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

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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. Instead, 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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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 these findings are focused on short-term job loss, while a smaller literature looks at the mortality consequences of larger long-term negative shocks. However, there is 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 death (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.

Estimating the effect of labor demand shocks on mortality requires addressing the endogeneity of fracking production, which can manifest in several ways. Local regulations on drilling operations can limit or outright ban fracking, and these decisions may be directly related to factors that influence mortality, such as the strength of local labor markets and investments in public health. Additionally, places that 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. Specifically, we employ a difference-in-differences (DD) strategy that compares counties with higher geological potential for fracking to similar, adjacent counties with lower potential. Mechanically, we compare 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 amenable to fracking. We also use the differential timing of the adoption of modern fracking technologies across shale plays, which enabled producers to build 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 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 directly benefit from the growth in labor demand. This group experiences a reduction of 15 deaths per 100,000 people, constituting a 3% reduction in all-cause mortality. The effects are strongest for older working-age men and women (45-64) who experience 3% and 5% reductions, respectively. 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 8.9 deaths per 100,000, which translates to 6.1% of the overall decline in mortality between 2000-2018.[4] We also demonstrate that migratory responses are not driving our results. We find only modest changes in overall population or age distributions between our treatment and control counties. Further, our mortality results are robust when directly controlling for any compositional changes in the population and when excluding counties within the Bakken shale play, which experienced a particularly large in-

[4]The age-adjusted death rate fell from 869 in 2000, the year before the first use of fracking, to 723.6 per 100,000 in 2018, the end of our sample period: https://www.cdc.gov/nchs/data-visualization/mortality-trends/index.htm.

migration of male workers (Wilson, 2020). Finally, we present a bounding exercise suggesting that the mortality reductions we find cannot be reasonably explained by changing migration.

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 use.[5] We do find suggestive evidence that health insurance coverage increases in boom counties by matching our fracking data to county-level coverage estimates constructed by the US Census Bureau. 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.

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

With the restrictive assumption that increased coverage drives our findings, our results would imply a 1 percentage point increase in coverage leads to a 2.1% overall mortality decline, falling between these reductions induced solely by health insurance expansions, supporting this as a plausible mechanism.[6] 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 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 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 study. Sullivan and Von Wachter (2009) exploit plant closings in Pennsylvania and find that sustained employment and earnings losses of around 10% after a decade lead 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

[6]While we do not directly test whether coverage increases stem from Medicaid versus employer-sponsored insurance, we find a reduction in Supplemental Nutrition Assistance Program participation, suggesting these coverage gains are likely driven by employer-sponsored insurance rather than expanded public programs.

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. 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. Instead, we aim 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.[7] In this way, our findings are more closely related to 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).[8] 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 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 wellbores 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 wellbore, 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 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

[7] 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.

[8] Our paper is also related to previous literature exploiting economic conditions generated by the coal boom and bust to test economic models of human capital (Black et al., 2005) and fertility choice (Black et al., 2013).

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. Figure 1 Panel B plots the dramatic increase in fracking production over time from 2000, when 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 following 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,[9] and our main sample includes data from 1990 to 2018. As we discuss below, our empirical strategy only compares counties over the same shale play; thus, we omit 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 519 counties (112 of which are in the top-quartile of the within-play RPI) and 29 years of data.

[9] If county boundaries change over time, we aggregate to the 2000 boundary definitions using initial population weights. For example, in 2001, Broomfield, Colorado was 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

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. There are sixteen Shale Plays in our sample, which constitute contiguous counties across different regions of the U.S. We obtained well-level production data from Enverus, a private oil and gas software company, through their academic outreach initiative. These data include monthly production levels and wellbore orientation, which we use to identify fracking wells.[10]

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.[11] We therefore follow Bartik et al. (2019) and identify counties that 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.

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 the 1990 Census (well before the technology that enabled fracking was first applied).

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

[11]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.

Different county characteristics are associated with differential mortality risk. For instance, educational and racial gradients in mortality have been well-documented in the literature (Case and Deaton, 2022). Similarly, areas with higher manufacturing employment have experienced different mortality trajectories due to long-run deindustrialization (Autor et al., 2019). In addition to these factors, we also control for median household income, the share of the population that are veterans, and other demographic information. 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 presents baseline 1990 summary statistics for top-quartile and other shale play counties, and shows that there are no statistically significant differences (in terms of 1990 characteristics) between treatment and control counties before the boom. Of course, given the nature of our empirical strategy, it is the trends rather than the level differences that matter, which we will explore in our analysis.

We show these same baseline differences across all shale play counties and the rest of the lower 48 states in Appendix Table B.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 began exploratory adoption of new fracking technologies in the Barnett shale play in Texas as early as 2001, more well-known fracking hotspots like the Barnett shale play in North Dakota and the Marcellus Shale plays in the Mid-Atlantic did not begin widespread fracking production until 2007 and 2008, respectively. Despite an initial lag, top-quartile RPI counties produce substantially more than the 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 (LEHD). These data are primarily based on unemployment insurance earnings data from participating states[12] 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 census of all deaths in the United States: the restricted-access version of the National Vital Statistics System (NVSS) mortality files from 1990-2018. These data identify basic demographic information, primary/additional causes of death, and the county of residence and occurrence. We follow Stevens et al. (2015) by separating all causes of death into mutually exclusive categories based on 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, our use of consistent, broad categories ensures comparability across time.

Our primary outcome is crude death rate, or the number of deaths per 100,000 working-aged (25-64) individuals. We take these population data from estimates constructed by the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) Program. While the crude death rate is the total number of deaths for a specific demographic group divided by the current relevant population, the age-adjusted death rate is a weighted aver-

[12] 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.6 and Appendix Table B.7.

age of crude death rates across standard age categories, where the weights are the national population shares in those age categories in 2000.

III.D Health Insurance Data

We explore changes in insurance coverage using data from the Small Area Health Insurance Estimates (SAHIE) Program. The SAHIE is administered by the US Census Bureau, which produces model-based estimates at the county level by combining data from multiple sources, including the American Community Survey (ACS), federal tax returns, Supplemental Nutrition Assistance Program (SNAP) participation, Medicaid and Children's Health Insurance Program (CHIP) participation, and Census population estimates. It is the only source for single-year estimates of health insurance coverage for all US counties. We note that the SAHIE data become available only from 2008 onward with consistent methodology, which means we lack pre-boom coverage data for earlier adopting shale plays. Given this, we match the SAHIE data to counties in the six shale plays that begin fracking after 2008.

We focus on the estimated percentage of the county population ages 18-64 with health insurance coverage to best approximate employer-provided insurance coverage for working-age adults. One limitation of the SAHIE data is that they do not distinguish between public and private insurance coverage, so we are unable to identify our ideal outcome of interest directly.

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. 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. Further, the timing of fracking adoption may reflect unobserved factors that also affect mortality. For instance, firms may have prioritized drilling in plays with improving infrastructure, favorable regulatory policy shifts, or underlying demographic trends that would have influenced health outcomes independently of fracking. In addition, broader macroeconomic shocks, such as global energy price movements, could have both shifted the economic incentives for adopting fracking and indirectly influenced mortality through income channels.

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 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:

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

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.[13] Our baseline specification uses the crude death rate as the outcome variable. We also control for time-invariant county characteristics with county fixed effects, λ_c. Regressions are weighted by the 2000 population 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 adjust for any contemporaneous events that might affect entire shale plays, such as regional economic cycles, region-specific regulatory changes, or energy price fluctuations that both incentivized frack-

[13] All the variables shown in Table 1 are included as controls aside from the initial age-adjusted death rate.

ing adoption and potentially affected mortality through other channels. These effectively aggregate estimates from each shale play.

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 before treatment and 7 years post treatment (including the year of initiation of fracking), or 18 event-years of data for each observation. We show the robustness of our results to different sample selections and weights in the Appendix.

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 before fracking's introduction:

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

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 the omitted year. Given the large number of zeros in many of the mortality outcomes, we also estimate exponential models of Equation (2) using Poisson regressions to obtain semi-elasticity interpretations without the issues that arise from log-like transformations of the outcome variables (Chen and Roth, 2024), as well as to test the robustness of the pre-trend assumption to different functional forms (Wooldridge, 2023).

A key feature of our identification strategy is that the RPI accurately predicts the highest intensity boom counties in terms of actual production. Appendix 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.[14] 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.

V Results

V.A Earnings and Employment

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 with the associated 95% confidence intervals. 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 3% increase in earnings and employment for men, the coefficients for later event-years are larger and settle closer to 3-5%. 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.

[14] 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.

Thus, these labor market effects are likely smaller than the overall impact of fracking.

Although fracking is an almost entirely male-dominated field, we find wage and employment growth for women.[15] For women, both earnings and employment increase by around 2%. However, both Bartik et al. (2019) and Feyrer et al. (2017) have shown 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 found differential sizes of the male and female labor demand shocks in response to the boom, and they found slightly larger effects for male earnings (4%) and the employment-to-population ratio (5%) than that of our results. We show in Appendix Table B.6 and Appendix Table B.7 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.

V.B Mortality Results

We now consider the reduced-form effects of fracking amenability on mortality. Table 2 looks at overall mortality. The dependent variable is the crude death rate per 100,000 working-aged individuals. Panel A uses the overall rate, and Panels B and C use male and female specific mortality rates, respectively. The first column only includes county and play-by-year fixed effects, the second column adds controls for the baseline percent of the county population that is white, in manufacturing, and that have a high school degree, three factors strongly associated with mortality trends over this period, and the third column adds all the remaining controls from Table 1.

Column (1) shows a negatively signed but statistically insignificant effect on mortality. However, including the baseline controls in column (2) increases the magnitude to a decline

[15] 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.

of 14.7 deaths per 100,000, suggesting that any confounding was attenuating our coefficients towards zero. Table 1 shows that top-quartile counties had a larger share of non-high school graduates, non-white populations, and manufacturing employment than that of their lower quartile counterparts. While these differences were insignificant, these factors may positively influence subsequent mortality trends, creating a positive bias working against any negative effect. Including the remaining controls in column (3) does not have a strong effect on the coefficient, showing an overall mortality decline by 15 deaths per 100,000 in top-quartile counties relative to their control counterparts. Panels B and C show that the male mortality effects drive the sensitivity to the baseline controls. Column (3) shows a decrease of 17 deaths per 100,000 and 14 deaths per 100,000 working-aged men and women, respectively. Given the lower overall mortality rates for women compared to that of men (reported in the bottom row of the Panels), this suggests a 3.1% decline in mortality for men and a 4.2% reduction for women.[16]

Figure 3 Panel A plots the estimates from equation (2) for overall mortality with their associated 95% confidence intervals. Panels B and C separately examine overall mortality for men and women, respectively. Overall, there is a reassuring absence of differential trends in mortality between treatment and control counties before the initiation of fracking.[17] After fracking, there begins a decline in overall mortality. There is an imprecisely estimated decline of 2 to 5 deaths per 100,000 starting 2-3 years following the initiation of fracking, which grows to a statistically significant reduction of 21 deaths per 100,000 six years after fracking.

Panel B looks at men. While the point estimates suggest a decline in overall mortality of 20 deaths per 100,000 six years after fracking, the individual point estimates are not statistically significant at the 5% level. For women, Panel C shows a statistically significant decline in overall mortality of 19 deaths per 100,000 4 years after the initiation of fracking, followed by a 22 per 100,000 decline after six years. The timing of these reductions in mortality are consistent with Sullivan and Von Wachter (2009) who find that displaced workers have a high and persistent risk of death 4-5 years after job loss.

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

[17] A joint test on whether all pre-event year coefficients are statistically significantly different from zero fails to reject (p-value = .4).

Given the sensitivity of the coefficients to the addition of base controls in Table 2, albeit suggesting that any influence they capture attenuates our estimates towards zero, we also visually inspect pre - trends and dynamics of overall mortality without including any controls Appendix Figure A.3. These reassuringly confirm an absence of pre-trends prior to the initiation of fracking, and show a similar but less precisely estimated reduction in mortality in the post years. Six years after the initiation of fracking, the coefficient shows a statistically significant reduction of 19 deaths per 100,000, similar to the change in Figure 3.

We now examine whether the results are robust to using the age-adjusted mortality rate discussed in Section *III.C* as the dependent variable. Column (1) of Appendix Table B.8 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, a 1% decline in terms of the sample period mean. The remaining columns show that the coefficients are negative and of similar magnitudes to the combined death rate for men and larger in magnitude for women, in line with the main specification. Appendix Figure A.2 then shows the event study estimates from (2) using the age-adjusted mortality rate. Both are similar to our main specification. We next estimate a nonlinear Poisson model of Equation (2). Appendix Figure A.4 reaffirms the absence of pre-trends and decline in mortality from the main results, lending further support to the parallel trends assumption (Wooldridge, 2023). The DD coefficients show a 2.8% reduction in the death rate for men and a 3.6% reduction for women.[18] Finally, Appendix Figure A.20 shows that our findings are robust to running unweighted specifications.

While we do not explicitly instrument overall earnings or employment, we can consider the implied 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 3% decline in the overall working-age mortality rate from the reported coefficient on top of Appendix Figure A.4 and the 2.7% increase in overall wages from column (3) of Appendix Table B.6, our estimates suggest that a 1% increase in wages leads to an approximately unit-elastic 1.1% decline in

[18]Following the recommendation of Chen and Roth (2024), we report transformed coefficients $e^{\hat{\beta}} - 1$.

overall mortality.

To further understand which groups drive our results, we follow Stevens et al. (2015) and create broadly defined age groups (under 25, 25-44, 45-64, and 65 and older) corresponding to different parts of an individual's working life. These age ranges also align with 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). Appendix Table B.2 shows estimates from Equation (1) and Appendix Figures A.5, A.6, and A.7 plot estimates from Equation (2) using these age ranges for all, male, and female respectively. Working-aged populations drive the mortality decline. Although we find negative coefficients for younger working-age individuals, mortality reductions are driven by those aged 45–64.[19] There is a decline of 27 and 21 deaths per 100,000 men and women aged 45-64, respectively. These suggest the mortality reductions are driven by the types of deaths found to decrease in response to increased access to medical care. In the next section, we explore this further by examining specific causes of death.[20]

V.C Heterogeneity by Cause of Death

Internal causes of death, such as circulatory and respiratory illnesses, increase following 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 as 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 by external causes, such as suicides and homicides. Panels A and B of Figure 4 present estimates of Equation

[19] A Wald test rejects the equality of the coefficients across the 25-44 and 45-64 regressions (p-value = .0003).

[20] Note, Denham et al. (2021) finds an increase in hospitalizations for those 65-99 (particularly for women) with additional fracking production. Our null findings here are likely due to both treatment and control counties experiencing some degree of fracking, exposing this susceptible age group to the same pollutants.

(2) for the death rates for 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. Top-quartile counties experience a decline of 13.6 internal causes of death per 100,000 relative to other counties. Appendix Figure A.8 breaks down these deaths by gender, again confirming that the main mortality effects are driven by internal causes of death, with a reduction of 14.7 and 12.6 deaths per 100,000 men and women, respectively.[21] Appendix Figures A.10 and A.11 show the same results using our Poisson specification, showing that the reductions translate to a 3.2% reduction for men and a 3.9% reduction for women.

In Figure 5, we separately consider event studies for cardiovascular mortality and non-cardiovascular, internal mortality (e.g. cancer, kidney disease, etc.). There is a reduction in cardiovascular mortality of 5 deaths per 100,000 and a reduction in non-cardiovascular type internal mortality of 8 deaths per 100,000. While we fail to reject the test of equality across the equations in Figure 5, the higher prevalence of non-cardiovascular type internal mortality (247 versus 114 cardiovascular deaths per 100,000) in our sample suggests a larger proportional decrease for cardiovascular mortality. The Poisson estimates in Figure A.12 confirm a reduction in the cardiovascular death rate of 5.3% compared to a 2.6% reduction for other internal mortality.

Examining external causes of death, Appendix Figures A.9 and A.13 show no effect for "deaths of despair", albeit imprecisely estimated for drug overdoses. Finally, Appendix Figure A.14 looks at specific types of internal (Panel A) and external (Panel B) causes for men and women separately. We interpret this exercise as exploratory and suggestive, as increasing the number of outcomes raises concerns over multiple hypothesis testing. Panel A shows statistically significant declines in cardiovascular and kidney/urethra related deaths (renal failure, kidney infections, etc.) for men, with mainly imprecisely estimated declines for women. In Panel B, we find no statistically significant but mostly negatively signed changes in external causes of death, except for traffic accidents. This is consistent with Moore and Evans (2012), who find that traffic accidents are pro-cyclical.[22]

[21] Performing a Wald test across equations, we reject equality of parameters across internal and external deaths (p-value ≈ 0, $\tilde{\chi}^2 = 17.9$). As earlier, we fail to reject that the effects on internal deaths are the same across genders (p-value = .5539).

