Casualties of the Cold War: Fallout, Irradiated Dairy, and the Mortality Effects of Nuclear Testing

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Abstract

During the Cold War, the United States detonated over one thousand atomic weapons. Prior to 1963, many of these nuclear tests were conducted above ground. These atmospheric tests generated tremendous amounts of radioactive pollution and released this radioactive material high up into the atmosphere. Much of this radioactive pollution would proceed to precipitate down in areas hundreds to thousands of miles from the initial detonation site in the days following the test. In Nevada, just northwest of Las Vegas, the U.S. Government performed scores of atmospheric tests and exposed millions of Americans to harmful radioactive pollutants. The domestic literature studying the effects of this radioactive fallout has focused primarily upon persons living in the few counties downwind from the Nevada Test Site (NTS). These studies, however, have not quantified the full geographic or temporal extent of the harm caused by these tests. This paper combines a new dataset measuring annual county level fallout patterns for the continental U.S. with public health data to analyze the health effects of atmospheric nuclear testing at the NTS. This paper finds that fallout exposure measured at the county level resulted in a persistent and substantial increases in gross mortality. Much of this increased mortality occurred in areas more than a thousand miles from the NTS.

1 Introduction

Pollution is often the byproduct of human activity and imposes significant costs upon the public. Many times the government attempts to address the external costs associated with polluting activities, but there are cases where government policy is the direct cause of harmful pollution. During the Cold War the United States detonated over one thousand nuclear weapons in the Nevada desert at the Nevada Test Site. Prior to 1963, many of these tests were conducted above ground and releases tremendous quantities of radioactive material into the environment. When a bomb was detonated it would vaporize and irradiate thousands of tons of material. The mushroom cloud would draw much of this debris high into the atmosphere wherein high altitude winds would carry this pollution hundreds to thousands of miles from the test site. One estimate places the total atmospheric release of radioactive material from the NTS as over 12 billion Curies for the years 1951 to 1963. In comparison, Chernobyl released an estimated 81 million Curies of radioactive material LeBaron (1998).

These nuclear tests exposed millions of Americans to harmful radioactive material and many people are still living with the consequences of this pollution today. This paper measures the external health costs of domestic atmospheric nuclear testing conducted between 1951 and 1958 for the entire continental United States. Back of the envelop estimates suggest that fallout from nuclear testing contributed to 330,000 to 441,000 excess deaths from 1951 to 1973.

The current medical and scientific literature studying the health effects of nuclear testing has focused primarily upon small samples of populations who lived in the regions downwind of the Nevada Test Site.[1] These studies examine the health effects of fallout exposure in downwind populations and then extrapolate out the potential health consequences for the nation from these results. Simon and Bouville (2015) of the National Cancer Institute (NCI) note that there is great uncertainly underlying these estimates. They estimate that fallout from domestic nuclear testing caused between 11,300 and 220,000 thyroid cancer deaths. Without nuclear testing they estimated that 400,000 cases of thyroid cancer would arise naturally in the same population. One of the major drawbacks of these medical and scientific studies is that they fail to capture the temporal and geographic scope of these health effects. Another issue is that there might be unknown health effects due to radiation exposure that might only become apparent in large samples.

To address the limitations of past studies, I combine estimates of radioactive fallout exposure from (National Cancer Institute, 1997) and public health data for the U.S. to analyze the mortality effects of atmospheric nuclear testing. I use mortality data to proxy for the health effects of radiation exposure. I use within county variation in fallout deposition across years to measure the effect fallout had upon crude death rates. The panel regression framework I use allows me to identify not only where the increases in the crude death rate are occurring but also when increases in mortality appear.

This paper relates to the economics literature studying the social costs of pollution. Even though radioactive pollutants pose significant health risk and the recent Fukushima Daiichi reignited the debate about nuclear power, the societal consequences of radioactive pollution have largely been understudied. The research studying the economic effects and consequences of radioactive pollutions has focused entirely on Scandinavian and Ukrainian populations. Lehmann and Wadsworth (2011); Danzer and Danzer (2016) study the cost of the Chernobyl disaster to Ukrainian populations. Another body of research has successfully used variation in pollution as a shock to test the fetal origins hypothesis (Currie et al., 2015; Currie and Schwandt, 2015; Currie, 2013; Almond and Currie, 2011; Almond et al., 2009a; Isen et al., 2014). With respect to radioactive pollution, both Almond et al. (2009b); Black et al. (2013) use radioactive pollution to test the fetal origins hypothesis in Scandinavia. Almond et al. (2009b) uses radioactive fallout from the Chernobyl disaster and associated negative educational outcomes with in-utero exposure to fallout in Swedish cohorts. Black et al. (2013)

[1]The Downwind region consists of a few counties in AZ, NV, and UT.

use data from 14 radiation monitoring stations in Norway to study exposure in cohorts born between 1956 and 1966. They find persistent reductions in educational attainment, earnings, and IQ scores among cohorts exposed during months three and four of gestation.

Another body of research quantifies the health costs of pollution by measuring the mortality effects of pollution activities. Recent work by Hanlon (2015, 2016) has sought to measure the mortality effect of coal consumption in the UK during the 19th Century. Work by Barreca et al. (2014); Clay et al. (2016) study the long term health consequences of using coal for heating and electricity generation for the United States. Troesken (2008); Clay et al. (2014) study how municipal policies relating to the adoption of lead water pipes affected public health.

This paper contributes to the literature measuring the economic costs of pollution in multiple ways. First, this paper provides an alternative look into the costs of the Cold War and radioactive pollution upon American populations. The medical literature and work by the NCI has studied primarily downwind populations. By using U.S. public health records, I am able to test whether fallout from tests conducted in Nevada had direct effects that would appear in the U.S. Vital Statistics and death records. Second, this paper expands upon the current economics literature studying the effects of radioactive pollution and is the first paper to in the economics literature to measure the effects of nuclear testing upon American health. In this project, I create a new machine readable dataset of domestic fallout exposure. These data tracks fallout patterns from their point of origin for each nuclear test conducted from 1951 to 1958.[2] These data measure radioactive pollution at much greater levels and cover a vastly larger geographic area than the Scandinavian or Chernobyl studies. The largest deposition measure in Almond et al. (2009b) is 1,459 nCi per m^2 and in Black et al. (2013) is 883 nCi per m^2. In comparison the average county level deposition for the U.S. from the 1953 Upshot Knothole series was 578 nCi per m^2 and the largest amount for this series was 25,330 nCi per m^2. [3] Finally, this paper provides evidence to substantiate potential biological channels through which Americans were exposed to radioactive fallout.

The paper is divided into six additional sections. Section 2 provides a brief history of the NTS and atmospheric nuclear testing. Section 3 discusses the biological effects of radiation poisoning and the scientific evidence that provides the foundation for the analysis of this paper. Section 4. Section 4 presents the empirical models and describes the data that is used in the empirical analyses of this paper. These models measure the short run and long run mortality effects of radioactive fallout for American populations. Section 5 presents a discussion of the empirical results. Section 6 substantiates the empirical results by presenting a number of robustness checks. Finally, section 7 summarizes the results of this paper and the implications for the economics literature.

[2]There were three tests in 1951 Ranger Series where monitoring station data was not available. The National Cancer Institute is currently trying to create estimates for deposition using weather patterns. These tests are not included in this paper's analysis.

[3]This paper reports radiation in SI rather than metric units. Almond et al. (2009b); Black et al. (2013) use Becquerels and I use Curies. For conversion 37 bq = nCi.

2 History of the NTS and Victims of Atomic Testing

In the 1950's, millions of Americans were unknowingly exposed to radioactive fallout through both the environment and the food supply. Exposure to this radioactive matter can generally be considered as a plausibly exogenous event. Radioactive pollution is often an invisible and imperceivable threat to human health. National security concerns in the 1950's motivated atomic testing at the NTS, and while the location of the base was not random, the base was not chosen due to surrounding characteristics of the residing population.

