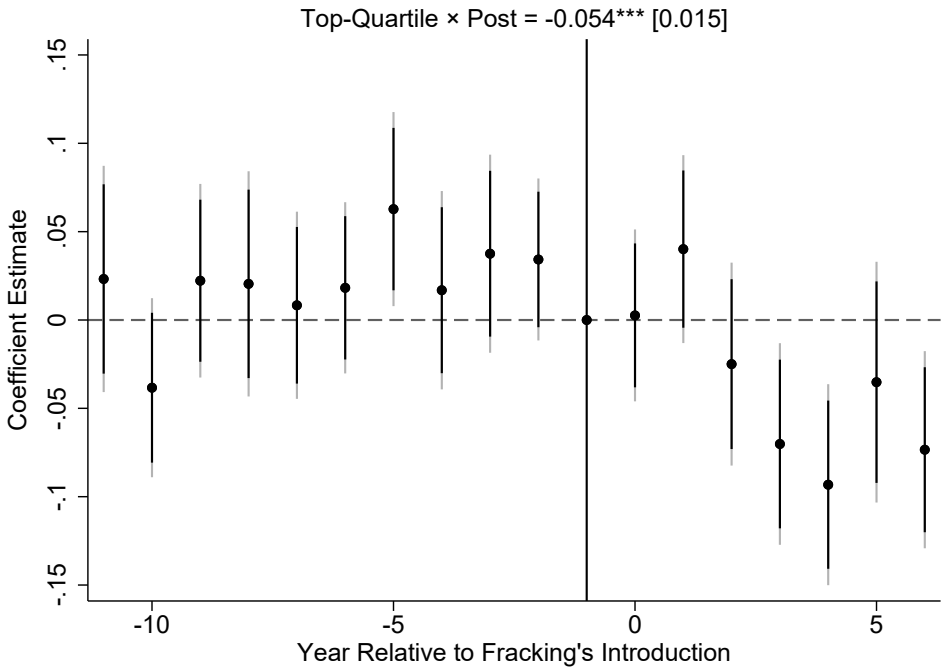
Panel D: External Deaths (Female)



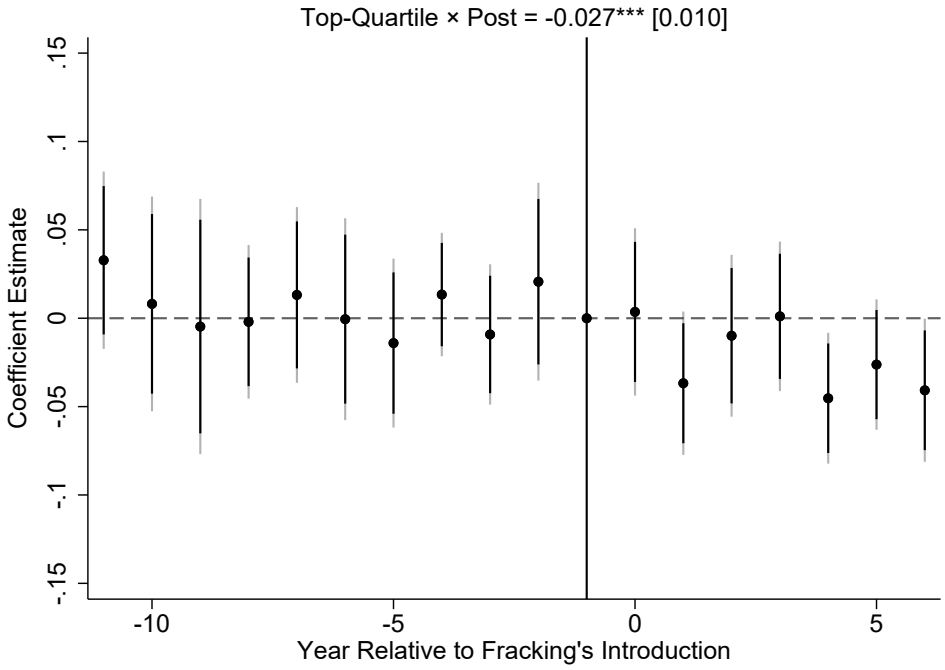
*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from a Poisson specification of Equation (2) for the balanced set of event-years, using the relevant age and gender population as the exposure variable. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. We use the standard method for age-adjustment by taking a weighted average of the crude death rates for different age categories within a county, where the national population shares in those age categories in 2000 are the weights. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Standard errors are adjusted for clustering at the county level. The difference-in-differences coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

Figure A.15: Poisson Regression: Cardiovascular vs. Other Internal Causes of Death (Ages 25-64)