[22] 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

V.D Health Insurance

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 both employment and 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 following the boom.[23] Wherry and Miller (2016) 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).[24] 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 turn to the SAHIE data discussed in Section *III.D*. We regress the share of individuals aged 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 1.6 percentage points, or a 2% 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 100,000, which is smaller than our estimated reduction of 15 deaths per 100,000. 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.

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 crude death rate of the number of accidents. These support increases in truck traffic accidents.

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

[24] 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).

While the SAHIE data do not allow us to distinguish whether this increase comes from employer-sponsored insurance or expansions in public programs such as Medicaid, several lines of reasoning suggest that private insurance is the likely source of these coverage increases. Recall that employment expands across many sectors beyond oil and gas following the fracking boom. However, even within oil and gas, anecdotal evidence suggests that fracking jobs provided fairly robust health insurance. Surveys from Rigzone, a large online oil and gas industry job posting site and career network platform, indicate that Oil and Gas professionals are accustomed to rich health benefit offerings, and 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 (Jones, 2019).

Given that Medicaid expansions are typically counter-cyclical (CDC, 2009; Jang and Lee, 2023), it is not likely that expansions in economic opportunity from the boom would result in expansions in Medicaid.[25] We can also examine trends in another key means-tested safety net program: the Supplemental Nutrition Assistance Program (SNAP). Because Medicaid and SNAP enrollment are often tightly linked at the household level, trends in SNAP participation can serve as a useful proxy for shifts in public insurance enrollment.[26] We turn to the US Department of Agriculture (USDA) SNAP Data System to obtain the active number of participants in SNAP by county between 1997-2011. We restrict our sample to plays that initiated fracking before 2009 to maintain a balanced panel with multiple pre and post years around the initiation of fracking. Appendix Figure A.15 presents estimates of Equation (2) for 4 years pre and 3 years post fracking using the share of the county population who are SNAP participants as the dependent variable, with the DD estimate reported at the top. These results show a reduction in SNAP participation in top-quartile counties relative to control counties following the initiation of fracking, with an overall decline of .5 percentage points, a 4.9% decline from the mean of 11% in the pre-period. Taken together, these findings all point to private employer-sponsored insurance as the likely source of the observed coverage increases.

[25] Similarly, Black et al. (2002) and Black et al. (2003) find reductions in disability payments and welfare expenditures on the Aid to Families with Dependent Children program from the 1970s coal boom.

[26] Wagner and Huguelet (2016) estimates that in 2014, about 3/4ths of households receiving SNAP also had a member of their household receiving health insurance coverage through Medicaid or CHIP.

V.E Additional Results and Robustness

Our findings suggest symmetric effects between long-term job loss and gains on internal mortality, 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 whether migratory responses or the direct health effects of fracking production play a role in our findings.

*V.E.*1 Migration

While it is not obvious ex ante that migrants are selected based on higher or lower mortality risk, particularly among older cohorts (Fletcher et al., 2022), Wilson (2020) found a sizable migration response to the fracking boom, mainly in the Bakken Shale play, intersecting North Dakota and Montana. Omitting these two states from our sample leaves our results largely unchanged (Appendix Figures A.18- A.20).

To adjust for any compositional demographic changes following the boom, Appendix Figures A.16 and A.17 include controls for the contemporaneous age shares of the relevant population and confirm similar mortality declines as before. Secondly, it may be the case that our treatment counties had better pre-existing access to healthcare (Finkelstein et al., 2021) or better prior health behaviors (Couillard et al., 2021) that led to reductions in mortality for in-migrants. However, in Appendix Figure A.21 we expand on Appendix Figure A.16 and proxy for such factors by including the baseline county age-adjusted death rate interacted with year fixed effects and again find similar results.

We also directly test for in-migration by estimating Equation (1) using both the population and age-shares as the dependent variables. Appendix Tables B.3 and B.4 and Table B.5 show statistically insignificant and modestly sized coefficients, though the point estimates are positive for the working-age groups.[27] Additionally, treatment counties initially had lower mortality rates than control counties (Table 1), making it unlikely that migration from control to treatment counties would explain the mortality reductions.[28]

[27]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 drive our results.

[28]Top-quartile counties have 7 fewer deaths per 100,000 on average compared to bottom quartile counties in the same play in

Finally, we perform several bounding exercises to gauge whether migration could plausibly drive our results. Assume there are two types of individuals: stayers (those living in counties as of the initiation of fracking) and migrants (those who arrive from non-fracking regions in response to new economic opportunities).[29] We can express the post-fracking mortality rate in treatment counties (M^{tq}) as a weighted average of the mortality rate for stayers (M^{stayers}) and that of migrants (M^{migrants}):

$$M^{\text{tq}} = M^{\text{stayers}} \times (1 - p^{\text{migrants}}) + M^{\text{migrants}} \times p^{\text{migrants}} \tag{3}$$

where p^{migrants} is the share of the population who are migrants, and $p^{\text{migrants}} + p^{\text{stayers}} = 1$.

With this framework, we first ask: if stayer mortality remains constant, how much lower would the mortality rate of movers need to be to generate our results? Conservatively, we assume stayer mortality is held constant at its pre-treatment (event time -1) level of 424 deaths per 100,000.[30] Table 2 shows a reduction of 15 deaths per 100,000, making the observed post-fracking mortality rate $M^{\text{tq}} = 424 - 15 = 409$. Appendix Table B.3 shows that top-quartile counties experienced a (statistically insignificant) 1.2% increase in the working-age population, which we use to approximate p^{migrants}.[31] Plugging these values into Equation (3) implies that $M^{\text{migrants}} = -826$, suggesting that the actual level of population change we observe cannot feasibly generate the mortality reductions in our estimates alone.

Given this, we relax the assumption that the rising economic opportunity does not lower mortality for stayers at all. Instead we now ask: if fracking does lead to lower stayer mortality, how much might we be overstating the effects assuming the healthiest movers? For this calculation, we assume that migrants arrive solely from Minnesota, the state with the lowest working-age mortality rate in 2000 (the last pre-treatment year) of 263 deaths per 100,000, and that they constitute 1.2% of the post-fracking population. We then solve for the implied

the year before the initiation of fracking.

[29] We assume no out-migrants: people leaving top-quartile counties in response to higher economic opportunity.

[30] The crude death rate for working-aged adults weakly increases over time, so choosing the death rate in the event year before fracking provides a more restrictive assumption than linearly interpolating the mortality rate based on the pre-fracking event years.

[31] When we observe a 1.2% increase in population, this is approximately equivalent to saying migrants constitute 1.2% of the post-fracking population. More precisely, if the initial population I increases by a percentage x, then migrants as a share of the new total population is $\frac{x*I}{(1+x)*I} = \frac{x}{1+x}$, which for small values of x is very close to x. The average population of working-age individuals in top-quartile counties in event time -1 was 44,700 people, which implies an average increase of 536 working-age individuals in treatment counties.

post-fracking mortality rate of stayers ($M^{stayers}$). This yields $M^{stayers} = 410.8$ per 100,000. Even under the assumption of low mortality in-migrants, stayers' mortality would still decline by 13.2 deaths per 100,000, or 88% of the overall observed decline in mortality.

Conversely, we finally ask: given the healthiest possible movers, what is the share of the post-fracking population that would need to be movers to generate our results? We again assume that migrants have Minnesota's low mortality rate of 263 per 100,000 (M^{migrants}). Now solving for p^{migrants} implies that migrants must constitute 9.3% of the post-fracking population. This corresponds to a 10.3% increase over the pre-fracking population, roughly 10 times our actual estimate of the population increase of 1.2%.

We can also compare these figures to the migratory response found in the previous literature. Wilson (2020) finds that a 1% increase in earnings from fracking increased the population by 0.11%, and examining a similar economic shock, Black et al. (2005) finds that a 1% increase in earnings from the coal boom increased the population by 0.16%. Appendix Table B.6 shows that top-quartile counties experienced a 2.7% increase in earnings. To generate the reduction in mortality we find, a 1% increase in earnings from fracking would need to generate a 3.8% increase in the population, an elasticity over an order of magnitude larger than migration responses found in other work.

Overall, these bounding exercises demonstrate that even under assumptions that strongly favor a migration-based explanation, the observed effects are too large to be plausibly driven by migratory changes alone.

*V.E.*2 Direct Effects of Fracking

Our empirical design compares counties within the same shale plays. Given that prior research documents negative health effects of fracking, especially near drilling sites (Black et al., 2021; Hill and Ma, 2022; Currie et al., 2017), any direct adverse impact of fracking would bias *against* detecting beneficial effects of local economic opportunity as treatment counties experience more drilling. We find no increase in mortality for those aged 65-99, and in Appendix Table B.9, we show no changes in infant mortality, both groups that are more susceptible to increased morbidity from fracking-induced pollution (Denham et al., 2021; Hill and Ma, 2022). Together, these results imply that direct fracking-related health effects are not driving our findings.

Black et al. (2021) stresses the difficulty in obtaining causal evidence within the fracking and health literature and Bartik et al. (2019) highlight significant imbalances between shale and non-shale counties. Here, we demonstrate this further in the mortality context by modifying our identification strategy to include comparisons with counties that do not lie over a shale play. 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 only compare shale play counties to other non-shale play counties within the same state.[32] 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 those of non-shale play counties within the same state, after the initiation of fracking relative to before.

Appendix Figure A.22 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* in overall and internal mortality. This finding may be driven by these differential pre-trends or partly by fracking production itself (e.g., pollution). We do not aim to distinguish this here, but this exercise complements Bartik et al. (2019) and Black et al. (2021) by stressing the importance of making the correct comparisons when identifying the mortality impacts from the economic opportunity generated by the fracking boom.

[32]Note that this effectively identifies the estimate only from counties that reside in a state that overlaps with a shale play.

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 recently, 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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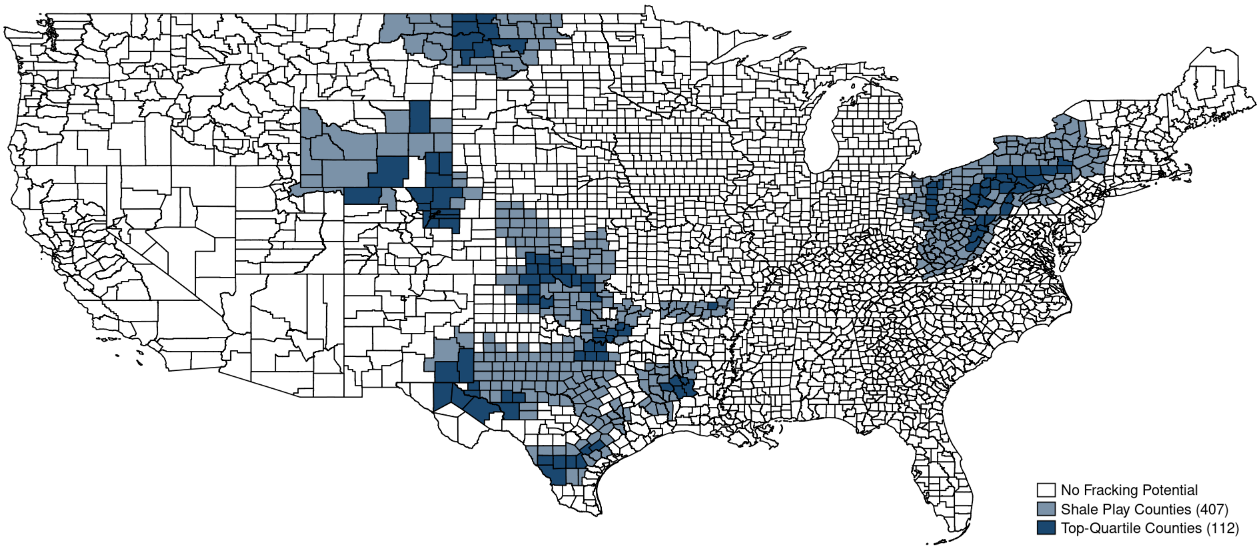
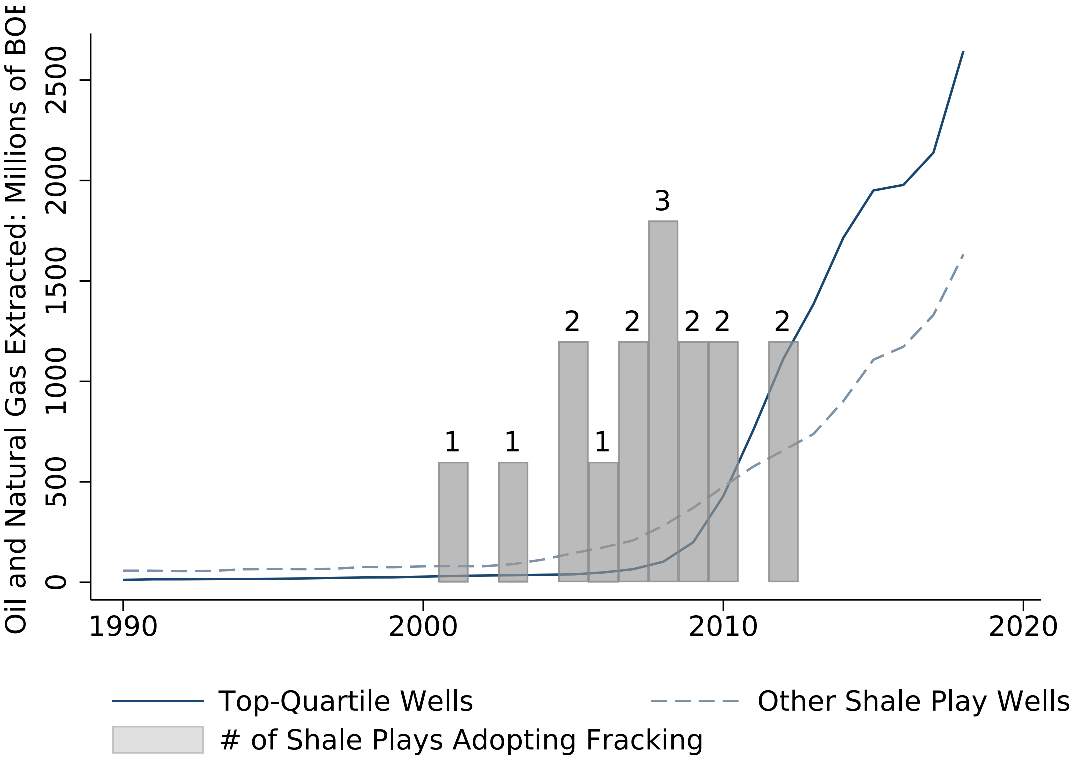
VII Figures

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

Panel A: Mapping RPI by US Counties



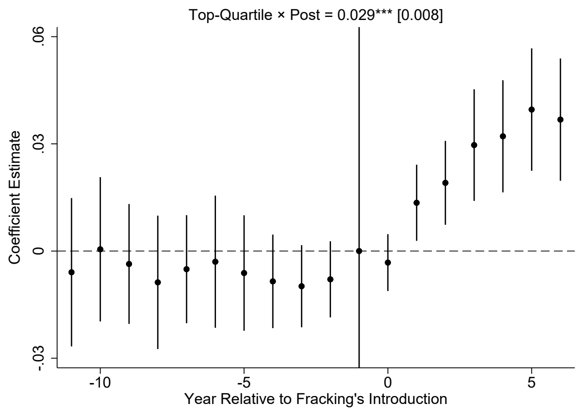
Panel B: County-Level Fracking Production by RPI