Atmospheric atomic testing on U.S. soil was a deliberate policy decision made by domestic political leaders. In 1949, the Soviet Union detonated its first nuclear bomb Joe-1. Provoked and surprised by this sudden event, U.S. political and military leaders sought to accelerate America's own nuclear weapons program. Prior to this event, nuclear testing occurred in the Pacific.[4] The Pacific tests proved logistically costly, slow to implement, and expensive. American leaders sought a convenient testing location and settled on the Nevada Test Site due to its proximity to U.S. government labs, low levels of precipitation, and relatively secluded location (Center for Disease Control, 2006; National Cancer Institute, 1997). Nuclear testing occurred from 1951 until 1992. The period of atmospheric nuclear testing occurring between 1951 and 1963. During this period, the U.S. detonated 100 atmospheric bombs at the NTS (US Department of Energy, 2000).

During the 1950's, the public was largely unaware of the dangers that the NTS posed to public health. The Public Health Service (PHS) and Atomic Energy Commission (AEC) often sought to dismiss fears regarding the atomic testing and often misled the public. At best these organizations failed to adequately warn civilians living downwind of the test site of the health risks associated with these atomic tests. At worst they actively disseminated disinformation regarding radioactive threats (Fradkin, 2004; Ball, 1986; LeBaron, 1998). Only in 1978 did the plight of downwind populations receive national media attention. Subsequent Freedom of Information Act requests later revealed that the government knew of the dangers to public health the NTS tests posed and that the AEC had suppressed these medical reports (Fradkin, 2004). Nevertheless, persons living in the downwind region likely noticed increased incidences of leukemia and uncommon cancers. Ranchers and farmers noticed radiation burns on their animals. In 1954, following the Upshot Knothole series a group of ranchers from Iron County, UT sued the Federal Government after thousands of the sheep and lambs had died as a result of radiation poisoning. The PHS and AEC seized private veterinary records and covered up the damage. The court case was thrown out due to lack of evidence showing harm (US Government Printing Office, 1980).

However, most of the U.S. population that was exposed to radioactive fallout did not live downwind of the NTS and the exposure mechanisms for the general population was less unobservable. For victims of nuclear testing, the Federal government has provides some

[4] The three Trinity test in 1945 were conducted in New Mexico. All other tests conducted prior to the opening of the NTS occurred in the Pacific.

compensation. This compensation has focused on workers involved in the nuclear weapons program and those who lived downwind of the NTS during the 1950's. Starting in the 1990's, the U.S. Department of Justice started paying out compensation to domestic victims of the nuclear weapons program through the Radiation Exposure Compensation Act. As of 2015 the U.S. Department of Justice has paid out over $2 billion in compensation to victims (US Department of Justice, 2016).

3 The Health Consequences of Radiation Exposure

3.1 Related Scientific and Medical Literature

In this section I describe how radiation poison affects human health, explain the scientific literature relating radiation exposure with negative health outcomes in the long run, and introduce the linear no threshold model from the radiation safety literature. Understanding how radiation exposure adversely affects human health provides supports the hypothesis that radiation exposure would have an effect upon the mortality rate. Exposure to low doses of radioactive material can increase the risk of cancer. This increased risk would then suggest there is a greater risk for a person to die of cancer.

Radioactivity generally refers to dangerous particles given off by radioactive decay of matter. The weakest form of ionizing radiation are alpha particles and these particles generally cannot penetrate most thin physical barriers. Beta radiation is more dangerous and can penetrate deep into flesh causing damage. Gamma radiation is the most dangerous form of radioactivity and consists of highly energetic photons. Gamma radiation can travel easily through the body and causes immense damage to biological tissues.

With regards to nuclear testing there are three radioactive isotopes of concern to human health because of their relative radioactivity, prevalence, and how they are metabolized. These isotopes are I-131, Sr-90, and Cs-137. Other isotopes created during nuclear fission are less dangerous to human health because they do not remain in the body for extended periods. Many radioactive isotopes pass through the body and are secreted following ingestion or absorption. Iodine 131 is a potent radioactive poison. It possesses an 8 day half life, concentrates in the thyroid gland, and emits highly active forms of beta and gamma radiation as it decays (LeBaron, 1998). These traits of I-131 cause acute and rapid damage to tissue surrounding the thyroid. The primary I-131 exposure channel for humans was the dairy supply (National Cancer Institute, 1997). Strontium 90 also appeared in wheat and plant products. This isotope collects in bones and teeth. It decays over a long period and causes prolonged damage. Sr-90 possess a 25 year half life, diffuses across the body fairly uniformly and emits beta radiation (LeBaron, 1998). Cesium 137 was released in large quantities during the recent Fukushima Daiichi disaster. This isotope collects in fleshy tissue and does not concentrate into any particular organ. It has a half life of 33 years and emits both alpha and beta radiation(LeBaron, 1998).

The medical and scientific knowledge regarding the effects of human exposure to ionizing radiation comes many sources. Studies of Japanese atomic bomb survivors and persons living downwind of nuclear test sites provide much of this knowledge. In human population studies of radiation exposure, researchers have measured a variety of negative health and developmental consequences from exposure to ionizing radiation. Studies of atomic bomb survivors and persons exposed during pregnancy identify increased cancer risks, negative developmental and cognitive effects due to radiation exposure (Otake et al., 1993; Otake, 1996; Schull, 1997; Lee, 1999). Researchers studying Chernobyl have found greater incidences of thyroid cancers and lesions indicative of I-131 poisoning in exposed population (Shibata et al., 2001; Williams, 2002). Researchers studying downwind American populations have also found evidence of increased thyroid cancer and leukemia risks in domestic downwind cohorts (Stevens et al., 1990; Kerber et al., 1993; Gilbert et al., 2010). Scientific animal studies have revealed that detrimental health effects of I-131 poisoning appear at ingestion rates as low as 50 nCi a day (Bustad et al., 1957).

The medical and scientific literature suggest that exposure to ionizing radiation increases risks of various types of cancer and can have detrimental effects upon human growth and development. Within the radiation exposure and protection literature there is a model called the Linear No-Threshold Model of radiation exposure. This model formalizes the cancer risks associated with exposure to radiation. This conservative model assumes that there is no inherently safe dose of ionizing radiation and that the risk associated with exposure are linear. Figure 2 is a simple diagram explaining this model's relationship with regards to cancer risks and alternative hypotheses (National Research Council, 2006). If the linear no threshold model is an accurate measure of risk, then it is quite plausible that low level radiation could have a substantial effect upon overall mortality rates for the United States.

3.2 Exposure Mechanisms

Exposure to harmful radioactive fallout can occur either through direct channels or indirect channels. Radioactive material can enter the body if it lands on the skin with radioactive dust. Many people and animals living in the downwind counties surrounding the NTS were exposed to harmful fallout in this matter. People can inhale radioactive material when it is suspended in the air. Inhalation of radioactive dust would be the most likely in the downwind region. Research by the National Cancer Institute (1997); Center for Disease Control (2006) establish that the food supply served as the main indirect vector of exposure for most Americans during the atomic testing period. Scientific research during the 1950's and 1960's shows that harmful radioactive matter appeared in the food supply and thyroids during the testing period (Beierwaltes et al., 1960; Garner, 1963; Kulp et al., 1958; Olson, 1962; Van Middlesworth, 1956). The PHS also released research corroborating this evidence but denied the health risks associated with the radiation levels reported (Wolff, 1957, 1959; Flemming, 1959, 1960).