Panel A: Cardiovascular Mortality



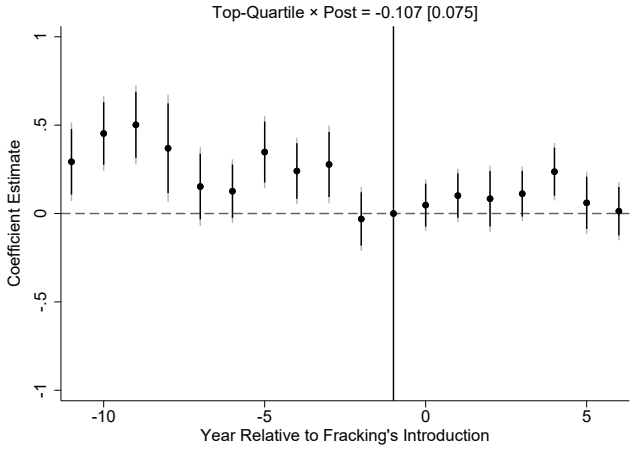
Panel B: Other Internal Mortality



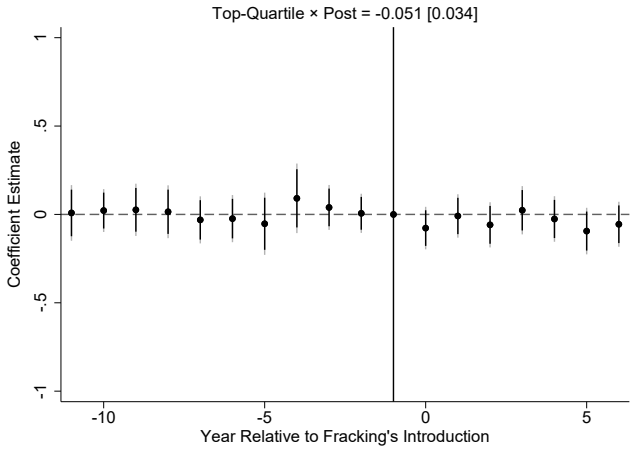
Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from a Poisson specification of Equation (2) for the balanced set of event-years using the relevant age and gender population as the exposure variable. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. All regressions were estimated using interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Standard errors are adjusted for clustering at the county level.

Figure A.16: Poisson Regressions: Deaths of Despair

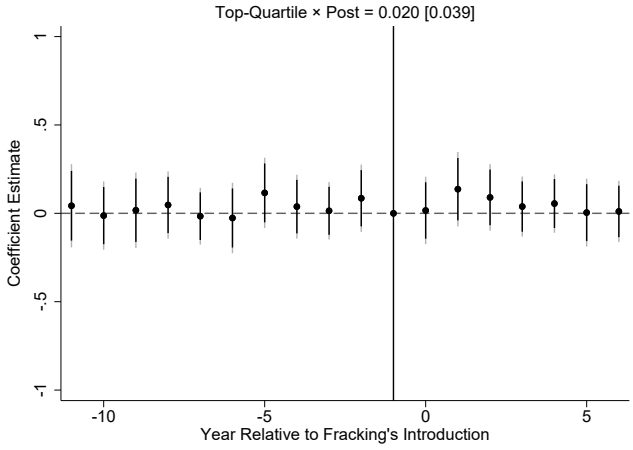
Panel A: Drug Overdoses



Panel B: Suicides



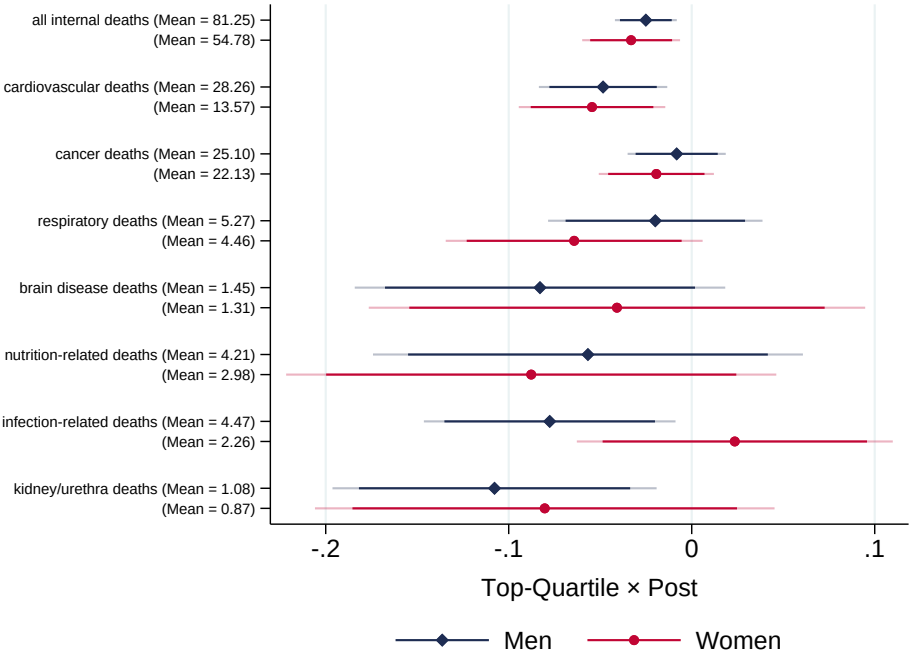
Panel C: Alcohol Related



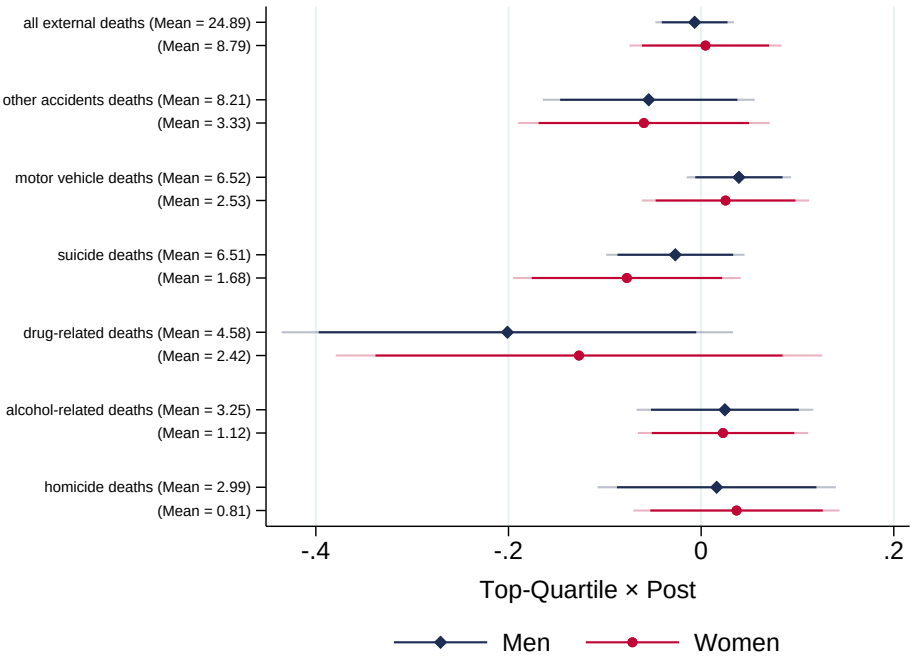
*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from a Poisson regression based off of Equation (2) for the balanced set of event-years, using the relevant gender and age population as the exposure variable. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Standard errors are adjusted for clustering at the county level. The difference-in-differences Poisson coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