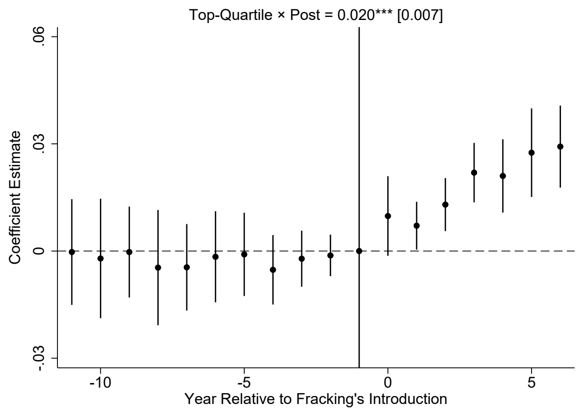
Notes: Panel A plots all US counties from the lower 48 states using 2000 census boundaries. White counties do not intersect with a shale play and are unable to benefit from the fracking boom. Lightly shaded counties (control) intersect with a shale play and are in the bottom three quartiles of the RPI, 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

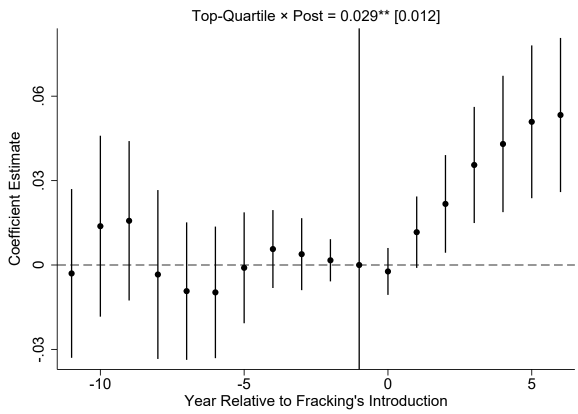
Panel A: Log of Male Earnings



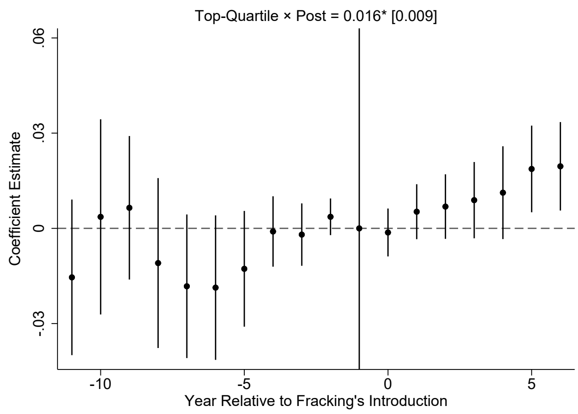
Panel B: Log of Female Earnings



Panel C: Log of Male Employment to Pop. Ratio



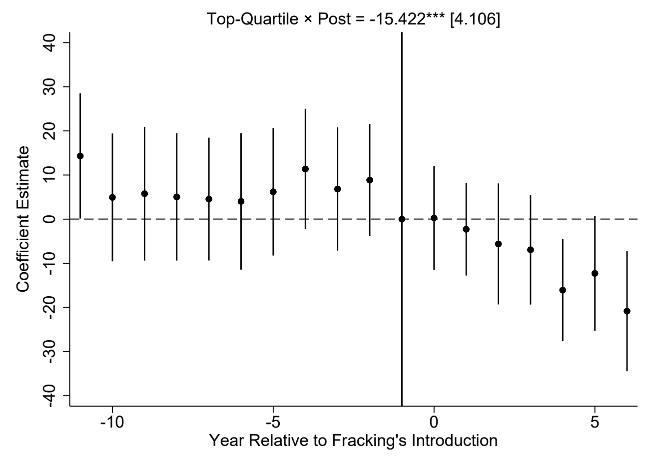
Panel D: Log of Female Employment to Pop. Ratio



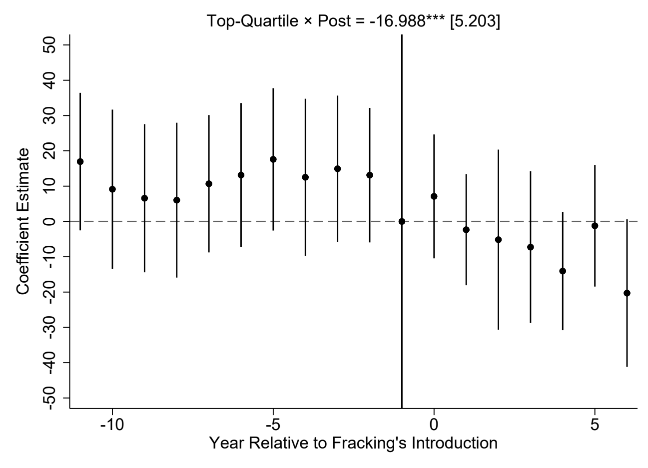
*Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) for the balanced set of event-years. We take earnings measures (adjusted to real 2010 $ amounts) and employment counts from the QWI database. We take population counts from SEER. All values are calculated for 14-99 year old individuals in each county. Each regression includes 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 3: Overall Mortality by Gender

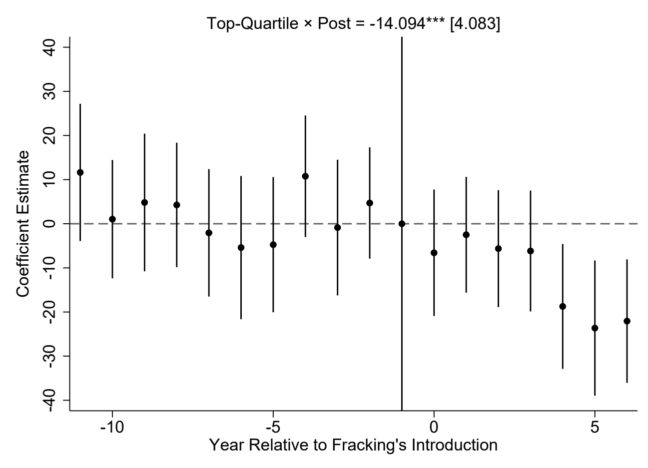
Panel A: Men and Women



Panel B: Men



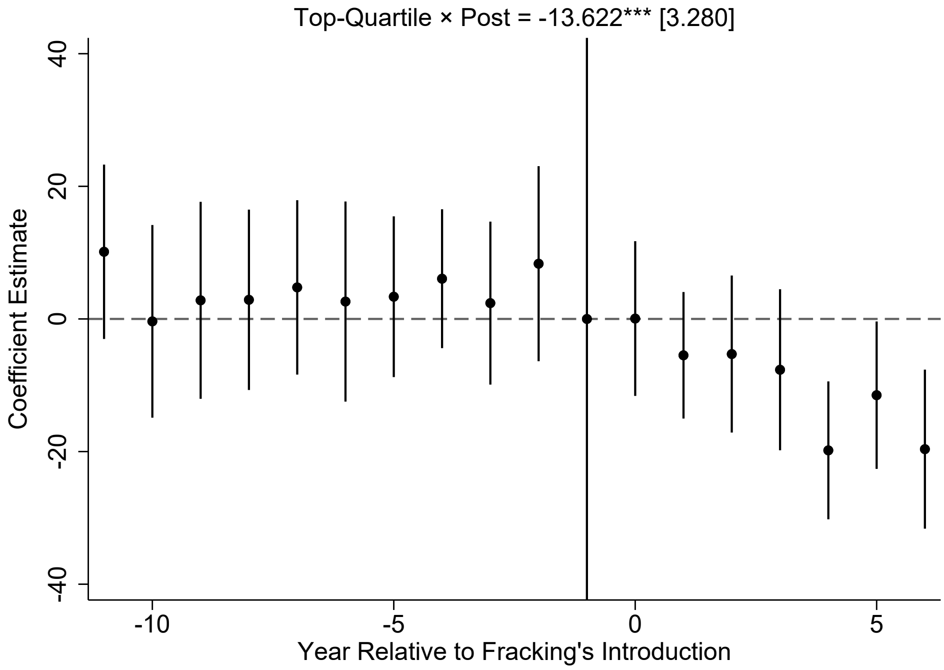
Panel C: Women



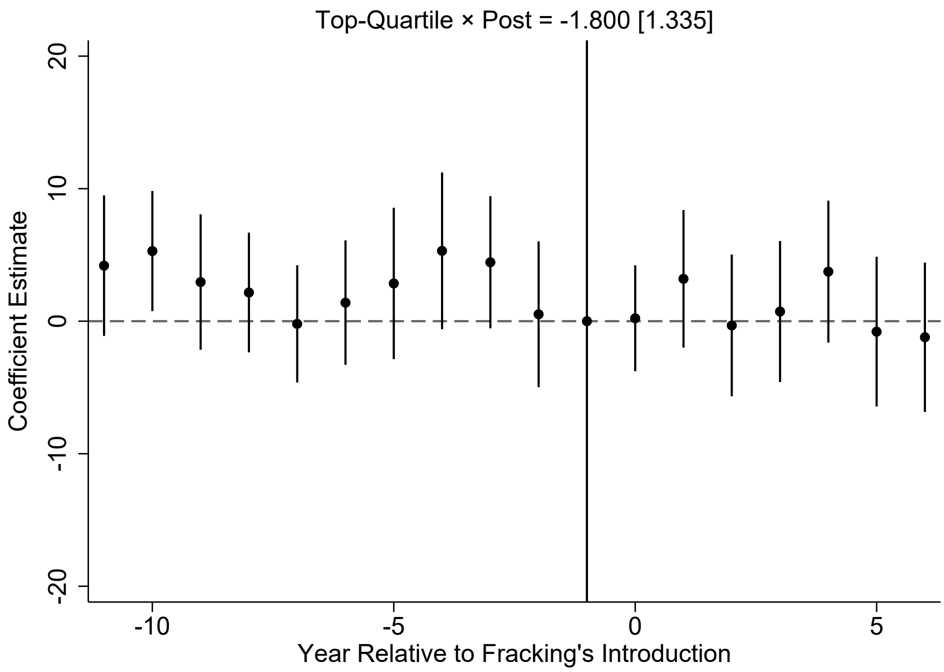
*Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the death rate per 100,000 people, using contemporaneous populations. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Each regression includes 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: Internal Causes of Death



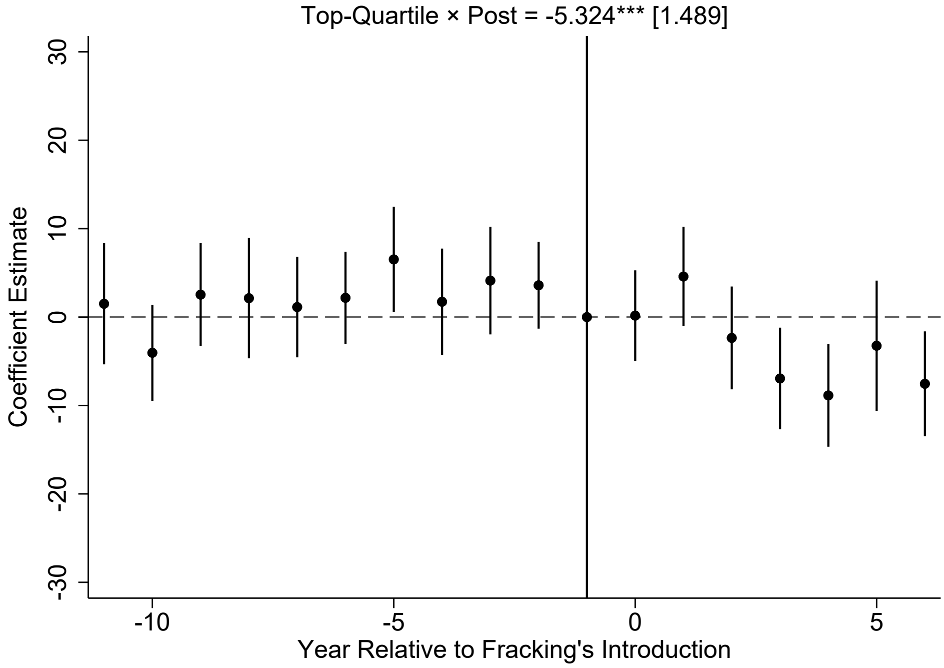
Panel B: External Causes of Death



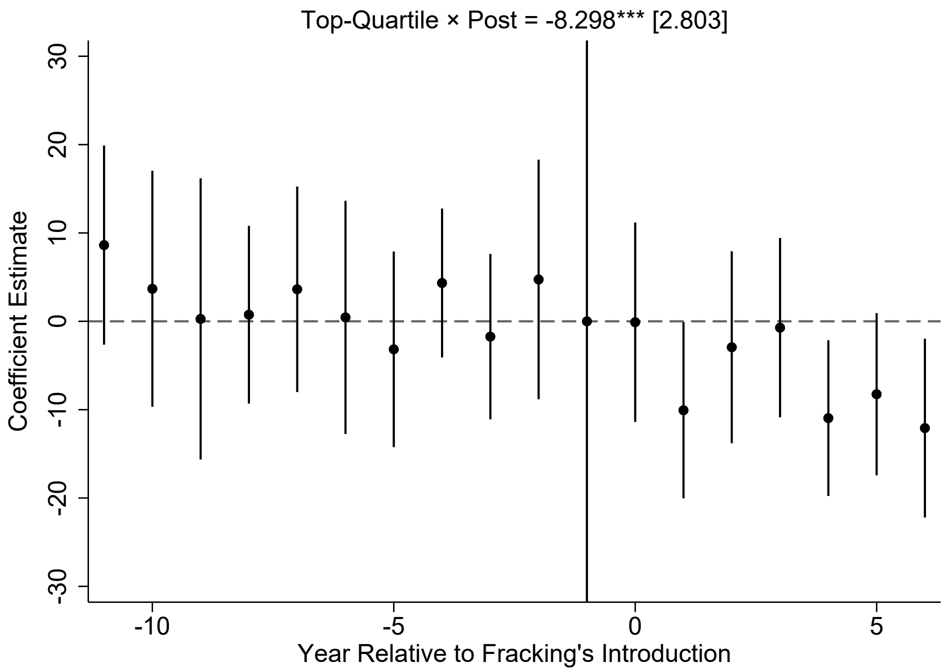
Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) for the balanced set of event-years. Dependent variables are the crude death rate per 100,000 individuals of working age. Panel A reports internal deaths and Panel B reports external deaths. 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. 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% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variables are the crude death per 100,000 individuals of working age for cardiovascular (Panel A) and non-cardiovascular internal (Panel B) mortality. 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. Standard errors are adjusted for clustering at the county level.

VIII Tables

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

	(1) Top-Quartile County	(2) Other Shale Play County	(3) Within Play Difference
% 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]
% White	91.04	90.88	-0.56
	(10.16)	(10.17)	[0.72]
% 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]
% 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]
Median Household Income	30532.81	29815.35	-111.46
	(7878.33)	(6442.70)	[597.67]
Age-Adjusted Death Rate	906.23	916.07	-2.60
	(146.75)	(124.18)	[15.17]
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 brackets.