In particular, the NCI establishes the dairy channel as a primary vector through which Americans were exposed to significant quantities of radioactive material. Most Americans would not be exposed to radioactive dust carried by low altitude winds. Instead, high altitude winds would carry the material far from the test site and the material would only deposit on the ground if it happened to be precipitating while the radiation cloud was overhead. This fallout would occur in the few days following the nuclear test. This radioactive material would deposit on crops and pasture. Some radioactive material would enter wheat and other plant products, but consumption of these products would not necessarily be in the same region they were produced. Dairy, however, during the 1950's and 1960's was generally produced and consumed locally (National Cancer Institute, 1997). This channel is unique in that cows would consume large quantities of irradiated pasture and concentrate radioactive material, in particular I-131, in milk. People living in the region where deposition occurred would then be more likely to consume this irradiated food product containing a potent radioactive poison in the days following the atomic test. In the empirical section I provide evidence to substantiate dairy as the main exposure mechanism driving increases in mortality.

4 Empirical Strategy

Suppose there is a homogeneous population consisting of persons exposed and not exposed to ionizing radiation. Let $X_i \epsilon \{0,1\}$ denote the outcome of developing cancer and $\kappa_i \epsilon \{0,1\}$ denote exposure. The probability of any individual developing cancer is increasing in this exposure variable $P(X_i = 1|\kappa_i = 1) > P(X_i = 1|\kappa_i = 0)$. Let $P(Death_i|X_t)$ denote the probability of an individual dying. Conditional on developing cancer and the individual's probability of dying increases. This is one channel through which fallout from nuclear tests would affect county level aggregate mortality.

I-131 collects in the thyroid gland and this organ is responsible for growth and development. Children are disproportionately affected by the milk exposure channel and thus I-131. It is plausible that long term health effects other than cancer might arise in these populations and that these effects might increase mortality risk. In the empirical section I first test the hypothesis that radiation exposure in counties led to increases in the crude death rate. Then I test whether the milk exposure channel was the primary driver of increased mortality. The dairy exposure channel also would increase the likelihood of developing specific cancers, namely thyroid cancers and leukemia. The dairy exposure channel also suggests that radiation exposure would increase the mortality risk of younger persons more than that of older individuals. Finally, it is plausible that areas that had greater access to locally produced dairy would have increased exposure risk. This risk would mean that radiation exposure would have a greater mortality effect in these dairy producing counties.[5]

[5]Only the crude death regressions are sufficiently complete to report in this draft of the paper.

4.1 Empirical Model:Measuring Short Term and Long Term Mortality Effects

Equation 1 provides a model to test whether or not radioactive fallout in locally produced milk or in the environment had a statistically significant relationship with mortality in the years directly following the test.

$$ln(y_{it}) = \Sigma_{k=0}^{5}\beta_k * Exposue.I131_{it-k} + \sigma * ln(pop10k)_{it} + \alpha_i + \gamma_t + Trend_{st} + \epsilon_{it} \quad (1)$$

The outcome denoted by y_{it} measures the number of total deaths in a given county i and year t. $Exposue.I131_{it-k}$ denotes the variable used to proxy for fallout exposure. $Milk.I131_{it}$ is a measure of radioactive iodine in locally produced milk in a given county year in thousands of nCi per day/Liter. $Dep.I131_{it}$ is a cumulative measure of total radioactive iodine deposited per square meter in a given county year and is used as an alternative measure of population exposure. $ln(pop10k_{it})$ is a measure of 10,000 divided by total county population.[6] County population is interpolated between Censuses. The NCI created daily integrated estimates of secreted iodine per liter of milk for each nuclear test. They then summed up these secretions over the entire test series. If a cow in a county produced one liter of milk each day, this would measure the amount of radioactive iodine secreted in all those liters of milk in a given year. This measure should make a fairly useful proxy of the amount of radioactive iodine in human food supply in a given county year.

Furthermore, the milk variable accounts for grazing practices across regions. Cows in upstate NY would not have been exposed to much radiation from February tests as they would have been inside barns consuming fodder while cows in NV or AZ would have been exposed. In some specifications I replace the Milk.I131 variable with Dep.131 which is a measure of I-131 deposited per $meter^2$ in a given county year. α_i and γ_t denote county and year fixed effects. These variables control for time invariant county characteristics and national yearly shocks. In my preferred specifications $Trend_{st}$ denotes state specific time trends and controls for any spurious underlying motions in the data that might be correlated with the exogenous variable of interest. The variable ϵ_{it} denotes the heteroskedastic error term and is clustered at the county level.

$$ln(y_{it}) = \Sigma_{j=0}^{5}\theta_j * \Sigma_{k=1}^{5}\frac{Exposure.I131_{it-k-5*j}}{5} + \sigma * ln(pop10k)_{it} + \alpha_i + \gamma_t + Trend_{it} + \epsilon_{it} \quad (2)$$

Equation 2 uses a similar framework to that of Equation 1, but the exposure of interest consists of lagged five year averages of the I-131 exposure measures. This distributed lag structure measures the dynamic mortality response to county level radiation exposure over a long time horizon.

[6] This control is econometrically identical to regressing on total deaths per 10,000 individuals and controlling for population.

4.2 Data

4.2.1 Public Health Data

This project uses three sources of mortality data. The first is a county level annual panel constructed by Bailey et al. (2016) of crude deaths, infant deaths and crude births from 1915 to 2007. This panel is used to measure the geographic and temporal extent to which radioactive pollution from the NTS harmed human health. The second and third sources of data provide detailed measures on cause of death.[7] From 1946 onwards, U.S. Vital Statistics provide cause of death tables for certain diseases, including cancer, at the county year level. Individual level death records from the Compressed Mortality File (CMF) provide very detailed information on cause of death and are available for the years between 1959 and 1988. The CMF panel is aggregated for a county level analysis and is combined with the county level panel constructed from U.S. Vital Statistics to explore the relationship between fallout exposure and cause of death. From 1959 to 1988, I can identify specific cancers that would be associated with I-131 poisoning. These include thyroid cancer, lymphoma, and leukemia.

4.2.2 Fallout Deposition Data

In 1983, Congress authorized the Secretary of Health and Human Services to investigate and measure thyroid doses from I-131 poising in American citizens. The NCI undertook the task of gathering radiation monitoring station data from historical records. With these records and weather station data the NCI was able to track the position of the radiation cloud, determine how much radiation would deposit with precipitation, and employ kriging to estimate fallout deposition in counties without monitoring stations. Much of the raw data came from national monitoring stations whose number varied across time, but never exceeded 100 stations. The military also engaged in air monitoring and used city-county stations around the NTS to track the radiation cloud (NCI 1997). These are the most complete and comprehensive measures for fallout deposition from nuclear tests for the United States. The data employed in this paper is derived from the NCI estimates. The NCI provides estimate for I-131 deposition for each nuclear test conducted from 1951 to 1958, with the exception of three tests in the Ranger 1951 series. The depositions are measured as nanoCuries (nCi) per meter squared and are reported for each day following a nuclear test until the next subsequent test in the series. Figures 3 to 4 provide maps of my deposition data for the Upshot Knothole and Plumbbob test series.

The NCI also provides daily integrated estimates for I-131 secreted in locally produced milk. These measures are a function of how cows metabolize and secrete iodine at different levels of exposure, grazing practices during the testing window, and the levels of radiation deposition estimated in the kriging model. This methodology can cause substantial differences between radiation presence in milk estimated at the county level and deposition. During the 1950's, many households consumed locally produced dairy, and I-131's short eight day half-

[7] The current iteration of this paper has not yet employed this data.

life means that persons would consume it before the radioactive I-131 would decay. Children would be especially vulnerable to this radiation exposure channel because they tended to drink more milk than adults, had smaller thyroids, and were still growing during this period (NCI 1997). Since a child's thyroid is smaller than an adult's, the same quantity of I-131 would cause greater damage because it would be concentrated into a smaller area. Furthermore, the thyroid regulates growth and development and harm to this organ might lead to unanticipated long term health problems. Figures 5 to 6 provide maps of my milk exposure data for the Upshot Knothole and Plumbbob test series.[8]

4.3 Identification and Sample

Summary statistics for the sample used in the empirical regressions are provided in Table 1.[9] To test this mortality effect of fallout I conducted a set of fixed effects regressions using an annual county panel of gross mortality per 10,000 individuals for years between 1930 and 1990. I assumed that radiation deposition in years without testing was zero. While global fallout might have deposited in counties during the 1950's, it would have been relatively negligible and more diffuse relative to NTS fallout. The focus of this paper is on the effect of exogenous fallout resulting directly from the NTS.