Figure A.17: Internal vs. External Causes of Death: Differences by Gender

Panel A: IHS of Internal Deaths (Ages 25-64)



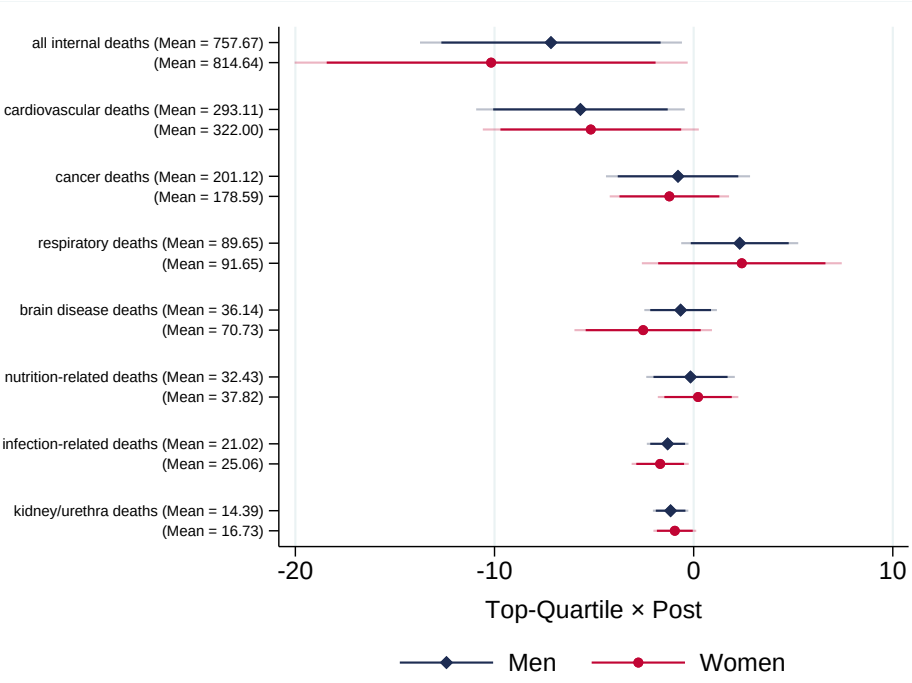
Panel B: IHS of External Deaths (Ages 25-64)



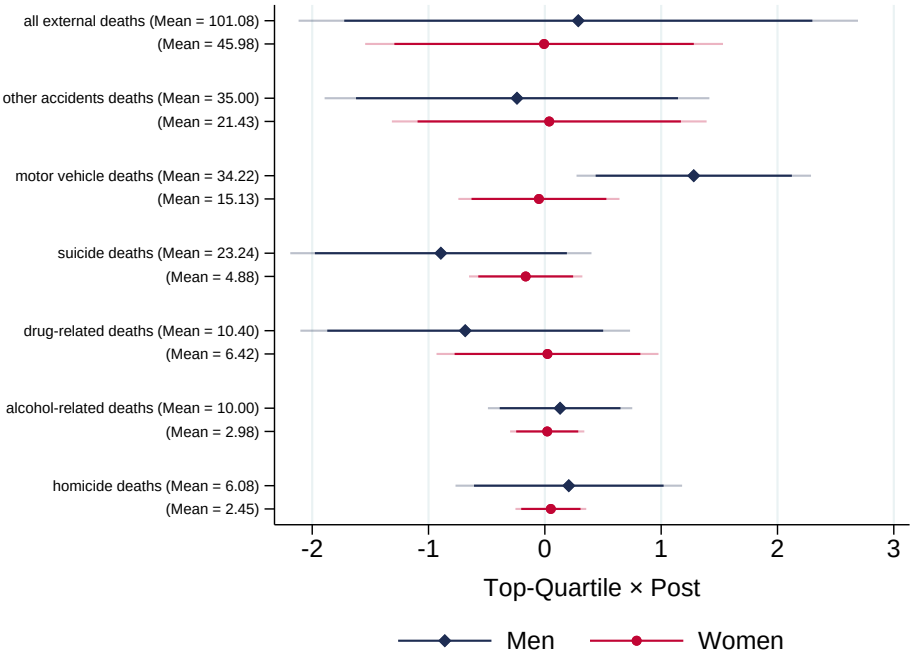
Notes: Each point represents the outcome from a separate regression (Equation (1)), and the dark and lighter shaded bars represent the associated 90% and 95% confidence intervals, respectively. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Death categories are taken from Stevens et al. (2015), and represent consistent definitions across ICD-9 and ICD-10 cause of death codes. All regression were estimated using interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.