Table 2: Working-Age Overall Mortality Rates by Gender

Panel A: Overall			
	(1)	(2)	(3)
Top-Quartile × Post	-9.466	-14.701***	-15.422***
	[6.8753]	[5.5817]	[4.1062]
Controls	No	Base	All
Observations	9,341	9,341	9,341
Outcome Mean	438.35	438.35	438.35
R^2	0.867	0.873	0.881
Panel B: Men			
	(1)	(2)	(3)
Top-Quartile × Post	-8.962	-16.089**	-16.988***
	[7.8050]	[6.6549]	[5.2032]
Controls	No	Base	All
Observations	9,341	9,341	9,341
Outcome Mean	542.48	542.48	542.48
R^2	0.823	0.830	0.837
Panel C: Women			
	(1)	(2)	(3)
Top-Quartile × Post	-10.527	-13.677**	-14.094***
	[6.8750]	[5.4459]	[4.0830]
Controls	No	Base	All
Observations	9,341	9,341	9,341
Outcome Mean	333.78	333.78	333.78
R^2	0.745	0.751	0.761

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. Dependent variables are the crude death rate per 100,000 individuals. Panel A reports uses death rates for both men and women, Panel B for men, and Panel C for women. Column (1) uses no controls, Column (2) adds controls for baseline socioeconomic factors (percent high school educated, percent manufacturing, and percentage white), and column (3) adds the remaining controls from Table (1), all taken from the 1990 Census. All controls include interactions of a full set of year dummies (excluding 1990), regressions include 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.*

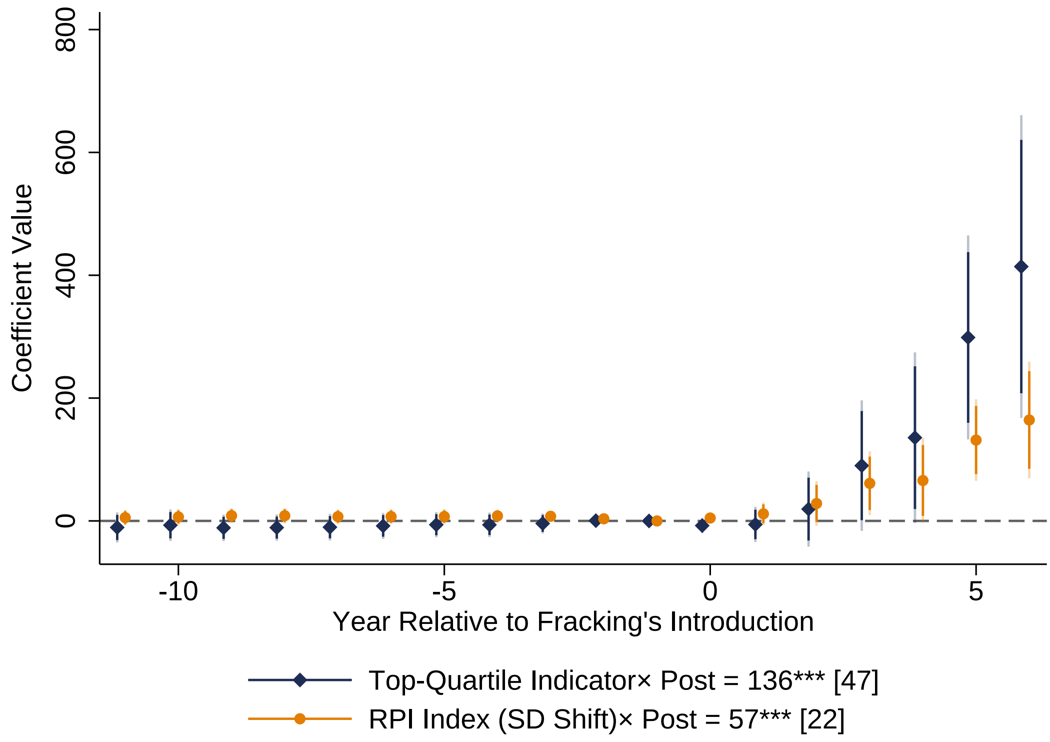
Table 3: Health Insurance Coverage by Gender: Ages 18-64

	(1) Overall	(2) Men	(3) Women
Top-Quartile × Post	0.016***	0.015***	0.018***
	[0.0048]	[0.0051]	[0.0047]
Controls	All	All	All
Outcome Mean	0.81	0.79	0.82
Observations	1,208	1,208	1,208

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. We take all insurance estimates from the Small Area Health Insurance Estimates (SAHIE) Program. The sample is restricted to plays that began fracking after 2008, the first year of data availability. 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

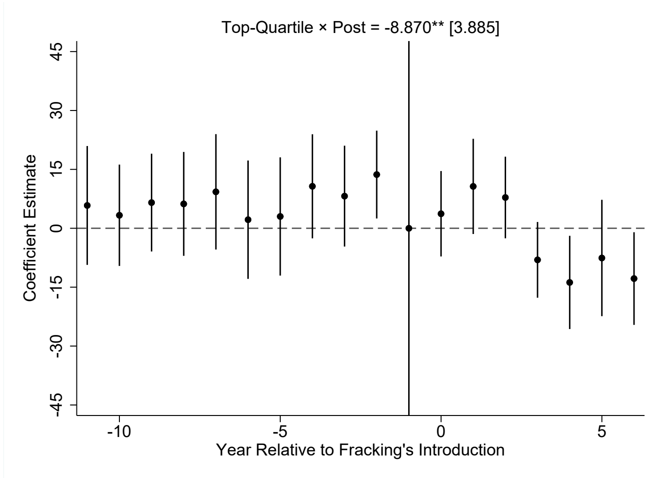
Figure A.1: Horizontal Well Production: Millions of $ of BOE



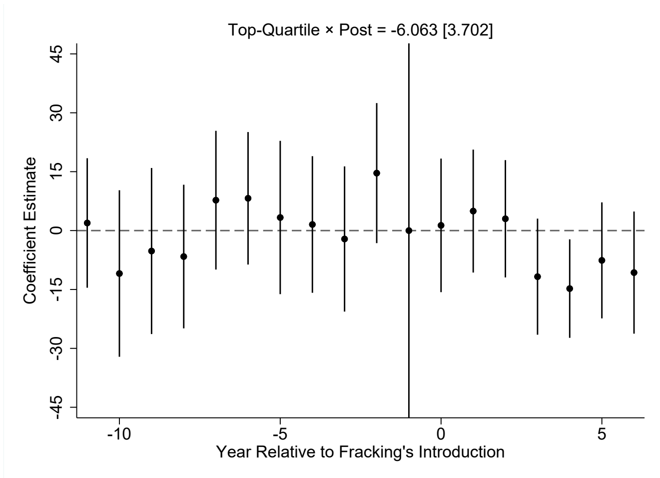
*Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) for the balanced set of event-years. Here, we show coefficients from 2 separate regressions where the coefficient of interest is a different transformation of the RPI. Monthly, well-level production 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 includes 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 100,000

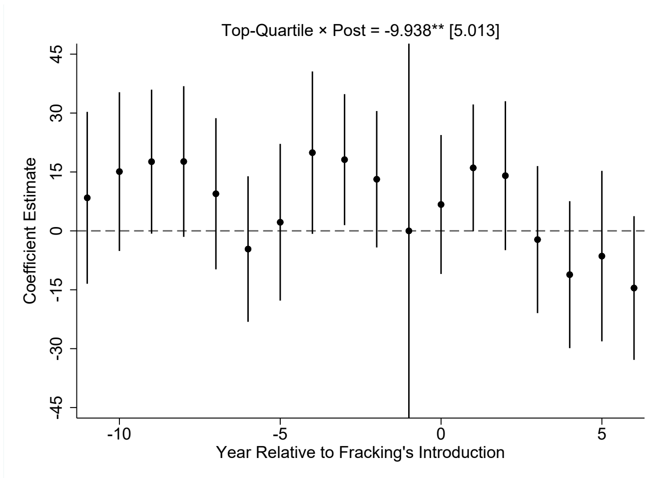
Panel A: Men and Women



Panel B: Men



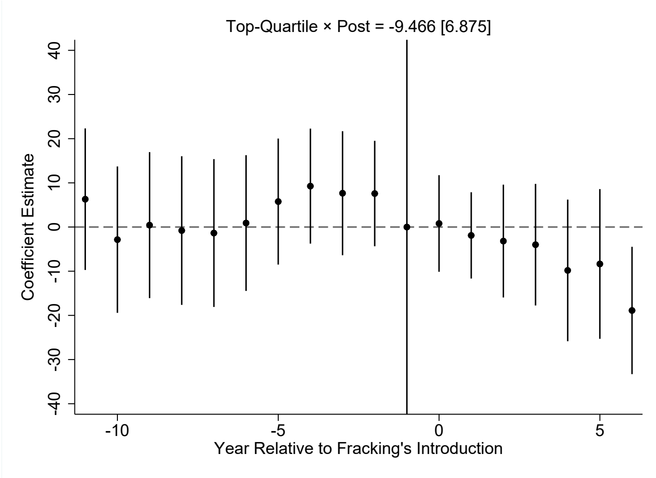
Panel C: Women



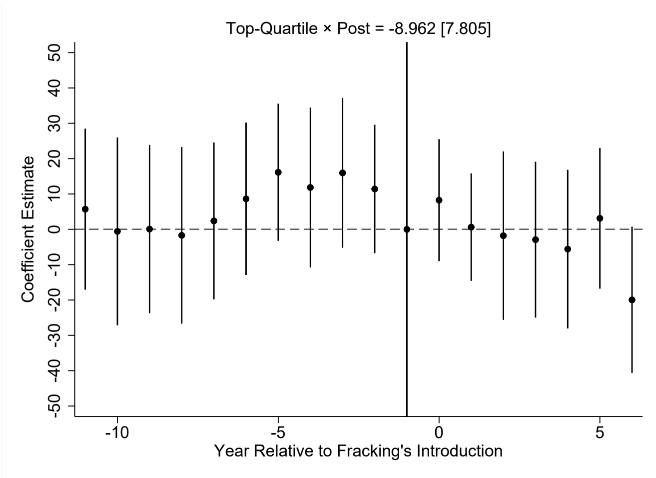
*Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) for the balanced set of event-years. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from 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 includes 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 100,000: No Controls

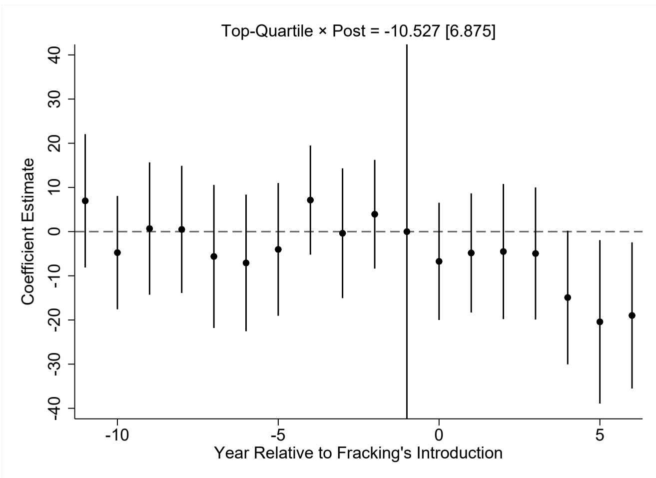
Panel A: Men and Women



Panel B: Men



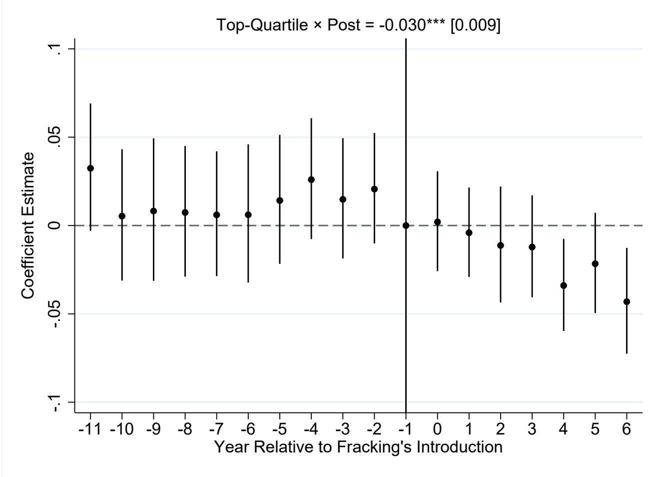
Panel C: Women



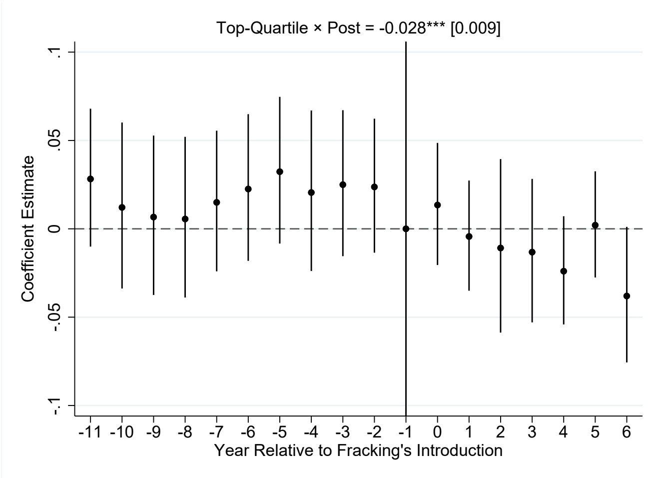
*Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) for the balanced set of event-years. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. The dependent variable is the death rate per 100,000 people, using contemporaneous populations. Each regression uses 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

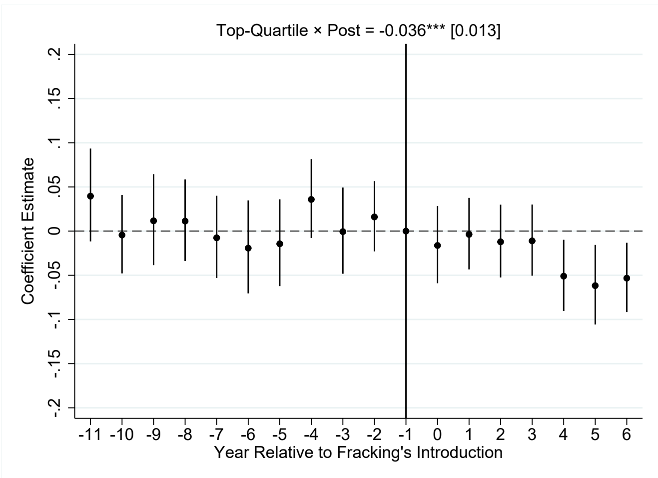
Panel A: Men and Women



Panel B: Men



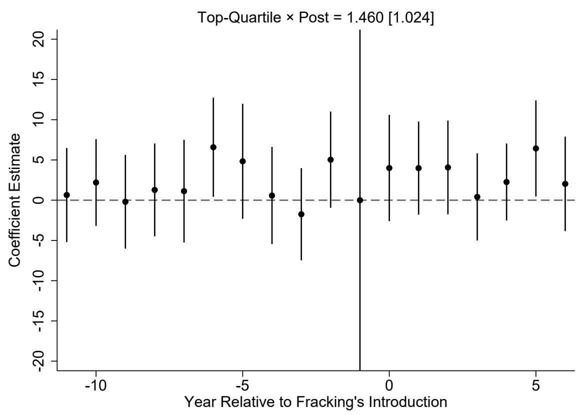
Panel C: Women



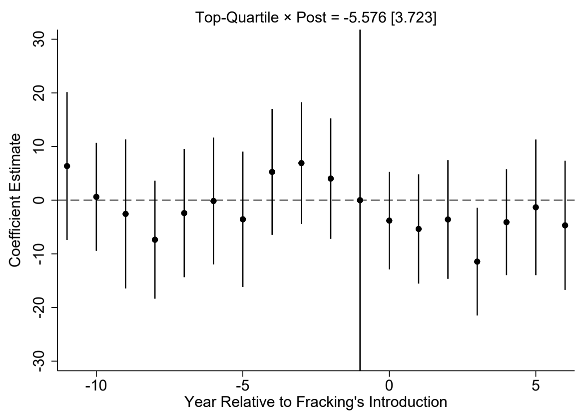
Notes: Each panel reports the point estimates with their associated 95% intervals from a Poisson regression based off of Equation (2) for the balanced set of event-years, using the relevant gender and age population as the exposure variable. We report the transformed coefficients $e^{\hat{\beta}} - 1$ *using endpoint transformation for confidence limits. 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 includes 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 transformed 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