I leverage two different sources of variation in radiation exposure to measure the impact fallout from nuclear tests had upon the health of American populations. The first source is a measure of fallout deposition which resulted from radiation precipitating down from the atmosphere. The second source of variation comes from NCI estimates of radioactive I-131 present in locally produced milk. The medical and scientific literature suggests that the food supply served as the primary exposure vector. While persons living in the downwind region of the NTS were exposed to radioactive fallout through inhalation and environmental exposure, most of the American population would not have been exposed to large amounts of fallout through direct environmental exposure. The fact that most of the United States experienced fallout exposure as a result of precipitation coupled with the fact that radiological threats are necessarily perceptible, it is reasonable to assume that the radiation exposure measures are exogenous. Persons living downwind of the NTS may have had better knowledge regarding fallout and sought to mitigate some of the health risks. Government disinformation in the 1950's and the imprecise public knowledge regarding the effects of fallout exposure prior to 1978 suggests such behavior would be unlikely. Furthermore, such avoidance behavior would bias against finding a mortality effect.

Through these fallout patterns I identify the causal effect of radioactive pollution upon county level mortality in the short and long term. My main identifying assumption is that most people who were exposed to radioactive fallout eventually die in the county they were

[8]Many downwind counties in the milk measures need to be incorporated into the model. The estimates for these counties are at a finer level of aggregation and need addition work to be incorporated into the sample. Some counties and Virginia Independent Cities are omitted from the sample due to data limitations.

[9]The number of counties year observations change between Milk and Dep measures because I have yet to code up some downwind counties in AZ, CA, NV, and UT. There are also a few counties that are not reported in the milk data but are reported in the deposition measures.

exposed. Out migration of exposed individuals into regions with lower levels in which exposure would bias against finding an effect. I attempt to control for population changes using Census population measures which are linearly interpolated for the years between Censuses. If there was no outmigration from irradiated counties, these regressions would account for the total mortality effect of the Nevada nuclear tests. Migration would bias the results if somehow healthier individuals migrated systematically to less exposed areas or unhealthy individuals systematically migrated to more exposed regions. There is no reason to believe a priori that such systemic migration occurred. The more plausible scenario is that migration over time introduces measurement error and causes the estimated effect of mortality to attenuate over time.

5 Empirical Results

5.1 Panel Regression Results

The results on short term mortality effects of radiation exposure appear in Table 2 and are plotted in Figures 7 and 8. Specification 1 uses only year and county fixed effects. Specification 3 uses state by year fixed effects and is highly restrictive. It is included as a robustness check for this would control for possible spurious relationships between unobserved variables. State by year fixed effects will absorb much of the radiation exposure effect in many states and the identifying variation is the difference in radiation exposure from the state year average. Specification 3 includes state specific time trends and is the preferred specification. These state specific time trends control for long run regional trends that might be spuriously correlated with radiation exposure. Specification 2 in Table 2 suggests a 1,000 nCi increase in I-131 deposition resulted in a 0.71% increase in mortality two years following deposition. Four and five years following deposition, the model suggests a 0.92% and 0.75% increase in the crude death rate. These effects are all statistically significant at the 1% level. The inclusion of state by year fixed effects removes any statistical significance from these coefficients. The measures using the milk records are much more consistent and larger in magnitude. The mortality effects appear earlier, and in specification 2 all of the six coefficients of interest are statistically significant at the 1% level. One thousand nCi of I-131 in the local dairy supply this year, last year, two years ago, three years ago, four years ago, and five years ago increased the crude death rate by 3.23%, 3.52%, 3.65%, 2.46%, 3.82%, and 1.80% respectively. This pattern of mortality remains robust with the inclusion of state by year fixed effects.

The long term mortality effects of this radiation exposure appears to persist for many years following the initial radiation exposure event. Table 3 reports the results for these regressions and Figures 9 and 10 plot the coefficients for specification 2. I find that radiation deposition significantly increased mortality up to fifteen years after the initial exposure event. An average of 1,000 nCi deposited between 1 and 5 years ago, 6 and 10 years ago, and 11 and 15 years ago increased the crude death rate in a given county year by 3.22%, 3.23%, and 2.53% respectively. The regressions using the milk measures find a similar pattern.

The magnitudes of these estimates are larger in part due to the fact that average level of I-131 in milk is substantial smaller than the deposition estimates. An average of 1,000 nCi in locally produced milk between 1 and 5 years ago, 6 and 10 years ago, and 11 and 15 years ago increased the crude death rate in a given county year by 11.30%, 7.93%, and 5.13% respectively. In the next section I quantify these estimated coefficients into numbers of deaths. The negative coefficients in specification 1 and 3 on the later lags suggest that persons died at younger ages in irradiated counties. Attenuation bias from migration and culling could bias the mortality effect downwards. Future research will study the age distribution of the mortality effect.

5.2 Quantifying the Magnitude of the Effects

The effects upon crude mortality are large relative to Simon and Bouville (2015) estimates and the deaths from the atomic bombings of Hiroshima and Nagasaki. I perform a series of back of the envelope calculations to quantify the total mortality effect. Assume that fallout did not substantial increase the national average for deaths per 10,000 individuals for the years between 1930 to 1990. Equation 3 adds up all of the implied deaths using coefficients from the reduced form analysis. These estimates rely on this assumption to estimate the total number of excess deaths caused by atomic testing in Nevada. Under the more restrictive model, I use only the first three lags from specification 2 of Equation 2. The regression estimates for Equation 2 only identified a statistically significant effect upon the crude death rate up to fifteen years after the exposure event. Estimates of the effects of fallout upon crude deaths are available in Table 4.

$$TotalMortalityEffect = \Sigma_{t=1951}^{1990} \Sigma_{i=0}^{N} \overline{DPC}_t * pop10k_{it} * (\theta_1 * \Sigma_{k=1}^{5} \frac{Milk.I131_{it-k}}{5} + \theta_2 * \Sigma_{k=6}^{10} \frac{Milk.I131_{it-k}}{5} + \theta_3 * \Sigma_{k=11}^{15} \frac{Milk.I131_{it-k}}{5}) \quad (3)$$

$Milk.I313_{it}$ represents the measures of I-131 in milk in a given county year. The corresponding θ_j measures the average effect that 1000 nCi had upon crude deaths per 10,000 residents. $\overline{DPC_t}$ represents the national average for crude deaths per 10,000 for the United States in a given year. $pop10k_{it}$ represents the population in 10,000's of people in a given county year. From this information I estimate the effect of atmospheric nuclear testing upon crude deaths from 1930-1990. The average increase in mortality from I-131 in milk for a given county year between 1951 and 1990 observation 0.77 deaths per 10,000 with a standard deviation of 1.26. The largest increase in county year mortality was 21.06 deaths per 10,000. The average increase in mortality from radiation ground deposition for a given county year between 1951 and 1990 observation 0.46 deaths per 10,000 with a standard deviation of 0.79. The largest county year increase in mortality was 20.81 deaths per 10,000.

Equations 3 suggests that I-131 in milk contributed to approximately 440,000 excess deaths. These estimates do not yet include most of the downwind region surrounding the NTS. I find comparable but smaller results, when I use $Dep.I131_{it}$ as an alternative exposure measure. The deposition measures are more complete than the milk measures and include all regions downwind from the test site. The same method using fallout deposition suggests I-131 deposition contributed approximately to 310,000 excess deaths. Many of these estimated deaths occurred in regions far the NTS. Figures 11 and 12 report the average state effects of radiation exposure on mortality for years 1951 to 1973 (The last test series was in 1958 and the mortality effect lasted 15 years.) It's clear that the model suggests much of the overall mortality death appears in the Midwest and Eastern U.S. where larger populations would have been exposed. The per capita mortality effects tend to be greatest out west in the Plains and states north and east of the NTS.