Figure A.18: Internal and External Causes of Death: Differences by Gender

Panel A: Internal Causes (Age-Adjusted Death Rate per 100K)



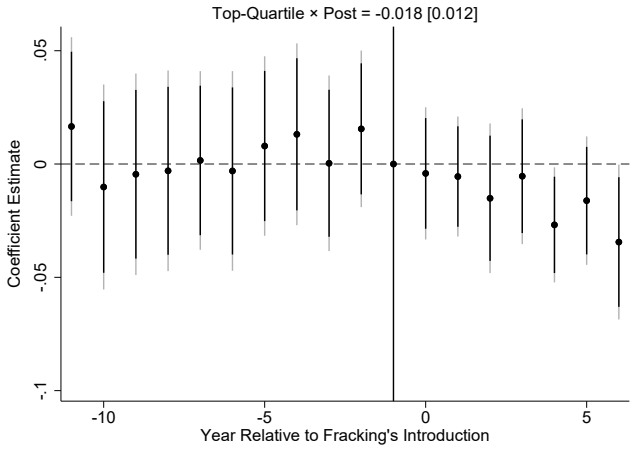
Panel B: External Causes (Age-Adjusted Death Rate per 100K)



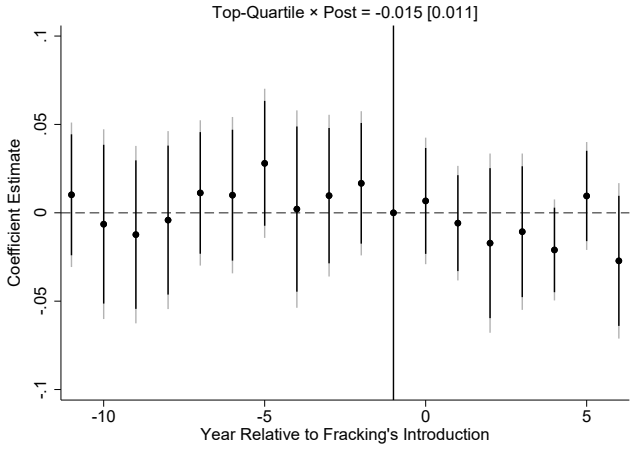
Notes: We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Death categories are taken from Stevens et al. (2015), and represent consistent definitions across ICD-9 and ICD-10 cause of death codes. The definitions of suicides, drug-related and alcohol-related deaths are taken from the Joint Economic Committee of the United States Congress. Each point represents the outcome from a separate regression (Equation (1)), and the dark and lighter shaded bars represent the associated 90% and 95% confidence intervals, respectively. All regressions were estimated using interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.

Figure A.19: Overall Mortality: Controlling for Compositional Changes

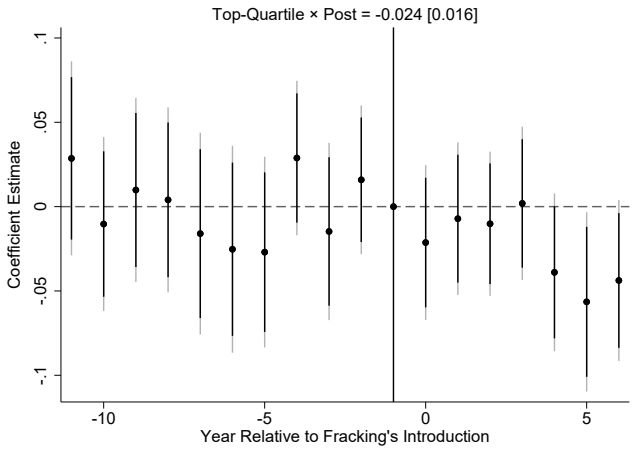
Panel A: Men and Women



Panel B: Men



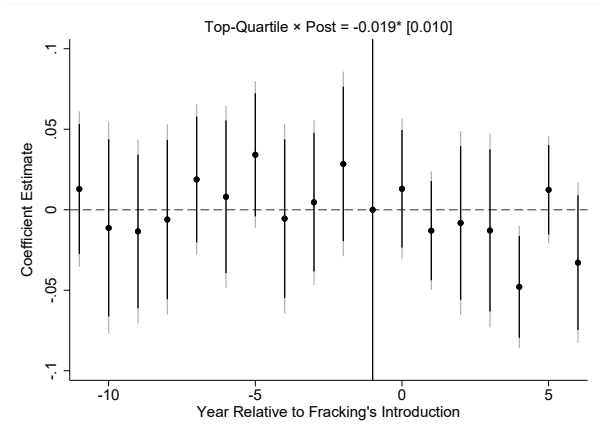
Panel C: Women



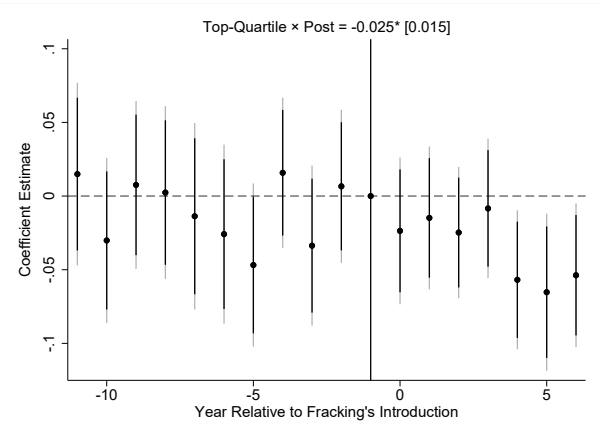
*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the inverse hyperbolic sine of overall mortality, and we control for the shares of relevant gender population belonging to the age categories 0-24, 25-44, 45-64, and 65-99. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Standard errors are adjusted for clustering at the county level. The difference-in-differences coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

Figure A.20: Internal and External Causes of Death: Controlling for Compositional Changes

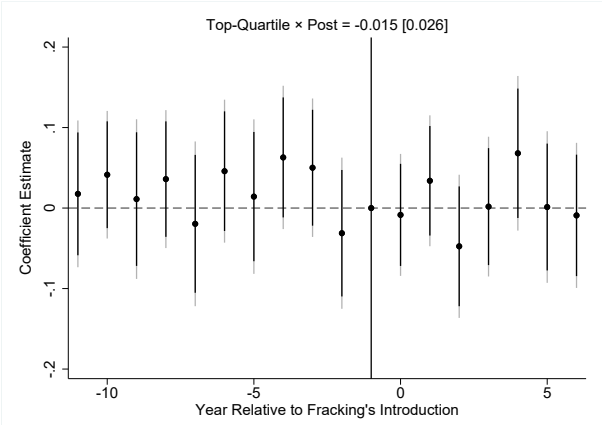
Panel A: IHS of Internal Deaths (Male)