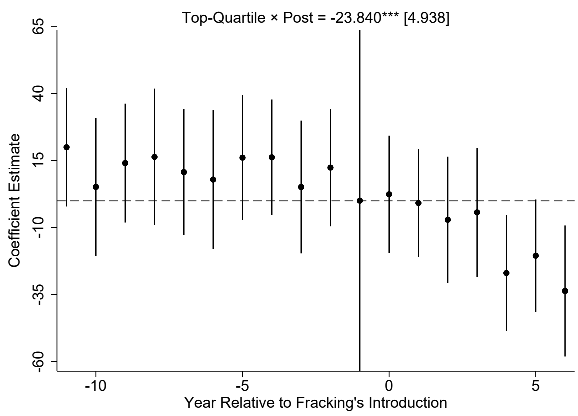
Panel A: Ages: Under 25



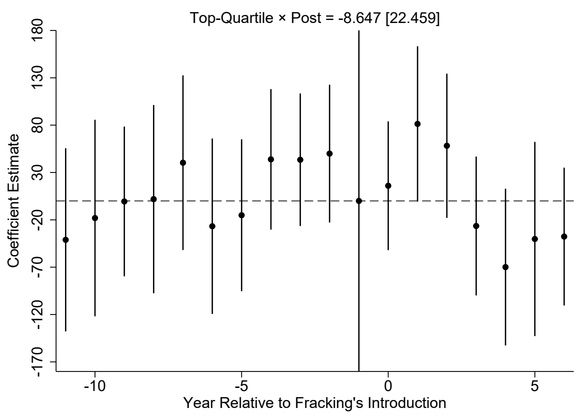
Panel B: Ages: 25-44



Panel C: Ages: 45-64

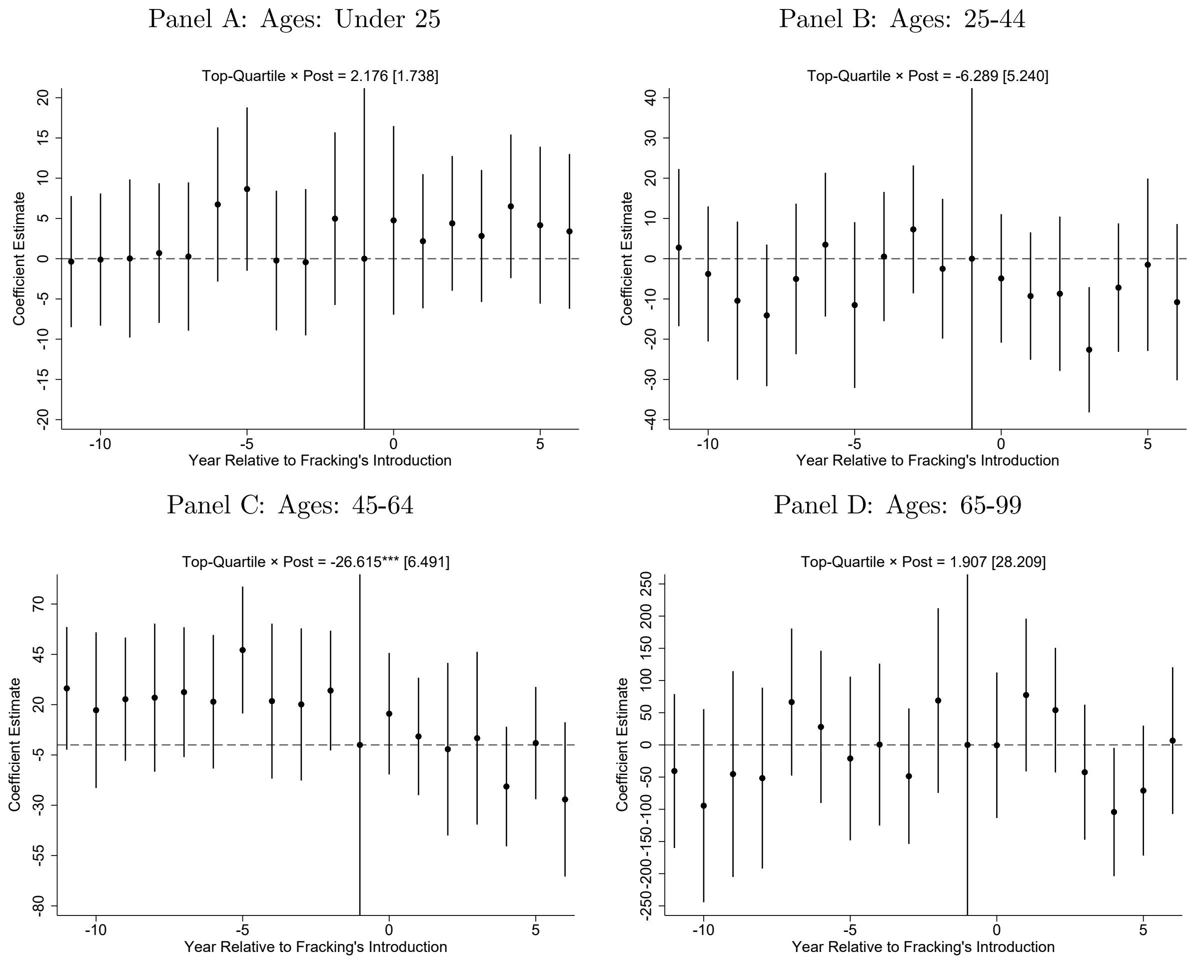


Panel D: Ages: 65-99



*Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the death rate per 100,000 individuals of the given age group. 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 includes 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%.*

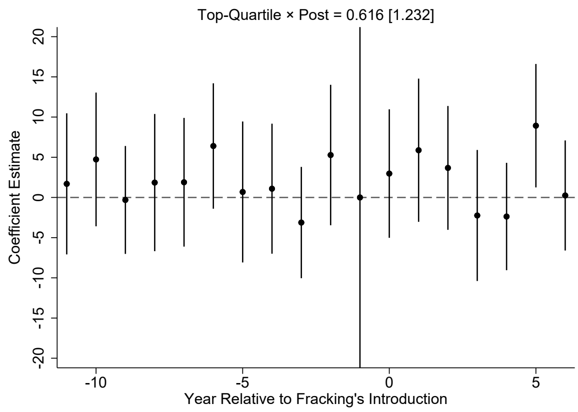
Figure A.6: Overall Mortality Effects by Age: Male



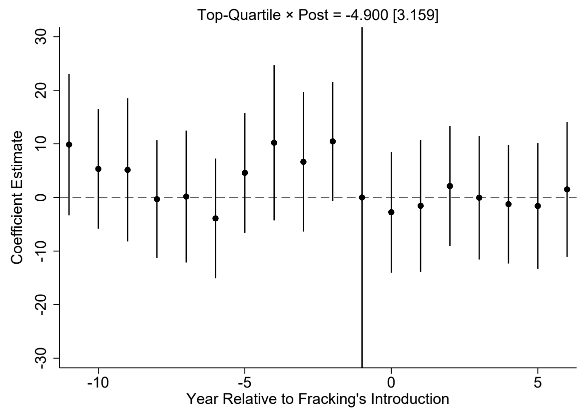
Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) *for the balanced set of event-years. The dependent variable is the death rate per 100,000 individuals of the given age group. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Each regression includes 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

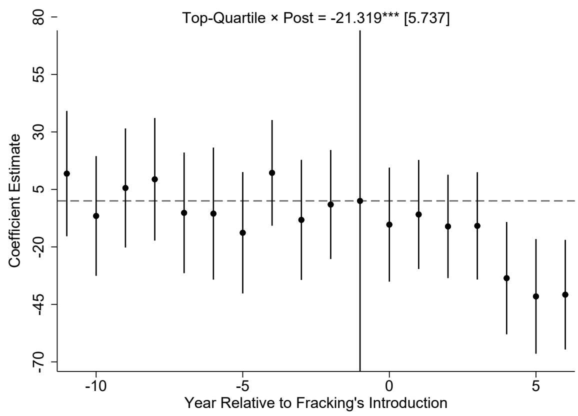
Panel A: Ages: Under 25



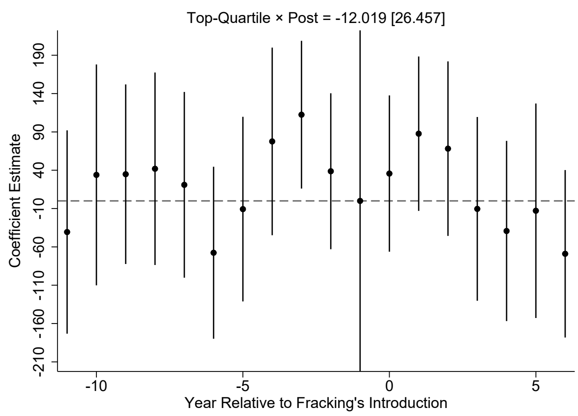
Panel B: Ages: 25-44



Panel C: Ages: 45-64



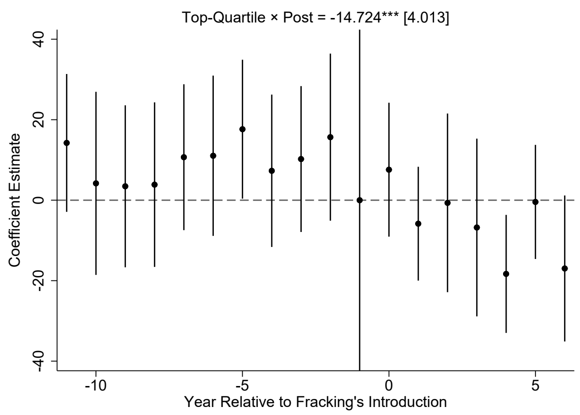
Panel D: Ages: 65-99



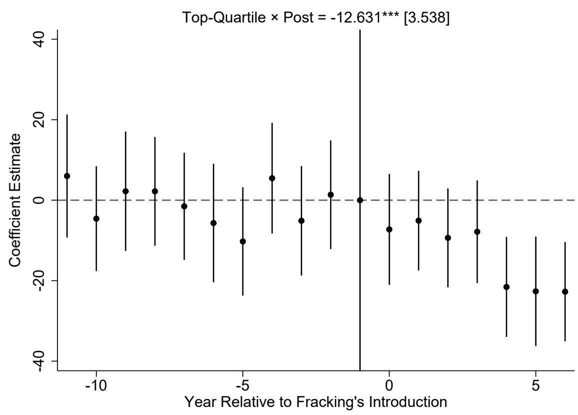
*Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the death rate per 100,000 individuals of the given age group. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Each regression includes 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: Crude Death Rates: Internal and External Causes of Death by Gender

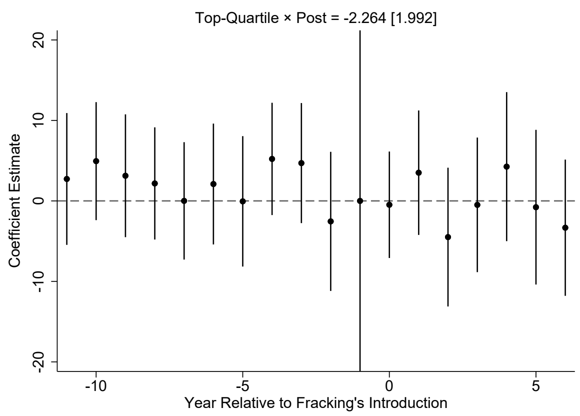
Panel A: Internal Death Rates per 100,000: Male



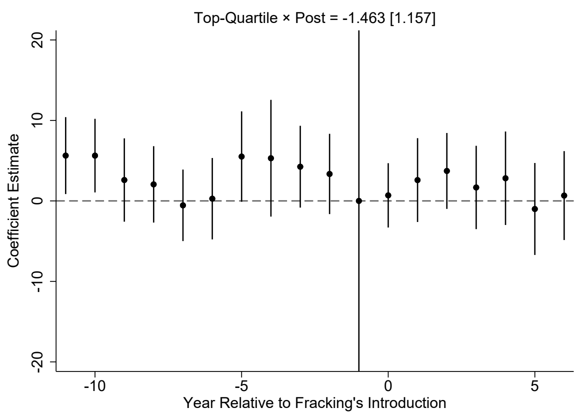
Panel B: Internal Death Rates per 100,000: Female



Panel C: External Death Rates per 100,000: Male



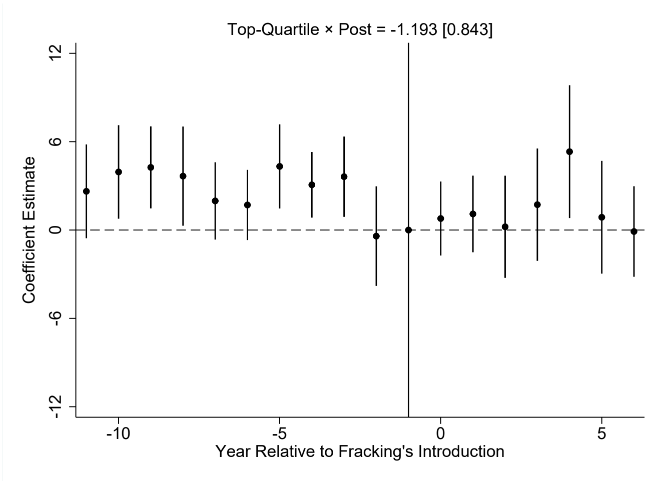
Panel D: External Death Rates per 100,000: Female



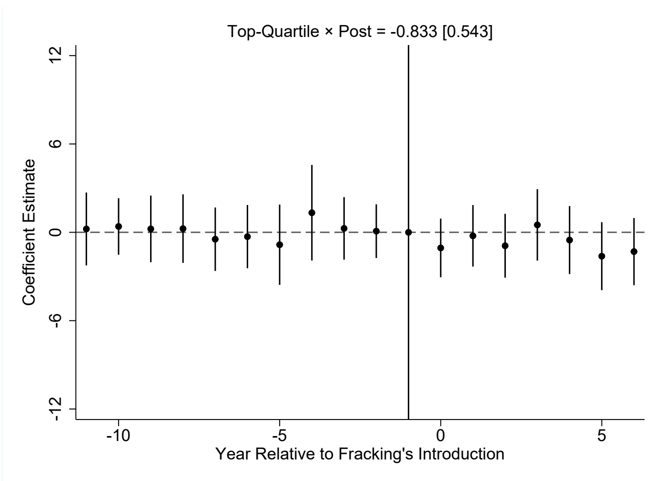
Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) *for the balanced set of event-years. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Dependent variables are the crude death rate per 100,000 individuals of working age. Each regression includes 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.9: External Causes of Death: Deaths of Despair

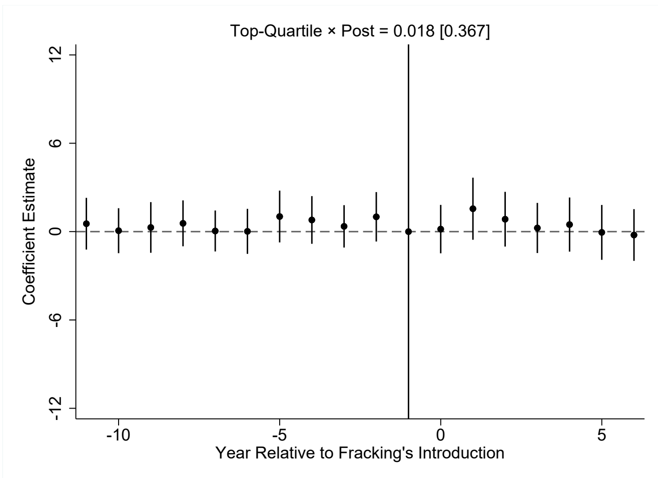
Panel A: Drug Overdoses



Panel B: Suicides



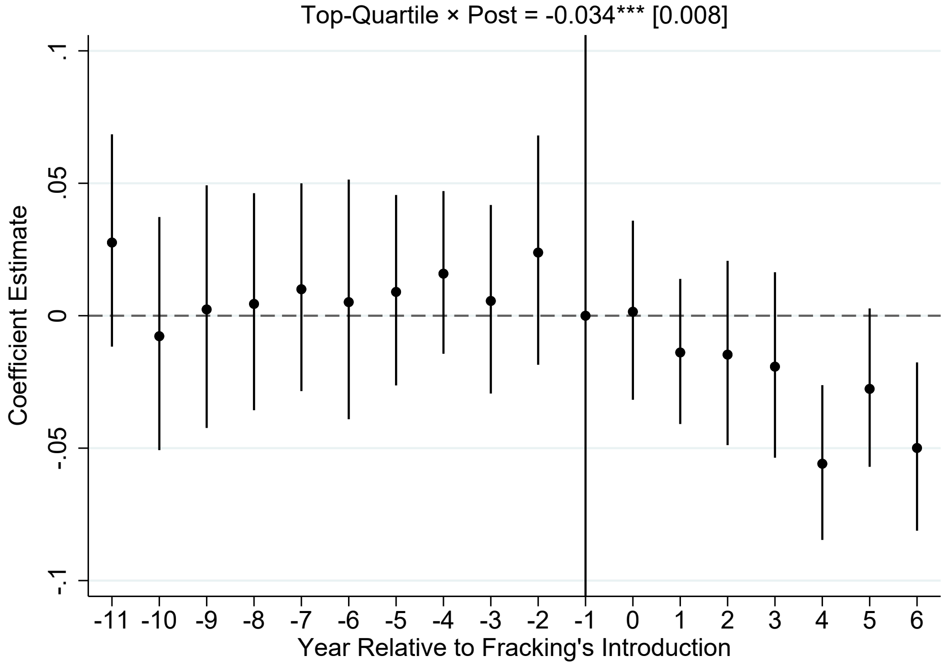
Panel C: Alcohol Related



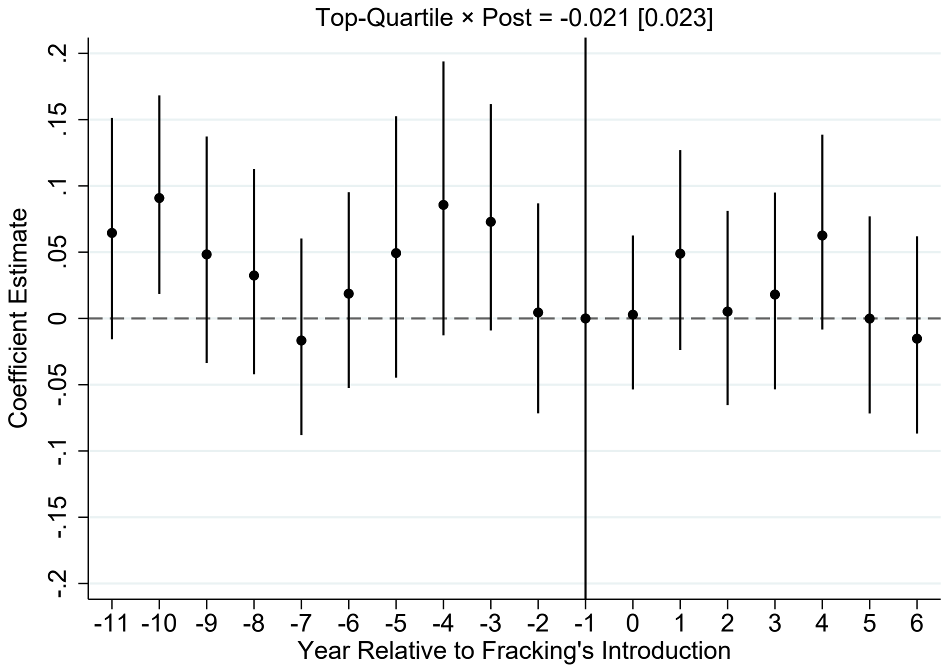
Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the death rate per 100,000 people of drug overdose (Panel A), suicide (Panel B), and alcohol-related (Panel C) mortality. We take 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. Standard errors are adjusted for clustering at the county level.