Policy makers often assign accounting values to human lives when evaluating policy decisions. Viscusi (1993); Viscusi and Aldy (2003) survey these valuations placed on human life. For the years 1988 to 2000, valuations of human life by U.S. Federal Government agencies ranged between $1.4 million and $8.8 million in 2016$. Theses values and my estimates place the value of lost life between $434 billion and $3,881 billion in 2016$. Costa and Kahn (2004) use a hedonic wage regressions on industrial sector mortality risks to back out plausible market values for human life for each decade from 1940 to 1980. Using their values, I estimate the value of lost life from ground deposition as $1.12 trillion in 2016$. The estimates from milk exposure places the value of lost life at $1.6 trillion.

6 Robustness Checks

I perform a falsification test to test whether or not unobserved underlying factors were driving the crude death results. I select a sample of counties from 1911 to 1950 and shifted the radiation exposure measures back 20 years. I then ran Equation 1 and the results are available in Table 5. I find no evidence that either fallout deposition or fallout in milk had a systemic effect on the log crude death rate between 1911 and 1950.

7 Conclusion

This paper explores the temporal and geographic extent of damage caused by atmospheric atomic tests conducted in Nevada between 1951 and 1958. Using a new national dataset of radiation deposition and quantities of I-131 in the dairy supply, this paper finds that radiation exposure increased crude deaths in areas hundreds to thousands of miles from the test site. These results are consistent with a linear no threshold model of radiation exposure. The future work of this paper will focus on better identifying the exposure channels, determining which demographic groups were most effected, and measuring the effects on cancer rates.

Table 1: Summary Statistics

	Obs	Mean	Std. Dev.	Min	Max
I-131 Ground Deposition, 1,000's nCi per m^2	125649	0.05	0.24	0.00	13.74
I-131 Milk Exposure, 1,000's nCi	125677	0.03	0.14	0.00	4.60
I-131 Average 5 year Deposition	125537	0.05	0.15	0.00	6.61
I-131 Average 5 year Milk Exposure	125677	0.03	0.08	0.00	1.86
Population 10,000's	125677	6.49	22.84	0.01	887.82
Deaths Per 10,000	125677	10.08	2.51	0.86	56.49
Ranger 1951 I-131 Ground Deposition, nCi per m^2	125677	3.10	18.11	0.00	960.00
-I-131 Milk Exposure, nCi	125677	0.11	0.70	0.00	40.00
Buster Jangle 1951 I-131 Ground Deposition, nCi per m^2	125677	78.82	137.37	0.00	1222.00
-I-131 Milk Exposure, nCi	125677	14.47	25.89	0.00	210.00
Tumbler Snapper 1952 I-131 Ground Deposition, nCi per m^2	124989	467.20	528.61	0.00	4982.00
-I-131 Milk Exposure, nCi	125677	302.25	305.55	4.70	4600.00
Upshot Knothole 1953 I-131 Ground Deposition, nCi per m^2	125677	574.39	866.14	0.00	25330.00
-I-131 Milk Exposure, nCi	125677	271.37	194.09	0.26	2800.00
Teapot 1955 I-131 Ground Deposition, nCi per m^2	125677	389.01	517.91	0.00	6195.00
-I-131 Milk Exposure, nCi	125677	135.33	124.42	1.60	1200.00
Plumbbob 1957 I-131 Ground Deposition, nCi per m^2	125677	726.50	552.63	0.00	13736.00
-I-131 Milk Exposure, nCi	125677	479.31	350.40	2.00	2500.00
Hardtack 1958 I-131 Ground Deposition, nCi per m^2	124989	1.37	10.44	0.00	145.00
-I-131 Milk Exposure, nCi	125677	0.00	0.00	0.00	0.00

Table 2: Short Run: Log Number of Crude Deaths, Mortality Panel 1930-1990

	(1) lnD	(2) lnD	(3) lnD	(4) lnD	(5) lnD	(6) lnD
I-131 Milk, t	0.0470***	0.0323***	0.0228**			
	(0.00579)	(0.00538)	(0.00904)			
I-131 Milk, t-1	0.0492***	0.0352***	0.0320***			
	(0.00508)	(0.00488)	(0.00754)			
I-131 Milk, t-2	0.0474***	0.0365***	0.0273***			
	(0.00489)	(0.00471)	(0.00804)			
I-131 Milk, t-3	0.0312***	0.0246***	0.0177**			
	(0.00495)	(0.00483)	(0.00842)			
I-131 Milk, t-4	0.0435***	0.0382***	0.0178**			
	(0.00511)	(0.00511)	(0.00789)			
I-131 Milk, t-5	0.0213***	0.0180***	0.00700			
	(0.00471)	(0.00468)	(0.00719)			
I-131 Dep, t				0.00692***	0.000356	-0.000527
				(0.00239)	(0.00230)	(0.00318)
I-131 Dep, t-1				0.00895***	0.00332	0.00343
				(0.00295)	(0.00255)	(0.00412)
I-131 Dep, t-2				0.0111***	0.00708***	0.00474
				(0.00283)	(0.00256)	(0.00339)
I-131 Dep, t-3				0.00834***	0.00480	0.00486
				(0.00320)	(0.00295)	(0.00479)
I-131 Dep, t-4				0.0125***	0.00915***	0.00127
				(0.00314)	(0.00291)	(0.00332)
I-131 Dep, t-5				0.0102***	0.00748***	0.00217
				(0.00239)	(0.00224)	(0.00273)
Log Population 10k	-0.603***	-0.595***	-0.600***	-0.604***	-0.595***	-0.599***
	(0.0132)	(0.0108)	(0.0112)	(0.0132)	(0.0108)	(0.0112)
Year_FE	Yes	Yes	No	Yes	Yes	No
County_FE	Yes	Yes	Yes	Yes	Yes	Yes
State_Time_Trends	No	Yes	No	No	Yes	No
State_Year_FE	No	No	Yes	No	No	Yes
N	157557	157557	157543	157389	157389	157375
Adjusted r^2	0.983	0.985	0.985	0.983	0.985	0.985

All Standard Errors are Clustered by County

All regressions control for log total population. All coefficients are in 1000's nCi.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 3: Long Run: Log Number of Crude Deaths, Mortality Panel 1930-1990

	(1)	(2)	(3)	(4)	(5)	(6)
	lnD	lnD	lnD	lnD	lnD	lnD
Avg. Milk Exposure 1-5 yrs ago	0.245***	0.113***	0.122***			
	(0.0168)	(0.0138)	(0.0260)			
Avg. Milk Exposure 6-10 yrs ago	0.150***	0.0793***	0.0490*			
	(0.0136)	(0.0126)	(0.0260)			
Avg. Milk Exposure 11-15 yrs ago	0.0997***	0.0513***	0.00115			
	(0.0139)	(0.0133)	(0.0259)			
Avg. Milk Exposure 16-20 yrs ago	0.0353**	0.0149	-0.0842***			
	(0.0145)	(0.0142)	(0.0297)			
Avg. Milk Exposure 21-25 yrs ago	-0.0338**	-0.0104	-0.0638***			
	(0.0146)	(0.0126)	(0.0236)			
Avg. Exposure 1-5 yrs ago				0.0848***	0.0322***	0.0230
				(0.0154)	(0.0103)	(0.0199)
Avg. Exposure 6-10 yrs ago				0.0681***	0.0323***	0.0116
				(0.0127)	(0.00965)	(0.0161)
Avg. Exposure 11-15 yrs ago				0.0500***	0.0253***	0.0174
				(0.0106)	(0.00866)	(0.0142)
Avg. Exposure 16-20 yrs ago				0.0226**	0.00943	-0.0234**
				(0.00934)	(0.00858)	(0.0118)
Avg. Exposure 21-25 yrs ago				0.00578	0.00679	-0.00533
				(0.00642)	(0.00577)	(0.00998)
Log Population 10k	-0.629***	-0.604***	-0.613***	-0.636***	-0.604***	-0.613***
	(0.0135)	(0.0111)	(0.0114)	(0.0134)	(0.0111)	(0.0114)
Year_FE	Yes	Yes	No	Yes	Yes	No
County_FE	Yes	Yes	Yes	Yes	Yes	Yes
State_Time_Trends	No	Yes	No	No	Yes	No
State_Year_FE	No	No	Yes	No	No	Yes
N	125677	125677	125663	125243	125243	125229
Adjusted r^2	0.988	0.989	0.989	0.988	0.989	0.989