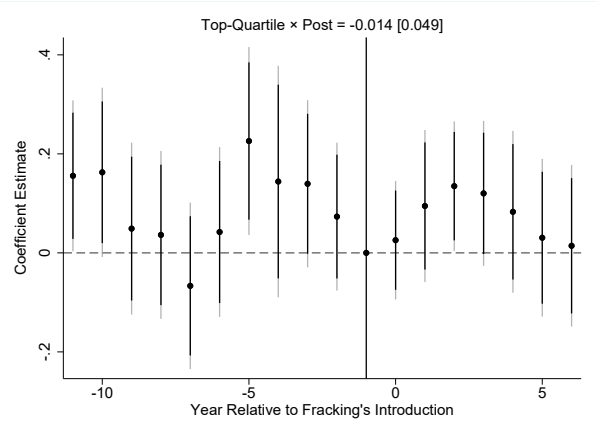
Panel B: IHS of Internal Deaths (Female)



Panel C: IHS of External Deaths (Male)



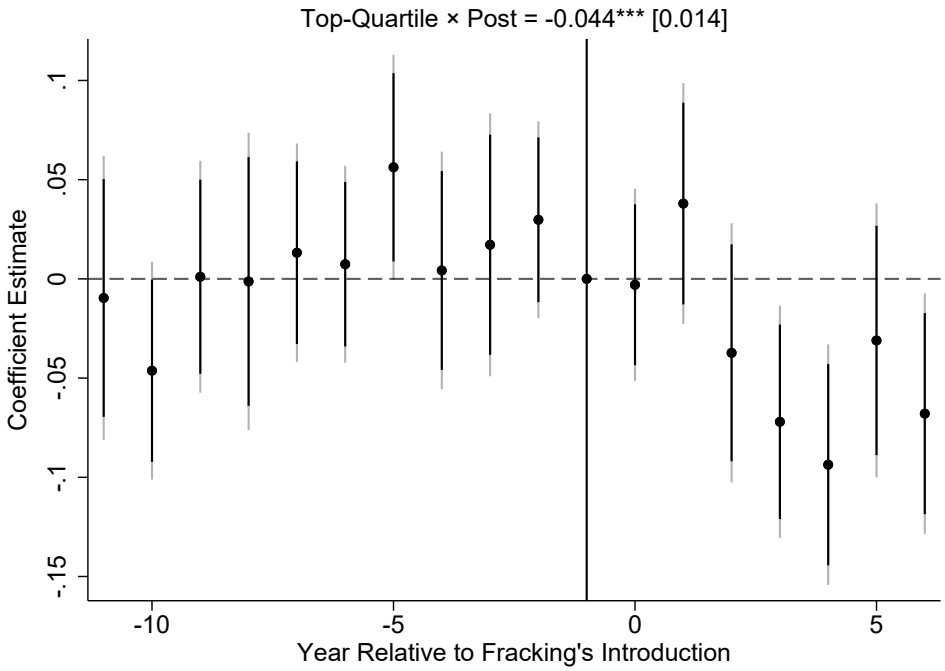
Panel D: IHS of External Deaths (Female)



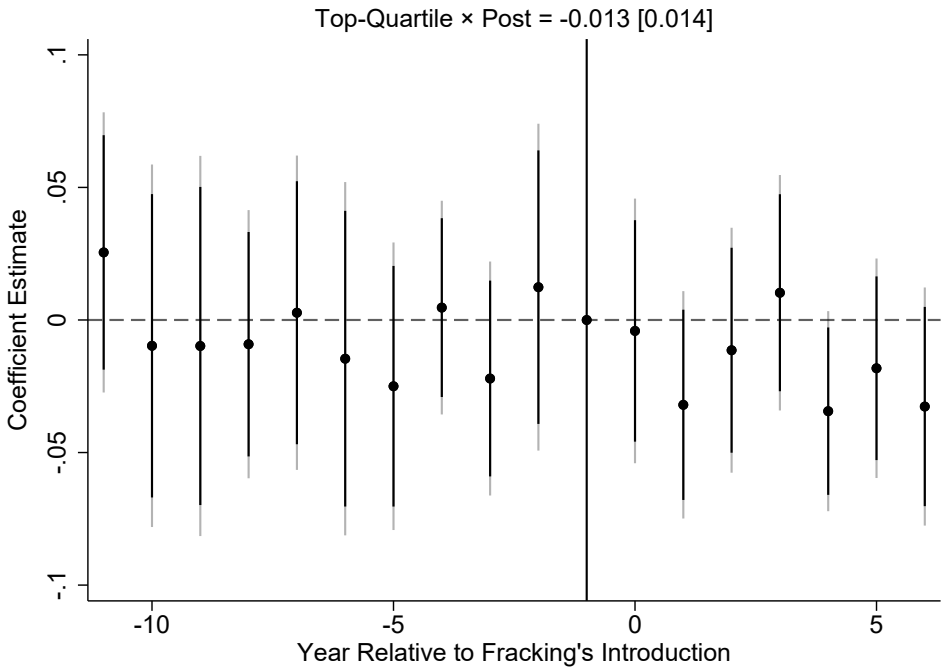
*Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Our primary outcome is the inverse hyperbolic sine (IHS) of the number of deaths, where the contemporaneous shares of the relevant demographic group for the ages 0-24, 25-44, 45-64, and 65-99 are included as controls. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Standard errors are adjusted for clustering at the county level. The difference-in-differences coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

Figure A.21: Cardiovascular vs Other Internal Mortality: Controlling for Compositional Changes