Figure A.10: Poisson Regression: Internal vs. External Causes of Death (Ages 25-64)

Panel A: Poisson Regression: Internal Causes of Death



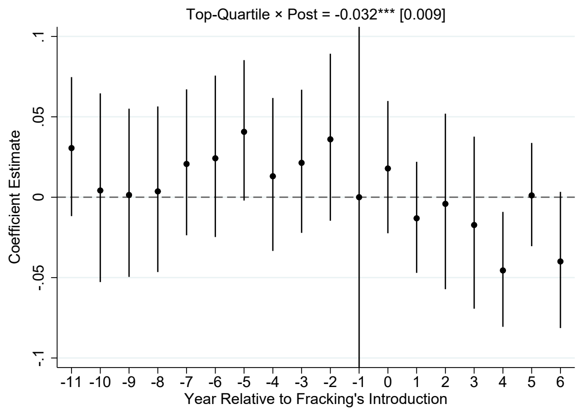
Panel B: Poisson Regression: External Causes of Death



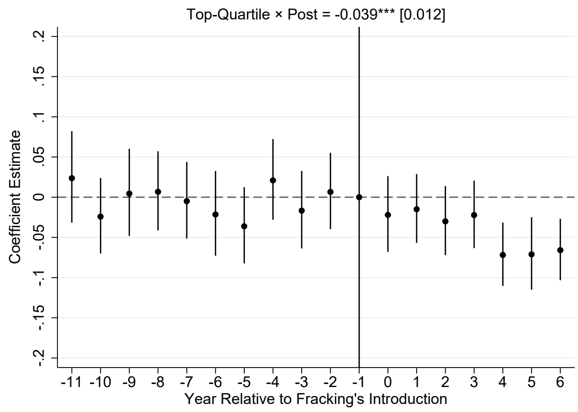
Notes: Each panel reports the point estimates with their associated 95% intervals from a Poisson regression based off of Equation (2) for the balanced set of event-years, using the relevant gender and age population as the exposure variable. We report the transformed coefficients $e^{\hat{\beta}} - 1$ using endpoint transformation for confidence limits. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. 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.11: Poisson Regressions: Internal and External Causes of Death by Gender

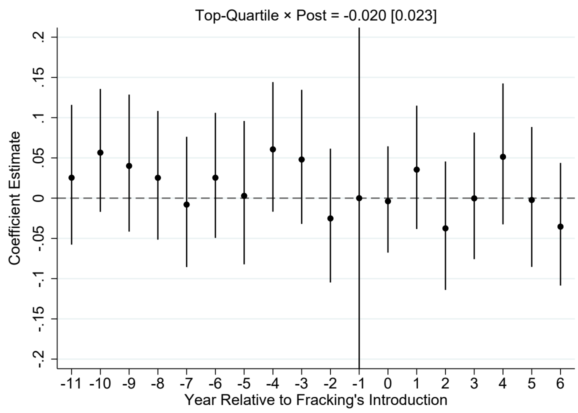
Panel A: Internal Deaths (Male)



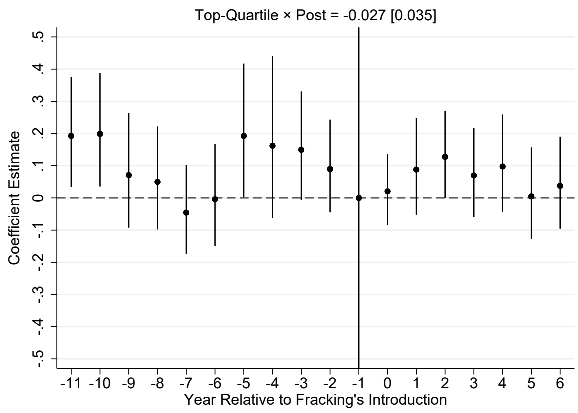
Panel B: Internal Deaths (Female)



Panel C: External Deaths (Male)



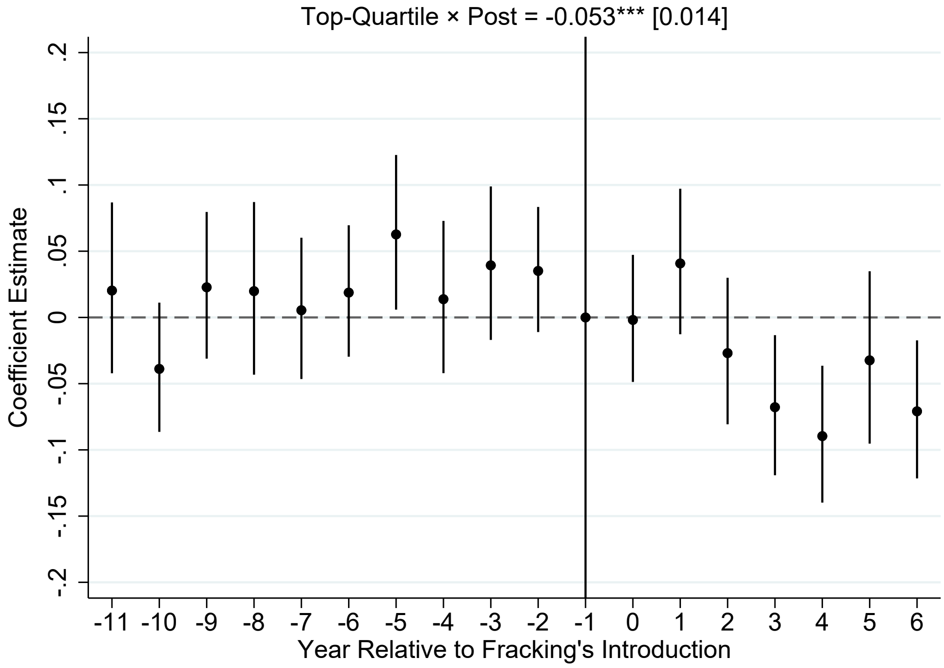
Panel D: External Deaths (Female)



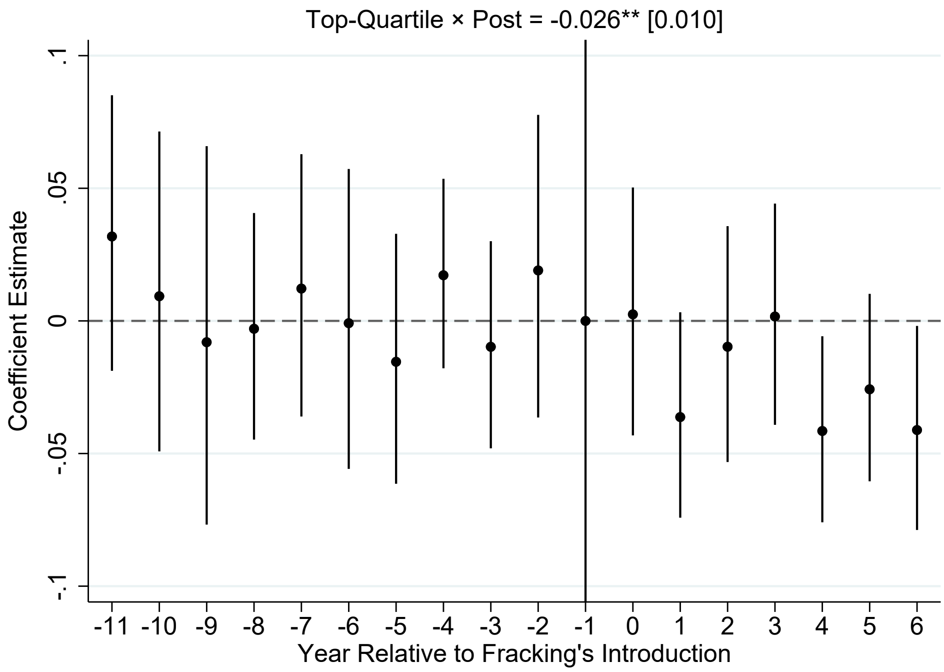
Notes: Each panel reports the point estimates with their associated 95% intervals from a Poisson regression based off of Equation (2) for the balanced set of event-years, using the relevant gender and age population as the exposure variable. We report the transformed coefficients $e^{\hat{\beta}} - 1$ *using endpoint transformation for confidence limits. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. 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.12: Poisson Regression: Cardiovascular vs. Other Internal Causes of Death (Ages 25-64)

Panel A: Cardiovascular Mortality



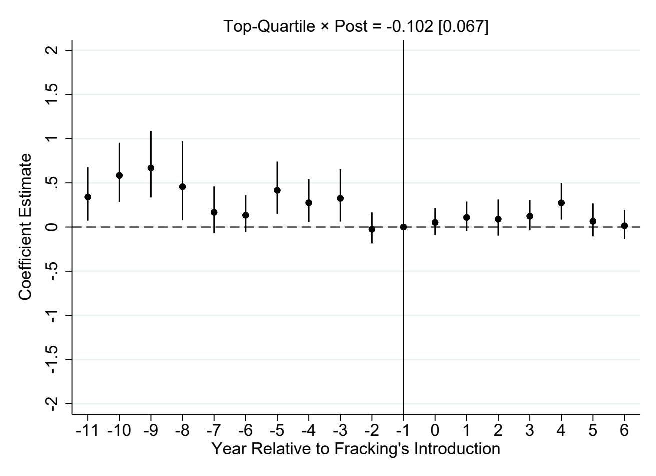
Panel B: Other Internal Mortality



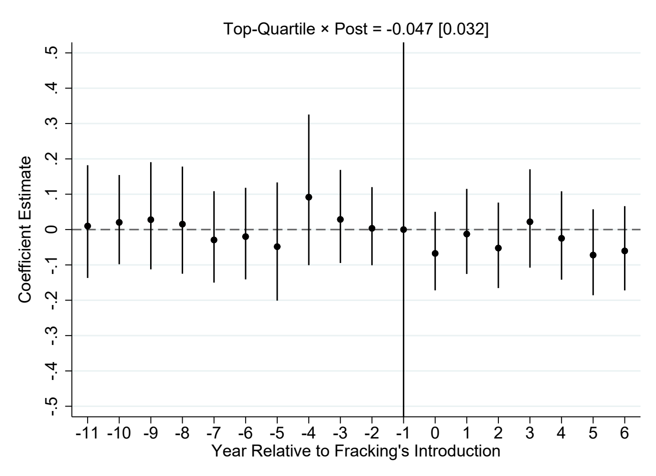
Notes: Each panel reports the point estimates with their associated 95% intervals from a Poisson regression based off of Equation (2) for the balanced set of event-years, using the relevant gender and age population as the exposure variable. We report the transformed coefficients $e^{\hat{\beta}} - 1$ *using endpoint transformation for confidence limits. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. 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.13: Poisson Regressions: Deaths of Despair

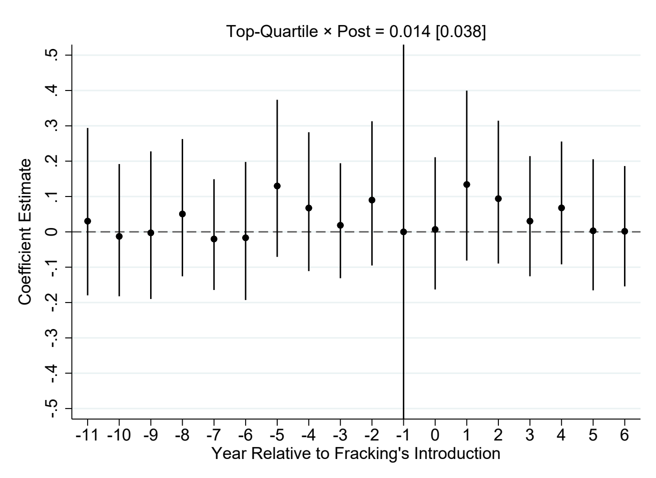
Panel A: Drug Overdoses



Panel B: Suicides



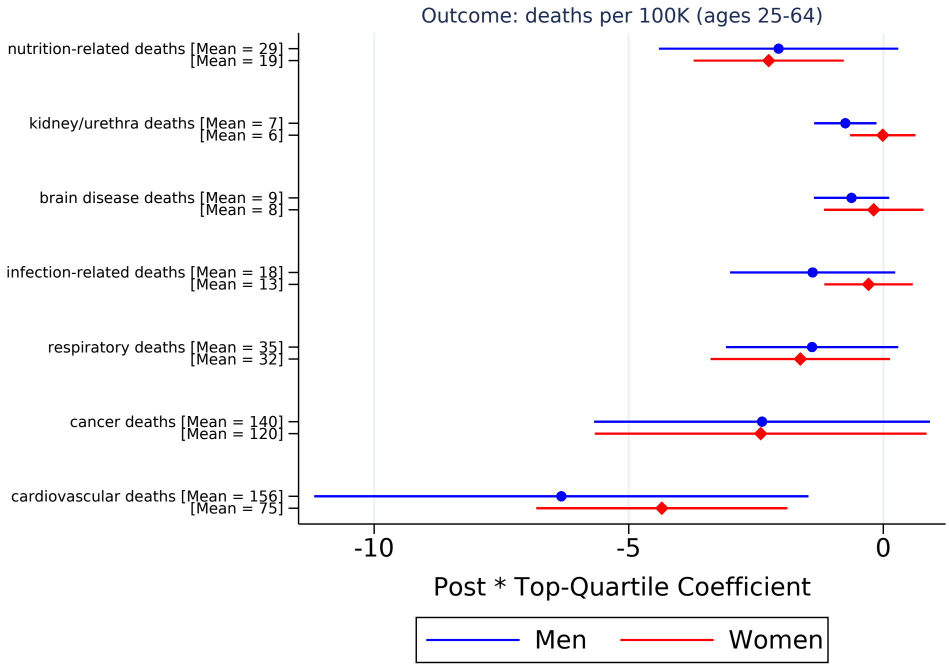
Panel C: Alcohol Related



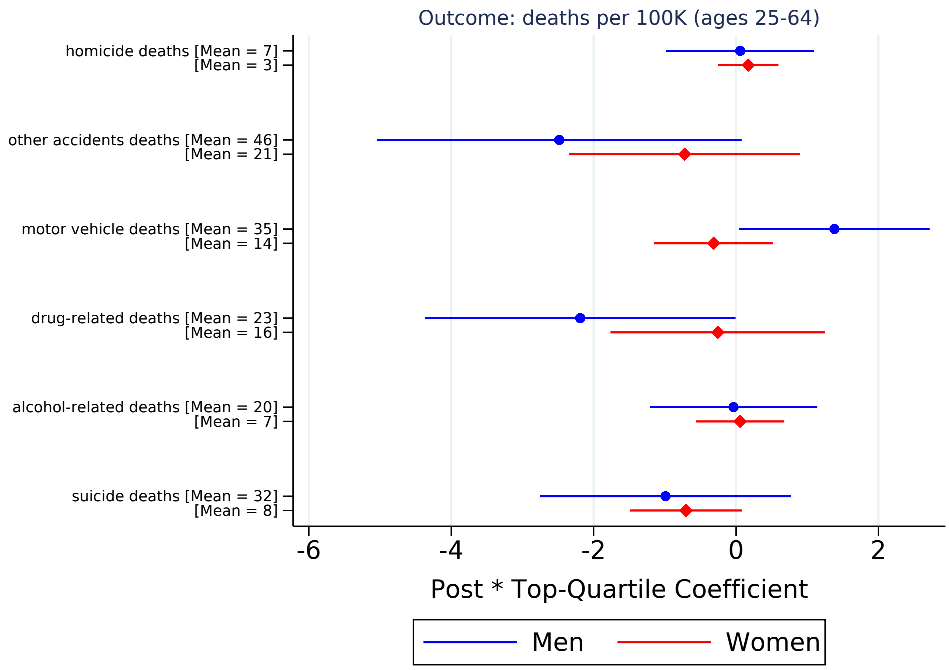
Notes: Each panel reports the point estimates with their associated 95% intervals from a Poisson regression based off of Equation (2) for the balanced set of event-years, using the relevant gender and age population as the exposure variable. We report the transformed coefficients $e^{\hat{\beta}} - 1$ *using endpoint transformation for confidence limits. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. 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: Internal and External Causes of Death: Differences by Gender