All Standard Errors are Clustered by County

All regressions control for log total population. All coefficients are in 1000's nCi.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table 4: Cumulative Crude Death Effects for County Years 1930-1990

MILK MEASURES				
Deaths Per 10,000				
Observations	Mean	Std. Dev.	Min	Max
125,677	.77	1.26	0	21.06
Cumulative National Total Deaths Effect from 1930-1990				
441,566				

DEPOSITION MEASURES				
Deaths Per 10,000				
Observations	Mean	Std. Dev.	Min	Max
125,243	0.46	0.79	0	20.81
Cumulative Deaths Effect from 1930-1990				
310,069.4				

Table 5: Short Run Placebo. Log Number of Crude Deaths, Mortality Panel 1911-1950

	(1) lnD	(2) lnD	(3) lnD	(4) lnD	(5) lnD	(6) lnD
I-131 Milk, t	-0.0209**	0.0122	0.00431			
	(0.00816)	(0.00974)	(0.0188)			
I-131 Milk, t-1	-0.0445***	-0.0148	0.0174			
	(0.00885)	(0.00994)	(0.0181)			
I-131 Milk, t-2	-0.0217***	-0.000838	0.00153			
	(0.00702)	(0.00783)	(0.0127)			
I-131 Milk, t-3	-0.0388***	-0.0156	0.00322			
	(0.00894)	(0.00998)	(0.0168)			
I-131 Milk, t-4	-0.0142**	0.00783	0.0111			
	(0.00650)	(0.00686)	(0.0105)			
I-131 Milk, t-5	-0.0167***	-0.00257	-0.0000175			
	(0.00610)	(0.00586)	(0.00950)			
I-131 Dep, t				0.00773	0.0152***	0.00955
				(0.00507)	(0.00537)	(0.00717)
I-131 Dep, t-1				0.00000270	0.00596	0.00629
				(0.00628)	(0.00615)	(0.00724)
I-131 Dep, t-2				0.00742*	0.00712	0.00231
				(0.00447)	(0.00492)	(0.00563)
I-131 Dep, t-3				-0.00622	-0.00748	-0.0180**
				(0.00481)	(0.00503)	(0.00763)
I-131 Dep, t-4				0.00109	-0.000518	-0.00533
				(0.00344)	(0.00336)	(0.00352)
I-131 Dep, t-5				-0.000991	-0.000235	0.000102
				(0.00285)	(0.00291)	(0.00358)
Log Population 10k	-0.577***	-0.566***	-0.569***	-0.554***	-0.567***	-0.569***
	(0.0182)	(0.0201)	(0.0203)	(0.0178)	(0.0201)	(0.0202)
Year_FE	Yes	Yes	Yes	Yes	Yes	Yes
County_FE	Yes	Yes	Yes	Yes	Yes	Yes
State_Time_Trends	No	Yes	No	No	Yes	No
State_Year_FE	No	No	Yes	No	No	Yes
N	42085	42085	42085	41987	41987	41987
r2_a	0.985	0.986	0.986	0.985	0.986	0.986

All Standard Errors are Clustered by County

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

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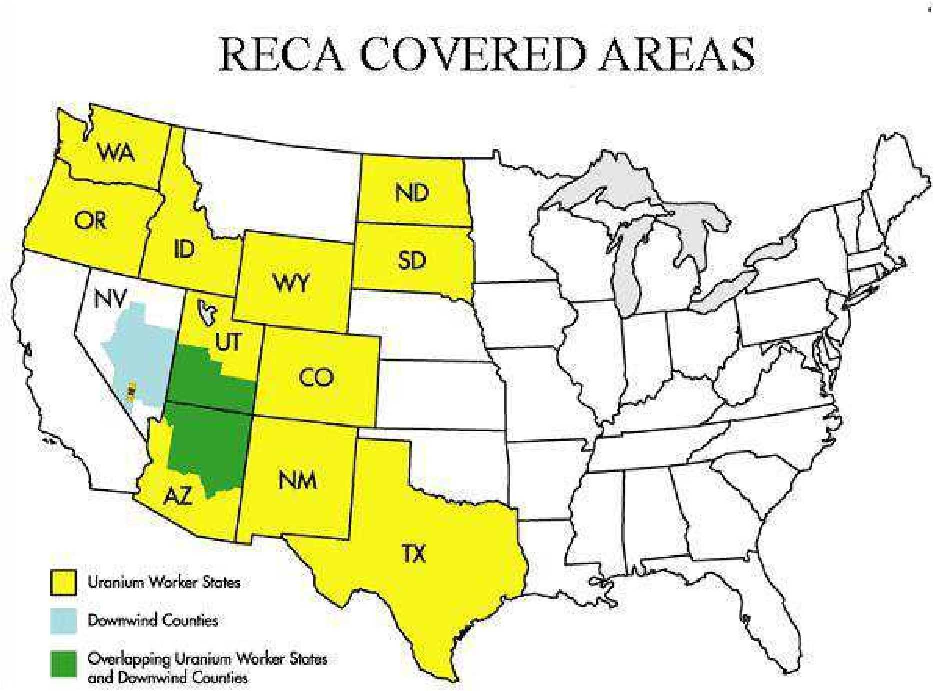

Figure 1

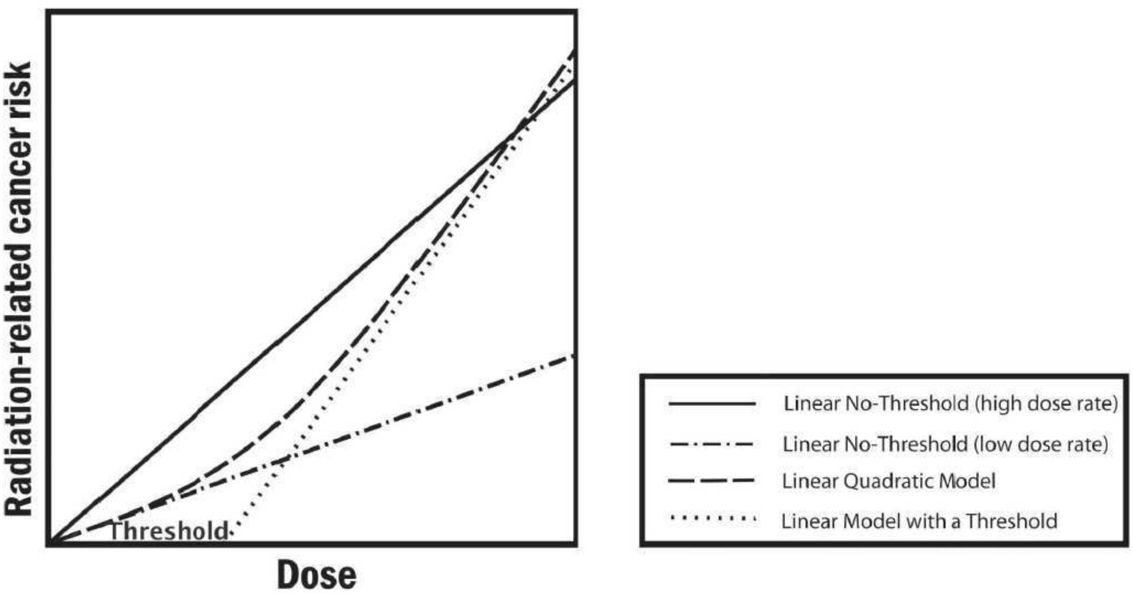

Figure 2: Source: National Research Council (2006)