Panel A: Cardiovascular



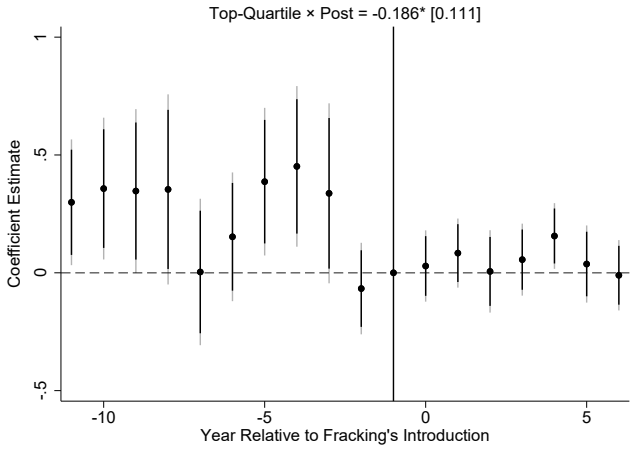
Panel B: Non-Cardiovascular



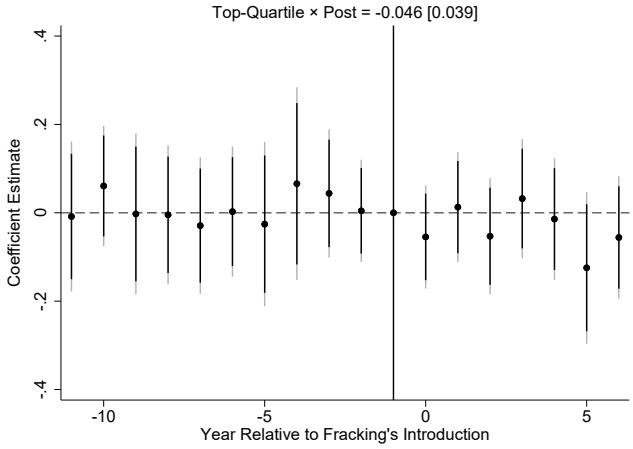
Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the inverse hyperbolic sine of Cardiovascular (Panel A) and Non-Cardiovascular Internal (Panel B) mortality, where the contemporaneous shares of the relevant demographic group for the ages 0-24, 25-44, 45-64, and 65-99 are included as controls. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. All regressions were estimated using interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights, and control for contemporaneous population. Standard errors are adjusted for clustering at the county level.

Figure A.22: Deaths of Despair: Controlling for Compositional Changes

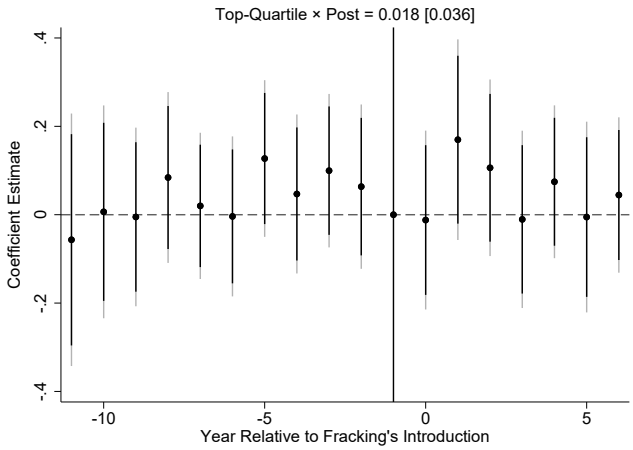
Panel A: Drug Overdoses



Panel B: Suicides



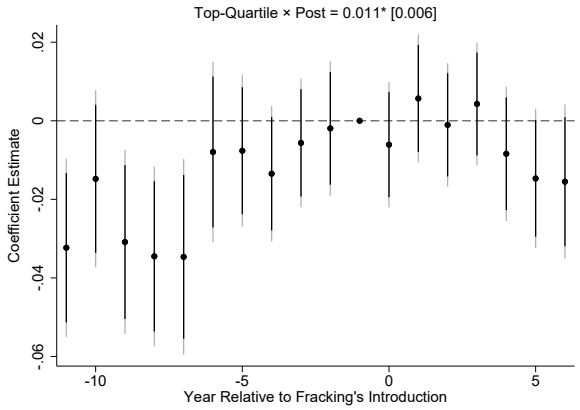
Panel C: Alcohol Related



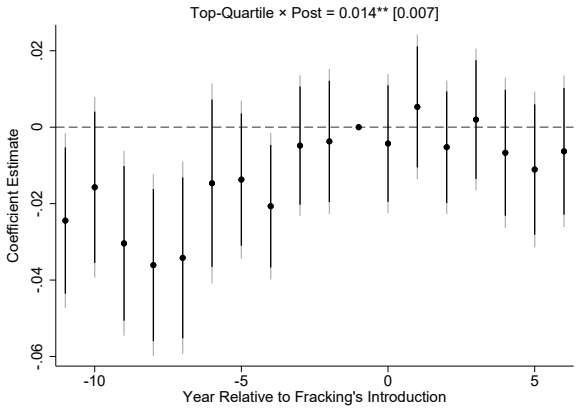
Notes: Each panel reports the point estimates with their associated 95% and 90% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the inverse hyperbolic sine of drug overdose (Panel A), suicide (Panel B), and alcohol-related (Panel C) mortality, where the contemporaneous shares of the relevant demographic group for the ages 0-24, 25-44, 45-64, and 65-99 are included as controls. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. All regressions were estimated using interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights, and control for contemporaneous population. Standard errors are adjusted for clustering at the county level.