Panel A: Internal Causes

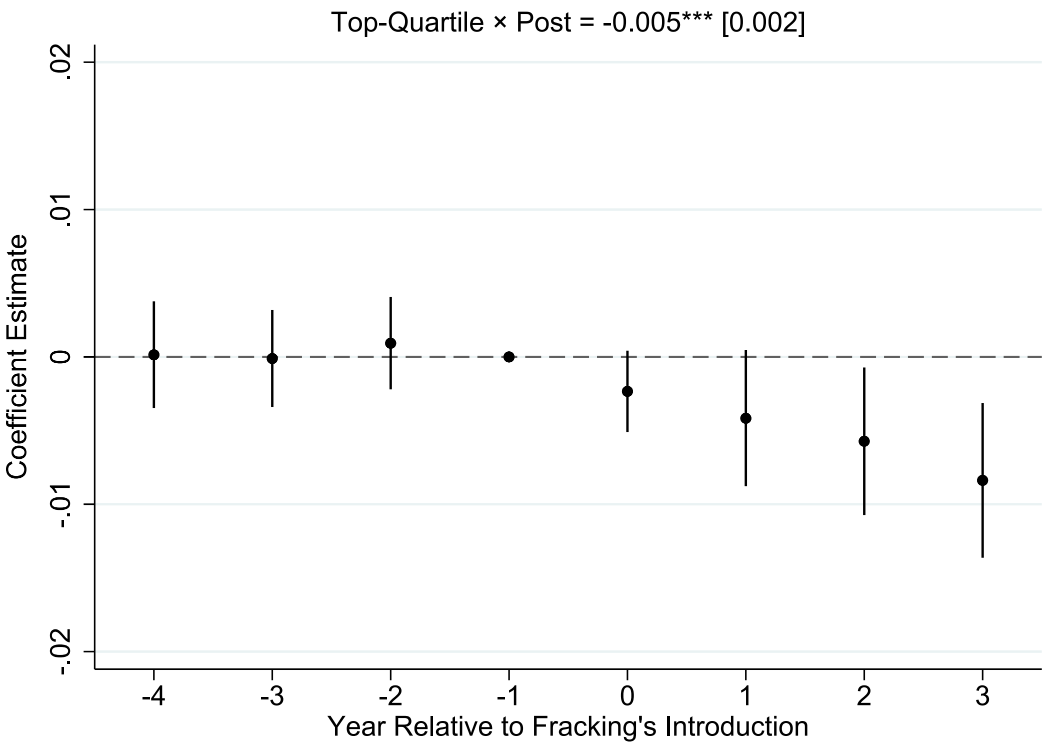


Panel B: External Causes



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 bars represent the associated 95% confidence intervals. 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.

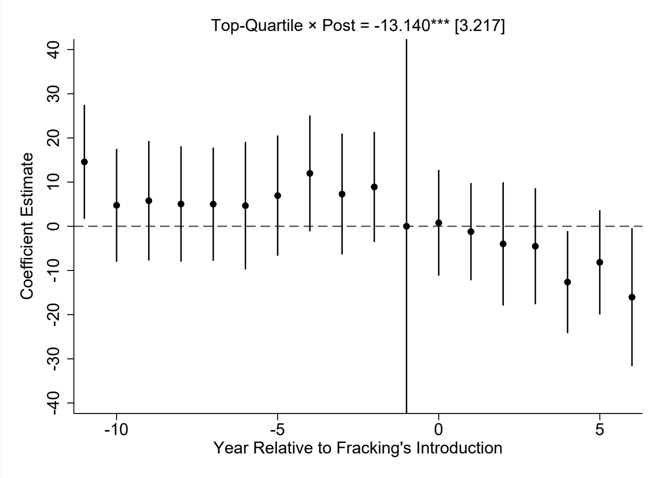
Figure A.15: SNAP Benefits



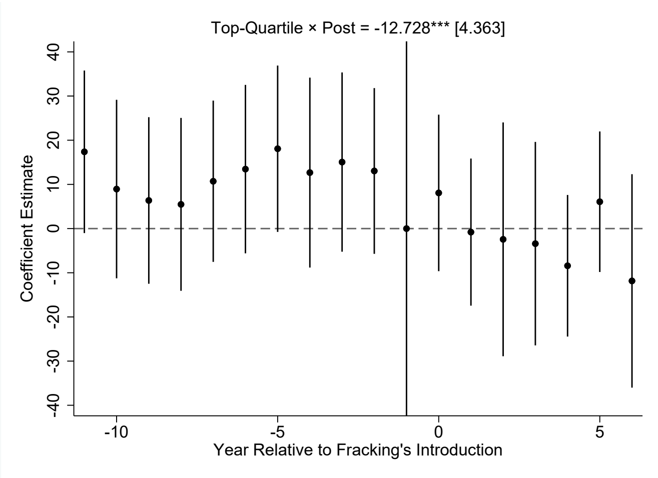
*Notes: Figure reports the point estimates with their associated 95% confidence intervals from Equation (2) for a balanced set of event-years. The dependent variable is the share of a county's population receiving Supplemental Nutrition Assistance Program (SNAP) benefits. Data is taken from the USDA's SNAP Data System. We take population counts for the same time period from SEER. We restrict our sample to plays that initiated fracking before 2009 to maintain a balanced panel, with SNAP data spanning 1997-2011. All regressions were estimated using interactions of a full set of year dummies with time-invariant county characteristics from the 1990 census. Standard errors are adjusted for clustering at the county level *** Significance 1%, ** 5%, * 10%.*

Figure A.16: Overall Mortality: Controlling for Compositional Changes

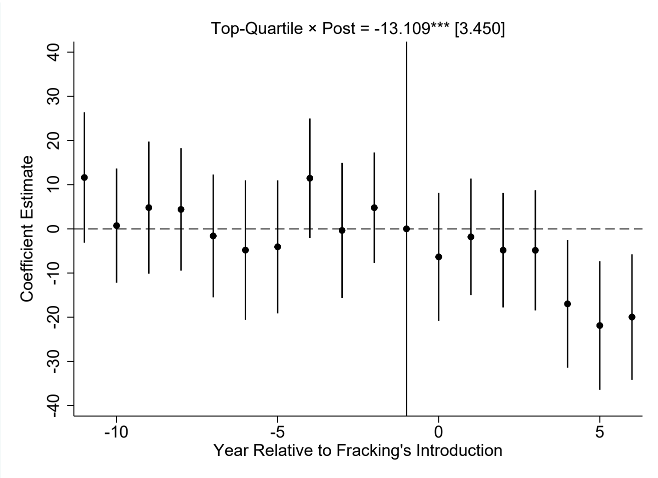
Panel A: Men and Women



Panel B: Men



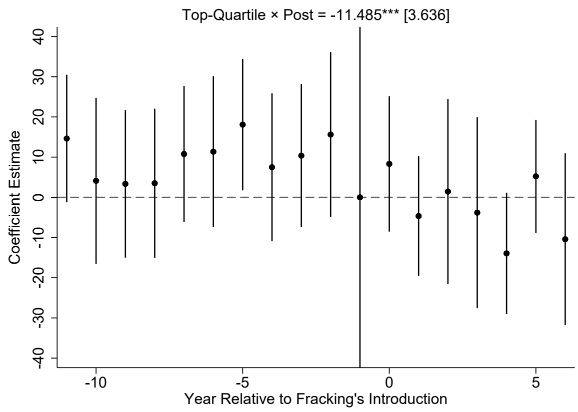
Panel C: Women



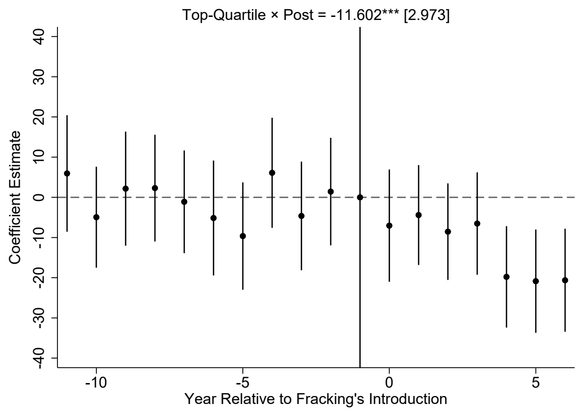
*Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the overall death rate per 100,000 individuals, and we control for the contemporaneous shares of relevant gender population belonging 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 includes 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.17: Internal and External Causes of Death: Controlling for Compositional Changes

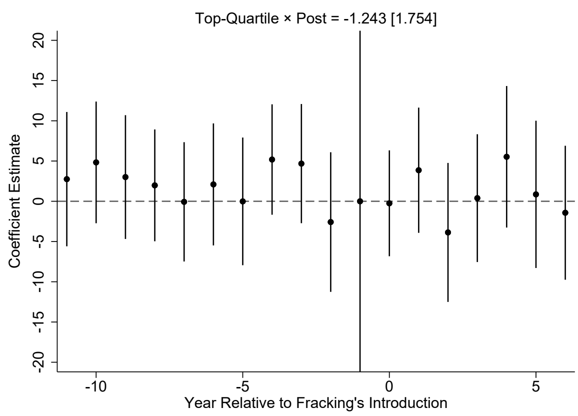
Panel A: Internal Deaths (Male)



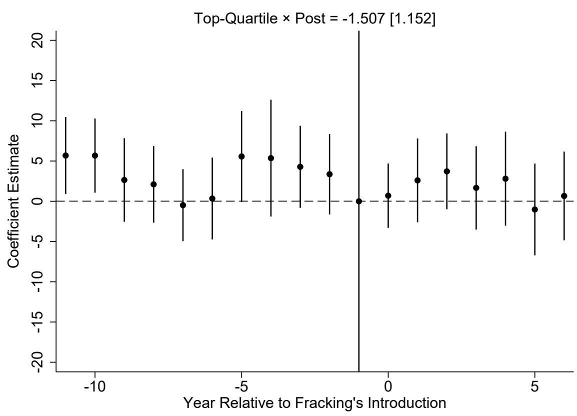
Panel B: Internal Deaths (Female)



Panel C: External Deaths (Male)



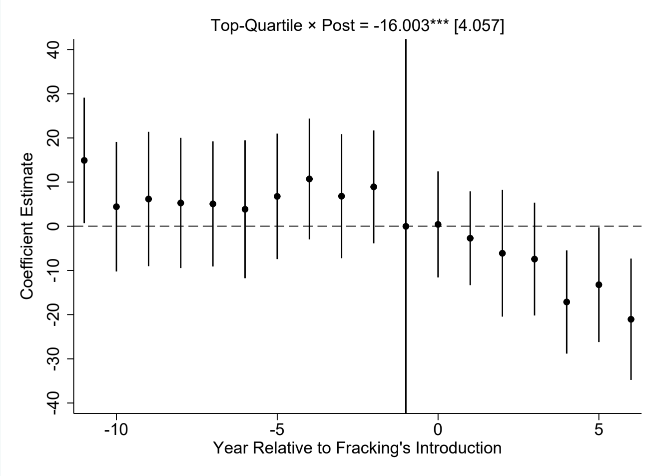
Panel D: External Deaths (Female)



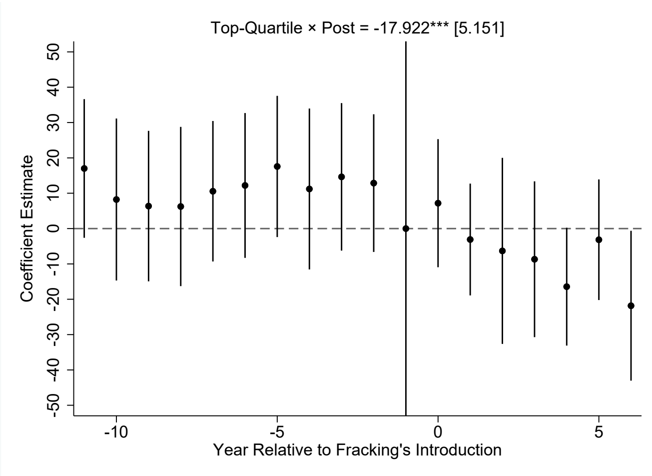
*Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) for the balanced set of event-years. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Our primary outcome is the death rate per 100,000 individuals, 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 includes 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.18: Overall Mortality by Gender: No Bakken Shale Play