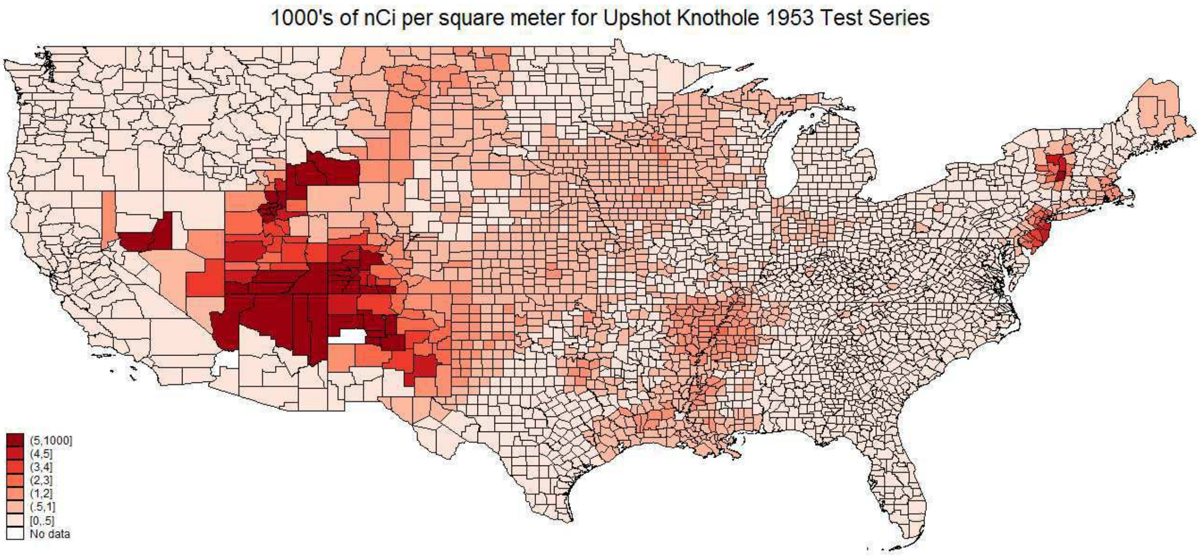

Figure 3: Cumulative I-131 Deposition from Upshot Knothole Series

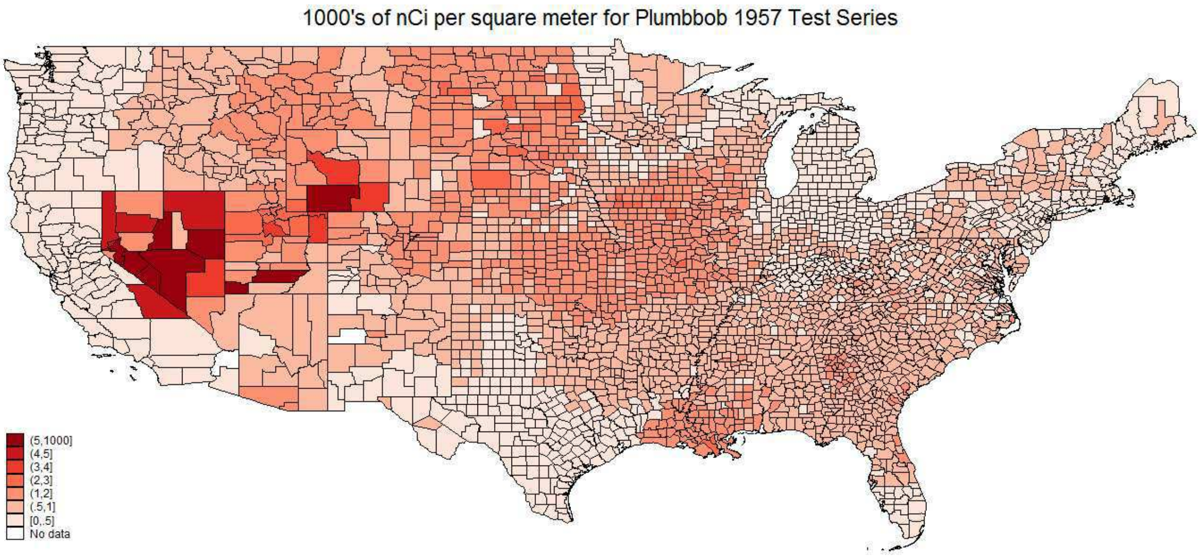

Figure 4: Cumulative I-131 Deposition from Plumbbob Series

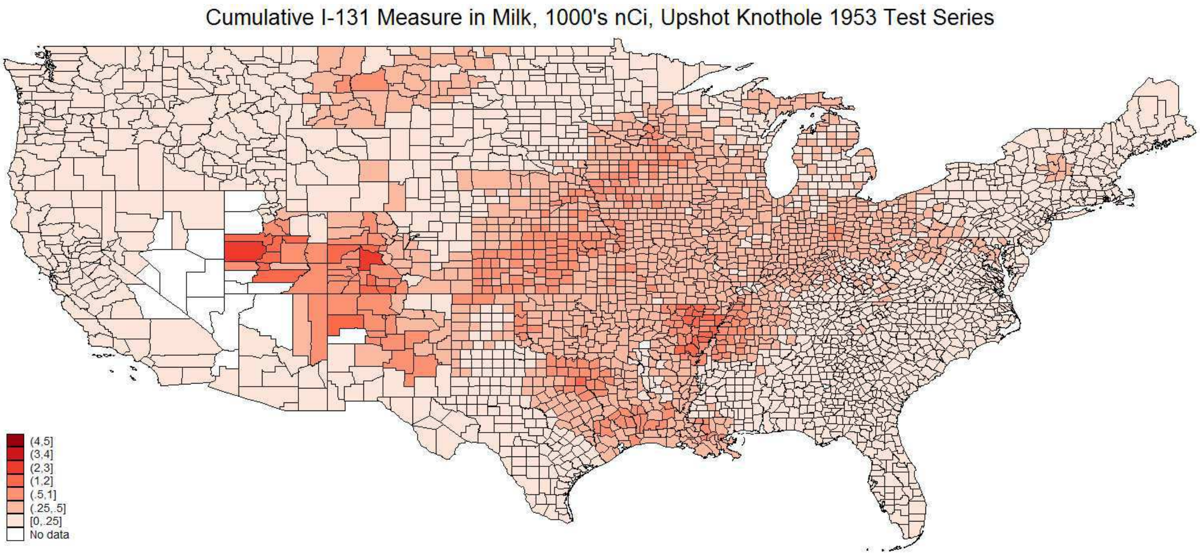

Figure 5: Cumulative I-131 Milk Measures from Upshot Knothole Series

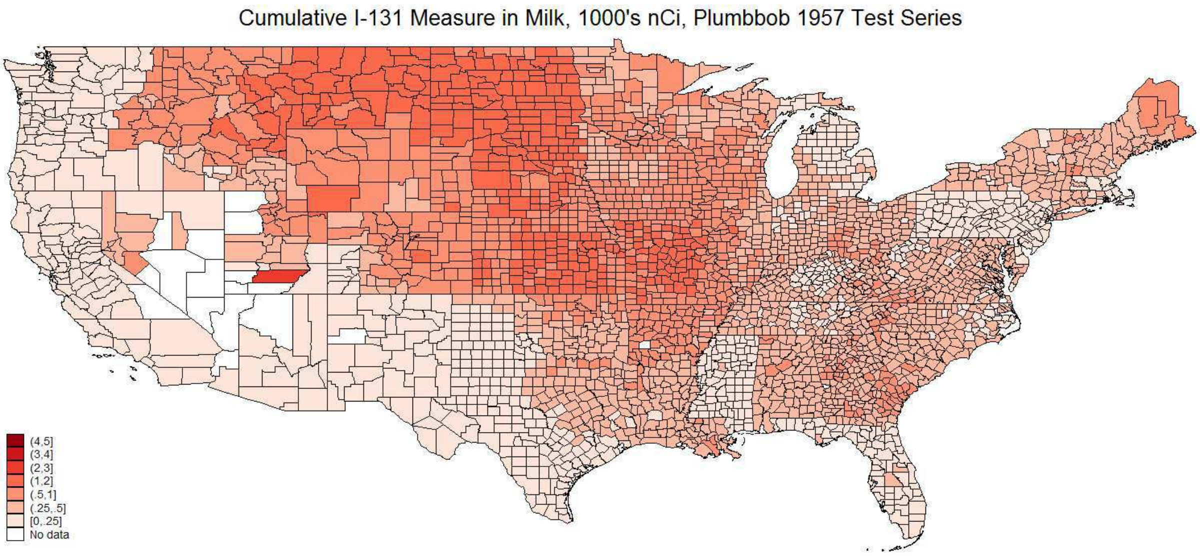