Figure A.23: Potential Confounding Comparing Shale to No Shale Counties within State

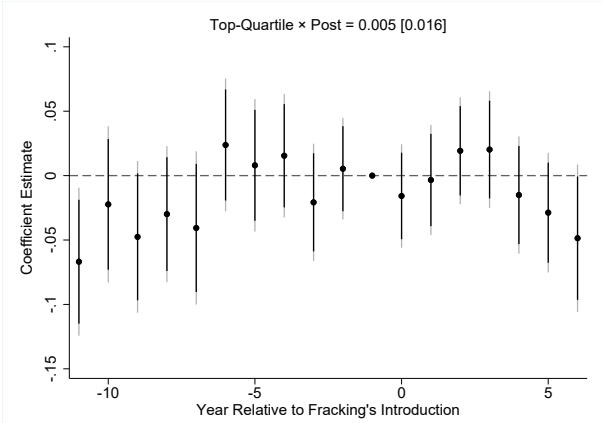
Panel A: Overall Mortality



Panel B: Internal Mortality



Panel C: External Mortality



*Notes: Represents an alternative specification to Equation (2) where we compare counties that reside over any shale play compared to those that do not reside under a shale play, within the same state. This is done in practice by defining treatment as an indicator equal to one if the county resides over any shale play, and including state-year fixed effects. The initiation of fracking in each state is defined as the earliest fracking date among plays within a state's border. Each panel reports the point estimates with their associated 95% and 90% confidence intervals. We take population counts for the same time period from SEER. Dependent variables are the inverse hyperbolic sine (IHS) of the relevant death counts, controlling for the relevant contemporaneous population. Each regression include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census and 2000 county-level population weights. Standard errors are adjusted for clustering at the county level. The difference-in-differences coefficient for each outcome is included above each event-study, as well as the relevant standard error in brackets. *** Significance 1%, ** 5%, * 10%.*

B Appendix Tables

Table B.1: Lower 48 States and Fracking Counties Comparison (1990 Variables)

	Any Shale Play	No Shale Play	Diff.
Age-Adjusted Death Rate	913.94	930.40	-16.46**
	(129.30)	(141.21)	[6.32]
Median Household Income	29970.18	31353.13	-1382.95***
	(6776.52)	(8703.30)	[343.13]
% High School Graduates	34.85	34.19	0.66*
	(6.64)	(6.04)	[0.31]
% in Manufacturing	5.75	8.61	-2.87***
	(4.41)	(6.48)	[0.23]
% Married	60.29	58.84	1.45***
	(5.46)	(6.61)	[0.27]
% Rural	62.15	63.67	-1.52
	(29.44)	(30.05)	[1.42]
% Veterans	14.65	14.80	-0.15
	(2.19)	(2.86)	[0.11]
% White	90.91	86.77	4.14***
	(10.15)	(16.14)	[0.55]
% Foreign Born	2.43	2.17	0.26
	(3.17)	(3.67)	[0.16]
% w/ a Bachelors Degree	8.94	9.02	-0.08
	(3.79)	(4.30)	[0.19]
Observations	519	2,589	3,108

Notes: All variables are measured at the county-level in 1990. Aside from the age-adjusted death rate, all variables are taken from the 1990 Decennial Census. The age-adjusted death rate is calculated using mortality data from the CDC's National Center for Health Statistics, and all the population data come from SEER.

Table B.2: Changes in Population

	Men				Women			
VARIABLES	Log Pop (<25)	Log Pop (25-44)	Log Pop (45-64)	Log Pop (65+)	Log Pop (<25)	Log Pop (25-44)	Log Pop (45-64)	Log Pop (65+)
Top-Quartile × Post	0.00813	0.0114	0.0160	-0.00856	0.00785	0.00738	0.0142	-0.000213
	(0.00931)	(0.0110)	(0.0111)	(0.0155)	(0.00842)	(0.0107)	(0.0129)	(0.0138)
Observations	8,712	8,712	8,712	8,712	8,712	8,712	8,712	8,712
Omits ND & MT?	YES	YES	YES	YES	YES	YES	YES	YES

*** p<0.01, ** p<0.05, * p<0.1

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. We take population counts from SEER. All values are calculated for individuals of every age in each county. All columns include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Some columns also omits all observations from North Dakota and Montana, and include 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.*

Table B.3: Changes in Population Composition

	Men				Women			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
VARIABLES	Share < 25	Share 25-44	Share 45-64	Share 65-99	Share < 25	Share 25-44	Share 45-64	Share 65-99
Top-Quartile × Post	0.000226	0.000623	0.00155	-0.00240*	0.000344	-0.000117	0.00124	-0.00147
	(0.00102)	(0.00194)	(0.000996)	(0.00140)	(0.00125)	(0.00130)	(0.00104)	(0.00157)
Observations	8,712	8,712	8,712	8,712	8,712	8,712	8,712	8,712
Omits ND & MT?	YES	YES	YES	YES	YES	YES	YES	YES

*** p<0.01, ** p<0.05, * p<0.1

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. We take population counts from SEER. All values are calculated for individuals of every age in each county. All columns include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Some columns also omits all observations from North Dakota and Montana, and include 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.*

Table B.4: Earnings by Gender - Robustness

	Men and Women				Men				Women			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Top-Quartile × Post	0.046***	0.041***	0.027***	0.024***	0.045***	0.041***	0.028***	0.023**	0.020***	0.017***	0.020***	0.021**
	[0.011]	[0.010]	[0.008]	[0.009]	[0.011]	[0.010]	[0.008]	[0.009]	[0.006]	[0.006]	[0.006]	[0.008]
No Missing Counties?	No	Yes	Yes	No	No	Yes	Yes	No	No	Yes	Yes	No
2000 Pop. Weights?	No	No	Yes	Yes	No	No	Yes	Yes	No	No	Yes	Yes
Omits ND & MT?	No	No	No	Yes	No	No	No	Yes	No	No	No	Yes
Outcome Mean	34,453	35,475	35,475	35,177	42,660	43,878	43,878	43,516	25,831	26,649	26,649	26,253
Observations	8,513	6,422	6,422	9,778	8,513	6,422	6,422	9,778	8,513	6,422	6,422	9,778

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. We take earnings measures (adjusted to real 2010 dollar amounts) and employment counts from the Quarterly Workforce Indicators database. We take population counts from SEER. All values are calculated for 14-99 year old individuals in each county. All columns include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Some columns also omits all observations from North Dakota and Montana, and include 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.*