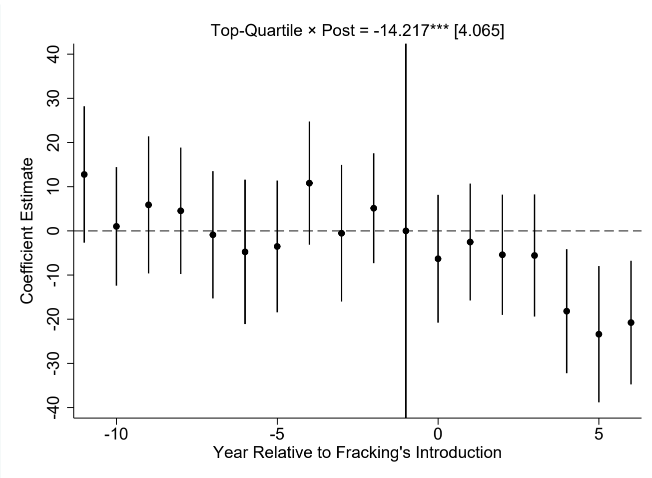
Panel A: Men and Women



Panel B: Men



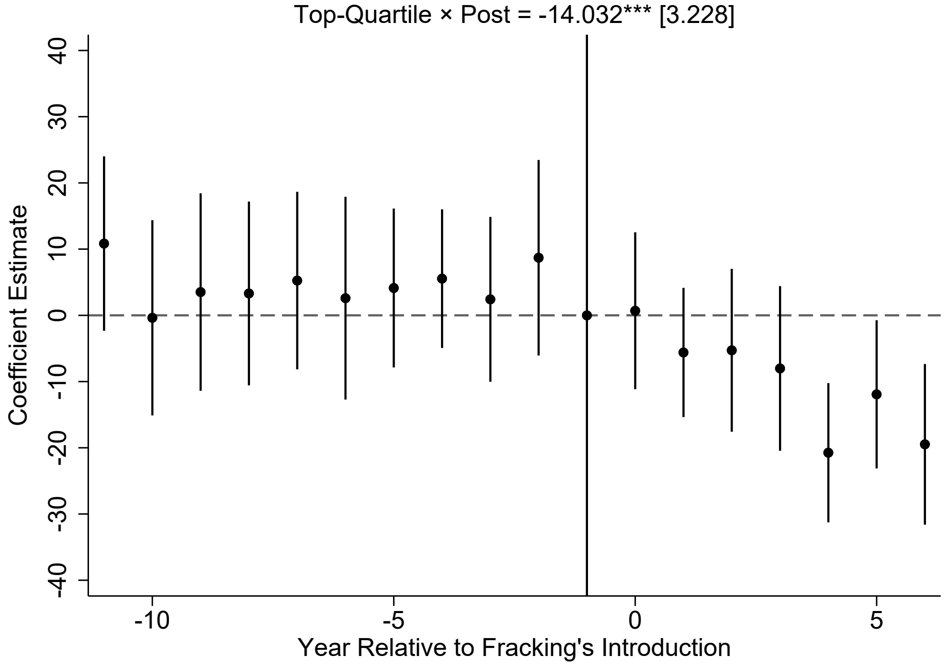
Panel C: Women



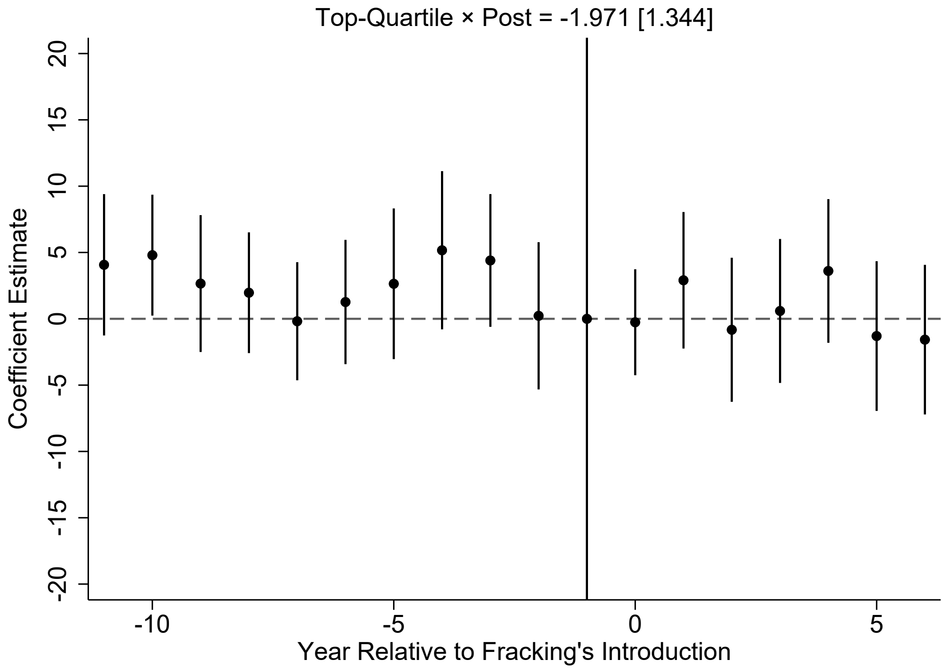
*Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the death rate per 100,000 people, using contemporaneous populations. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Each regression includes 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 drop observations from the Bakken shale play. 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.19: Internal vs. External Causes of Death: No Bakken Shale Play

Panel A: Internal Causes of Death

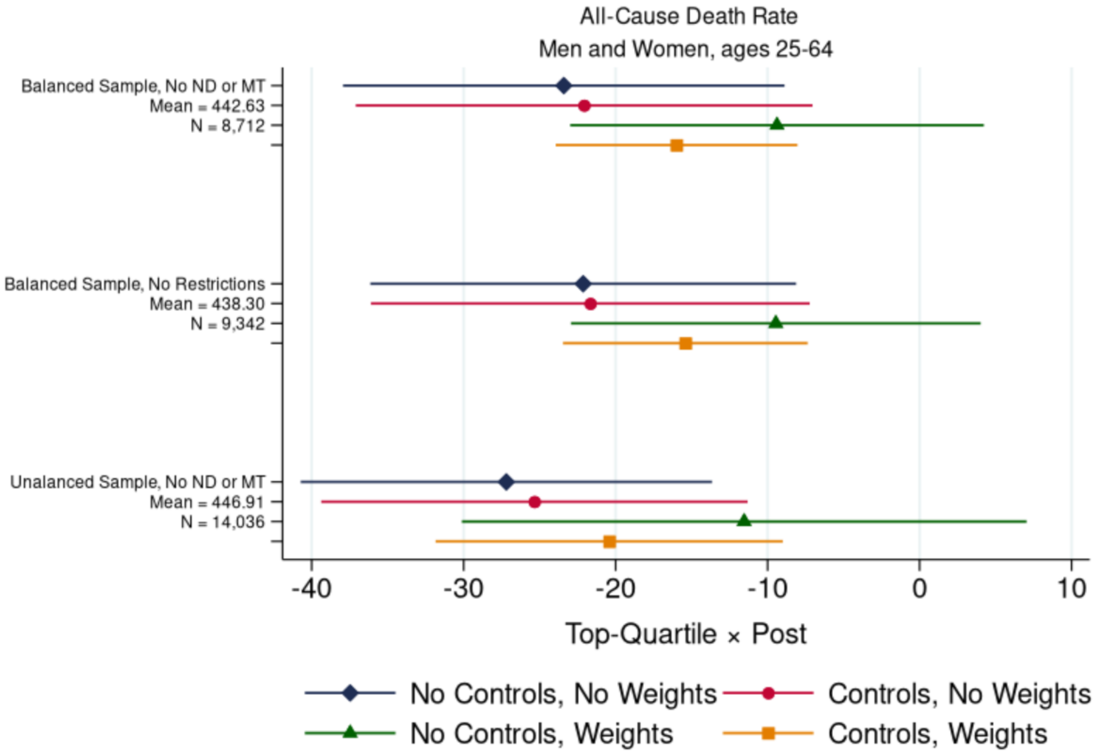


Panel B: External Causes of Death



Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) for the balanced set of event-years. Dependent variables are the crude death rate per 100,000 individuals of working age. Panel A reports internal deaths and Panel B reports external deaths. 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 drop observations from the Bakken shale play. Standard errors are adjusted for clustering at the county level.

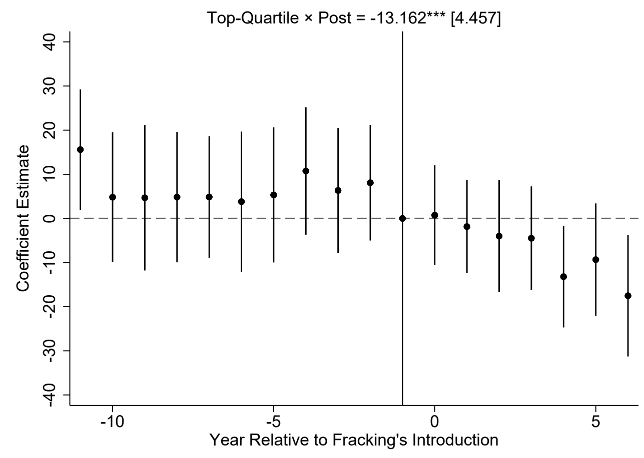
Figure A.20: Men/Women Working-Age Mortality Robustness



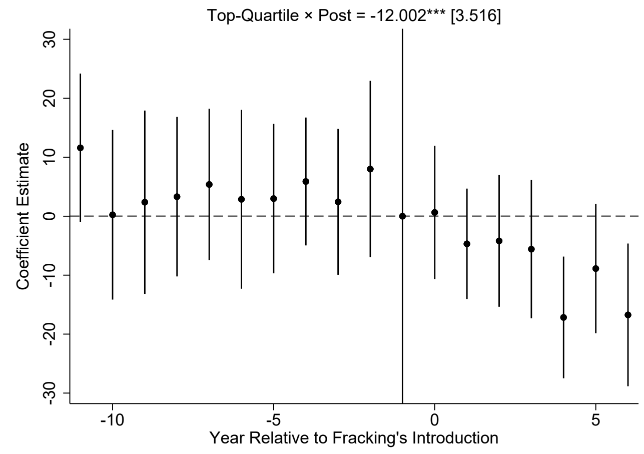
Notes:We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Death categories are taken from Stevens et al. (2015), and represent consistent definitions across ICD-9 and ICD-10 cause of death codes. 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.

Figure A.21: Mortality: Geographic/Place Based Factors

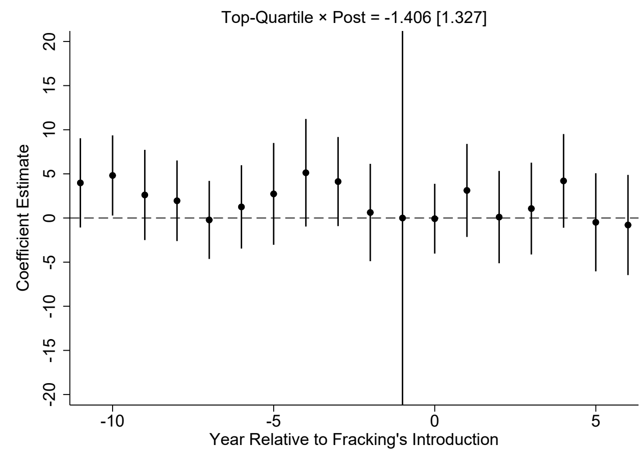
Panel A: Overall Mortality



Panel B: Internal Causes of Death



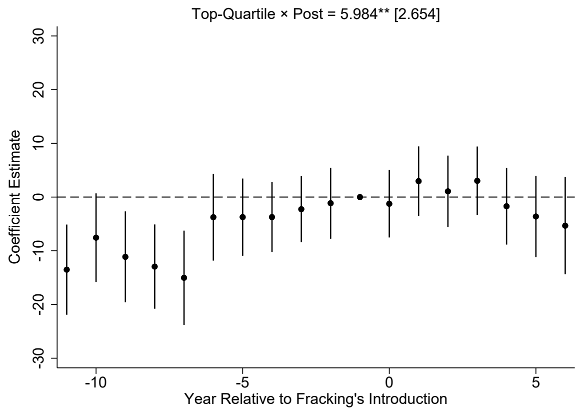
Panel C: External Causes of Death



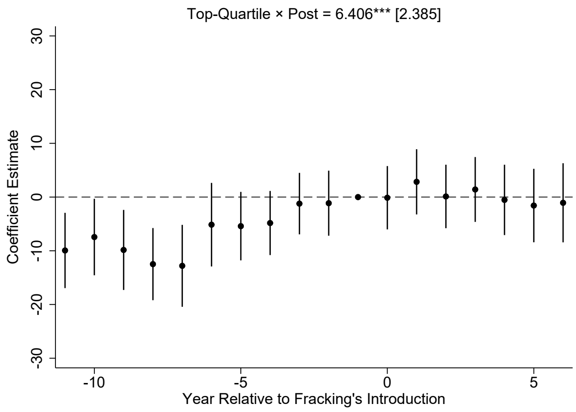
*Notes: Each panel reports the point estimates with their associated 95% confidence intervals from Equation (2) for the balanced set of event-years. The dependent variable is the death rate per 100,000 people, using contemporaneous populations. We take all death count data from the CDC's National Center for Health Statistics from 1990-2018. We take population counts for the same time period from SEER. Each regression includes 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. We additionally control for the log of the contemporaneous working-aged population as well as the baseline 1990 age-adjusted mortality rate interacted with year fixed effects. 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.22: Potential Confounding: Comparing Shale to No-Shale Counties within State

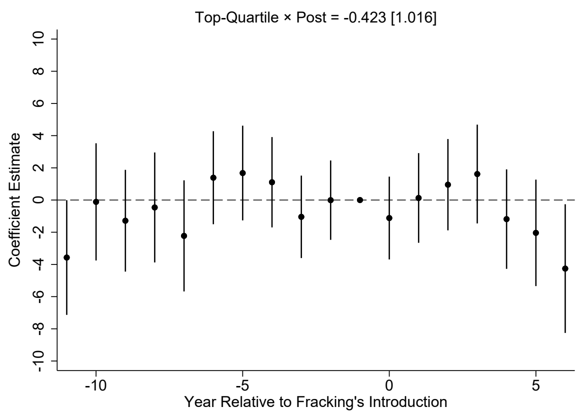
Panel A: Overall Mortality



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% confidence intervals. We take population counts for the same time period from SEER. Dependent variables are the crude death rates of the relevant cause of death, controlling for the relevant contemporaneous population. Each regression includes 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)

	(1) Any Shale Play	(2) No Shale Play	(3) 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: Age - Specific Mortality Rates by Gender

	Panel A: Overall			
	(1)	(2)	(3)	(4)
	0-24	25-44	45-64	65+
Top-Quartile × Post	1.460	-5.576	-23.840***	-8.647
	[1.0236]	[3.7229]	[4.9379]	[22.4589]
Controls	All	All	All	All
Observations	9,342	9,342	9,342	9,341
Outcome Mean	79.55	178.18	694.88	5082.60
	Panel B: Men			
	(1)	(2)	(3)	(4)
	0-24	25-44	45-64	65+
Top-Quartile × Post	2.176	-6.289	-26.615***	1.907
	[1.7377]	[5.2397]	[6.4912]	[28.2087]
Controls	All	All	All	All
Observations	9,342	9,342	9,342	9,341
Outcome Mean	99.85	230.68	857.35	5444.59
	Panel C: Women			
	(1)	(2)	(3)	(4)
	0-24	25-44	45-64	65+
Top-Quartile × Post	0.616	-4.900	-21.319***	-12.019
	[1.2323]	[3.1592]	[5.7370]	[26.4568]
Controls	All	All	All	All
Observations	9,342	9,342	9,342	9,341
Outcome Mean	57.96	124.96	533.87	4816.93

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. Dependent variables are the crude death rate per 100,000 individuals. Panel A reports uses death rates for both men and women, Panel B for men, and Panel C for women. All regressions include the full set of controls from Table (1), taken from the 1990 Census. All controls include interactions of a full set of year dummies (excluding 1990), and include 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.*

Table B.3: Changes in Population by Age

	Overall Population				
	(1) Log Pop (25-64)	(2) Log Pop (<25)	(3) Log Pop (25-44)	(4) Log Pop (45-64)	(5) Log Pop (65+)
Top-Quartile × Post	0.012	0.010	0.011	0.015	-0.004
	[0.0092]	[0.0090]	[0.0106]	[0.0121]	[0.0146]
Observations	9,342	9,342	9,342	9,342	9,342

*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. Regressions also include 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.*

Table B.4: Changes in Population by Gender

	Men				Women			
	(1) Log Pop (<25)	(2) Log Pop (25-44)	(3) Log Pop (45-64)	(4) Log Pop (65+)	(5) Log Pop (<25)	(6) Log Pop (25-44)	(7) Log Pop (45-64)	(8) Log Pop (65+)
Top-Quartile × Post	0.010	0.014	0.016	-0.009	0.009	0.009	0.015	-0.001
	[0.0096]	[0.0109]	[0.0112]	[0.0157]	[0.0086]	[0.0108]	[0.0131]	[0.0139]
Observations	9,342	9,342	9,342	9,342	9,342	9,342	9,342	9,342

*** 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. Regressions also include 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.*

Table B.5: 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	0.000	0.001	0.001	-0.003*	0.001	-0.000	0.001	-0.002
	[0.0010]	[0.0019]	[0.0010]	[0.0014]	[0.0012]	[0.0013]	[0.0010]	[0.0015]
Observations	9,342	9,342	9,342	9,342	9,342	9,342	9,342	9,342

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

*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, and include 2000 county-level population weights. Standard errors are adjusted for clustering at the county level.*

Table B.6: 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 omit 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: 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 omit 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: 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 omit 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: Infant Mortality Rates

	Overall	Men	Women
	(1)	(2)	(3)
Top-Quartile × Post	-0.199	-0.314	-0.088
	[0.1559]	[0.2281]	[0.2081]
Controls	YES	YES	YES
Outcome Mean	6.84	7.57	6.15
Observations	9,341	9,341	9,341

*Notes: *** Significance 1%, ** Significance 5%, * Significance 10%. The dependent variable is the infant mortality rate, defined as the number of deaths to children under 1 per 1000 population under one. All columns include interactions of a full set of year dummies (excluding 1990) with time-invariant county characteristics from the 1990 census. Regressions 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 (1)	Any Truck Involved (2)	No Truck Involved (3)
Top-Quartile × Post	1.185***	0.610***	0.575*
	[0.446]	[0.206]	[0.332]
Controls	All	All	All
Outcome Mean	25.28	4.64	20.65
Observations	9,342	9,342	9,342

*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.*