Table B.5: Employment to Population Ratio by Gender - Robustness

	Men and Women				Men				Women			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Top-Quartile × Post	0.032***	0.028***	0.010*	0.013**	0.048***	0.042***	0.013**	0.016**	0.012**	0.010**	0.005	0.010*
	[0.008]	[0.008]	[0.005]	[0.006]	[0.011]	[0.011]	[0.007]	[0.007]	[0.005]	[0.005]	[0.005]	[0.005]
No Missing Counties?	No	Yes	Yes	No	No	Yes	Yes	No	No	Yes	Yes	No
2000 Pop. Weights?	No	No	Yes	Yes	No	No	Yes	Yes	No	No	Yes	Yes
Omits ND & MT?	No	No	No	Yes	No	No	No	Yes	No	No	No	Yes
Outcome Mean	0.50	0.50	0.50	0.49	0.52	0.52	0.52	0.51	0.48	0.49	0.49	0.47
Observations	8,513	6,422	6,422	9,778	8,513	6,422	6,422	9,778	8,513	6,422	6,422	9,778

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. We take earnings measures (adjusted to real 2010 dollar amounts) and employment counts from the Quarterly Workforce Indicators database. We take population counts from SEER. All values are calculated for 14-99 year old individuals in each county. All columns include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Some columns also omits all observations from North Dakota and Montana, and include 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.*

Table B.6: Age-Adjusted Overall Mortality Rates by Gender

	Men and Women		Men		Women	
	(1)	(2)	(3)	(4)	(5)	(6)
Top-Quartile × Post	-8.870**	-9.428**	-6.063	-6.893*	-9.938**	-10.19**
	(3.885)	(3.867)	(3.702)	(3.670)	(5.013)	(5.039)
Omits ND & MT?	NO	YES	NO	YES	NO	YES
Outcome Mean	853.90	861.83	851.71	858.85	851.09	860.72
Observations	9,341	8,711	9,341	8,711	9,341	8,711

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. All death rates are age-adjusted using the national age distribution across standard age categories in 2000 to eliminate bias caused by changing demographics over time. All columns include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Some columns also omits all observations from North Dakota and Montana, and include 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.*

Table B.7: IHS of Overall Mortality by Gender

	Men and Women		Men		Women	
	(1)	(2)	(3)	(4)	(5)	(6)
Top-Quartile × Post	-0.017*	-0.015	-0.015	-0.017*	-0.021**	-0.012
	[0.009]	[0.009]	[0.013]	[0.009]	[0.010]	[0.010]
2000 Pop. Weights?	No	Yes	No	Yes	No	Yes
Omits ND & MT?	No	Yes	No	Yes	No	Yes
Outcome Mean	672.34	714.05	329.85	350.21	342.49	363.85
Observations	9,342	8,712	9,342	8,712	9,342	8,712

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. All regressions include a time-varying control for the inverse hyperbolic sine of the relevant population group. All columns include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Some columns also omits all observations from North Dakota and Montana, and include 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.*

Table B.8: Overall Mortality - Heterogeneity by Age (Men)

	Less than 1		Ages 1-4		Ages 5-14		Ages 15-64		65 and Older	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Top-Quartile × Post	-0.003	-0.044	-0.018	-0.064	0.014	0.054	-0.041**	-0.018**	-0.002	-0.003
	[0.028]	[0.033]	[0.025]	[0.050]	[0.028]	[0.048]	[0.020]	[0.009]	[0.015]	[0.005]
2000 Pop. Weights?	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Omits ND & MT?	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Outcome Mean	3.82	4.06	0.73	0.77	1.01	1.07	99.80	106.14	224.37	238.03
Observations	9,342	8,712	9,342	8,712	9,342	8,712	9,342	8,712	9,342	8,712

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. All regressions include a time-varying control for the relevant population group. All columns include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Some columns also omits all observations from North Dakota and Montana, and include 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.*

Table B.9: Overall Mortality - Heterogeneity by Age (Women)

	Less than 1		Ages 1-4		Ages 5-14		Ages 15-64		65 and Older	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Top-Quartile × Post	-0.028	-0.010	0.003	-0.170**	-0.021	-0.140*	-0.019	-0.028**	-0.021*	-0.003
	[0.031]	[0.039]	[0.024]	[0.079]	[0.024]	[0.084]	[0.021]	[0.014]	[0.011]	[0.006]
2000 Pop. Weights?	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Omits ND & MT?	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes
Outcome Mean	2.96	3.14	0.54	0.58	0.70	0.74	59.75	63.57	278.45	295.73
Observations	9,342	8,712	9,342	8,712	9,342	8,712	9,342	8,712	9,342	8,712

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. All regressions include a time-varying control for the relevant population group. All columns include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Some columns also omits all observations from North Dakota and Montana, and include 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.*

Table B.10: Traffic Accidents by Vehicle Type

	All Vehicles		Any Truck Involved		No Truck Involved	
	(1)	(2)	(3)	(4)	(5)	(6)
Top-Quartile × Post	0.104***	0.018	0.146***	0.049	0.066**	0.006
	[0.032]	[0.032]	[0.037]	[0.073]	[0.032]	[0.031]
2000 Pop. Weights?	No	Yes	No	Yes	No	Yes
Omits ND & MT?	Yes	Yes	Yes	Yes	Yes	Yes
Outcome Mean	9.98	9.98	1.32	1.32	8.66	8.66
Observations	8,712	8,712	8,712	8,712	8,712	8,712

Notes: All variables are measured at the county-level in 1990. Aside from the age-adjusted death rate, all variables are taken from the 1990 Decennial Census. The age-adjusted death rate is calculated using mortality data from the CDC's National Center for Health Statistics, and all the population data come from SEER.

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. All columns include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Standard errors are adjusted for clustering at the county level.*