Figure 6: Cumulative I-131 Milk Measures from Plumbbob Series

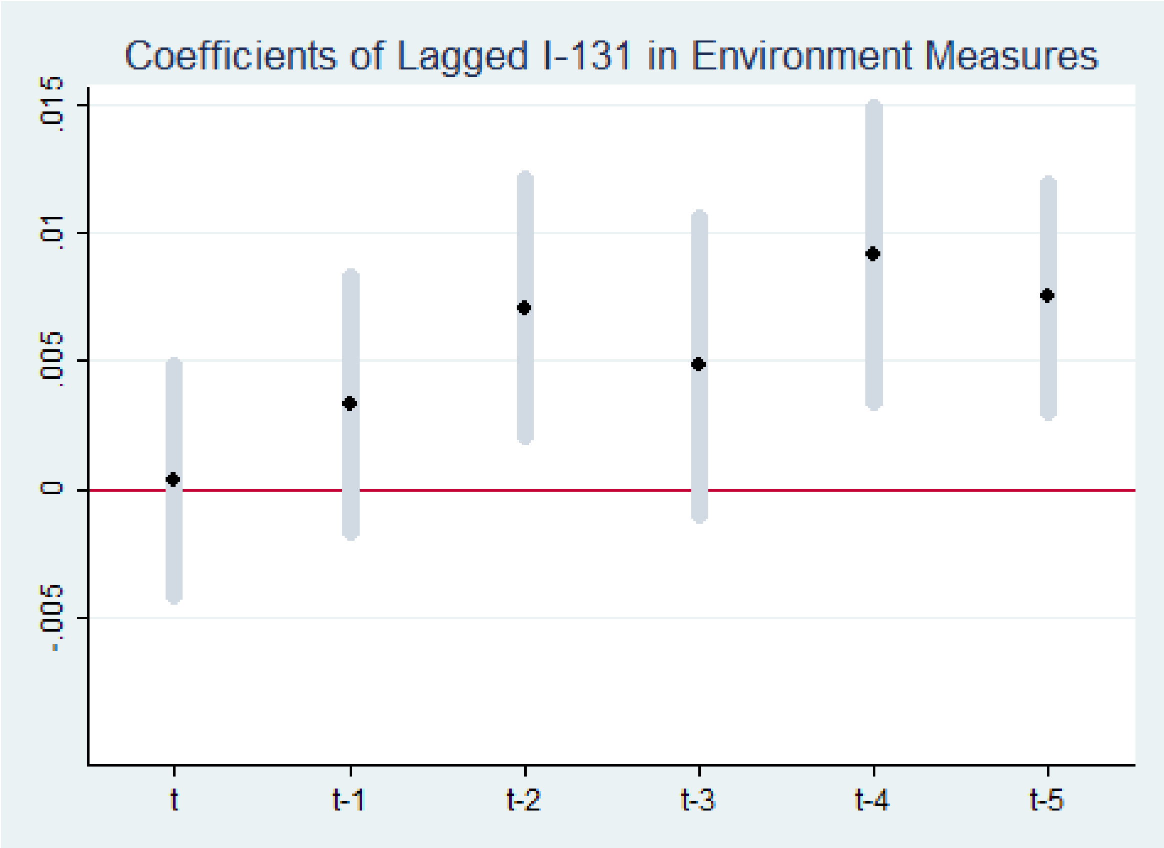

Figure 7: Coefficients on Lagged Deposition Measures on Crude Deaths, 95% CI

Figure 8: Coefficients on Lagged Milk Measures on Crude Deaths, 95% CI

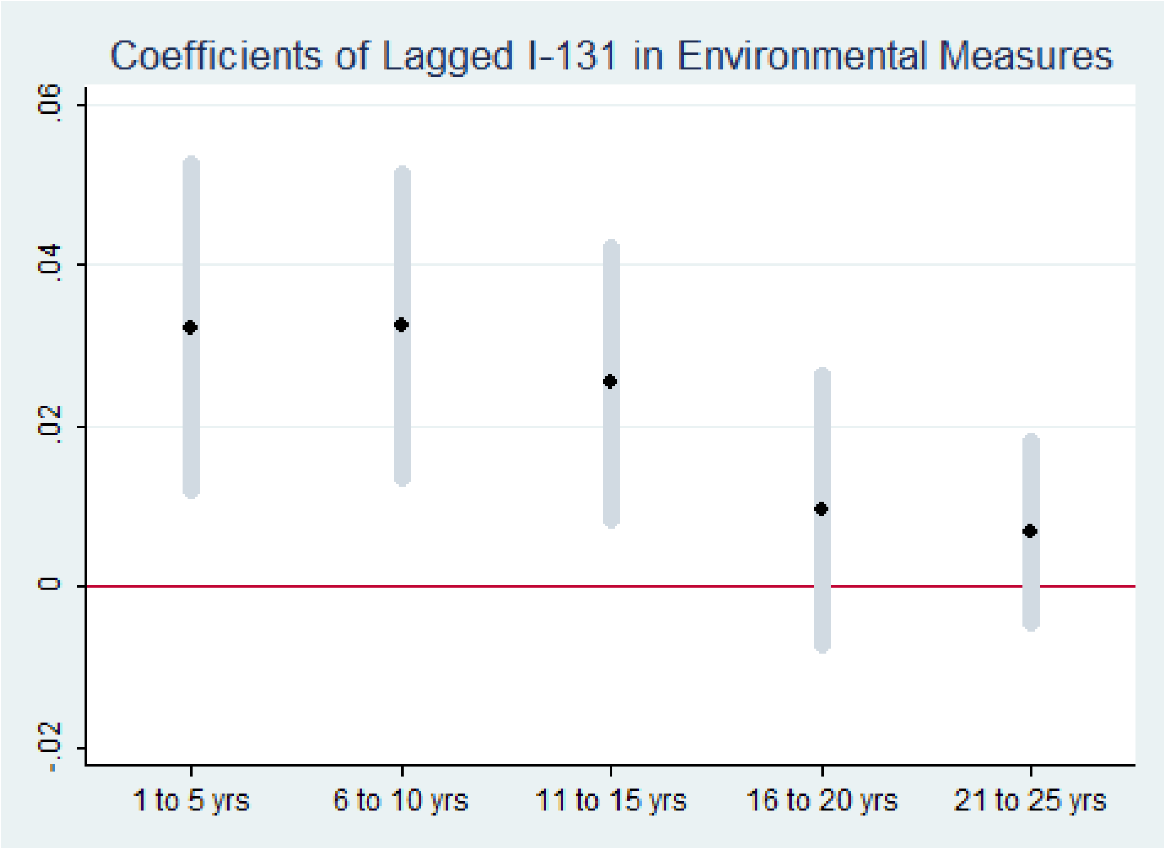

Figure 9: Coefficients on Lagged Deposition Measures on Crude Deaths, 95% CI

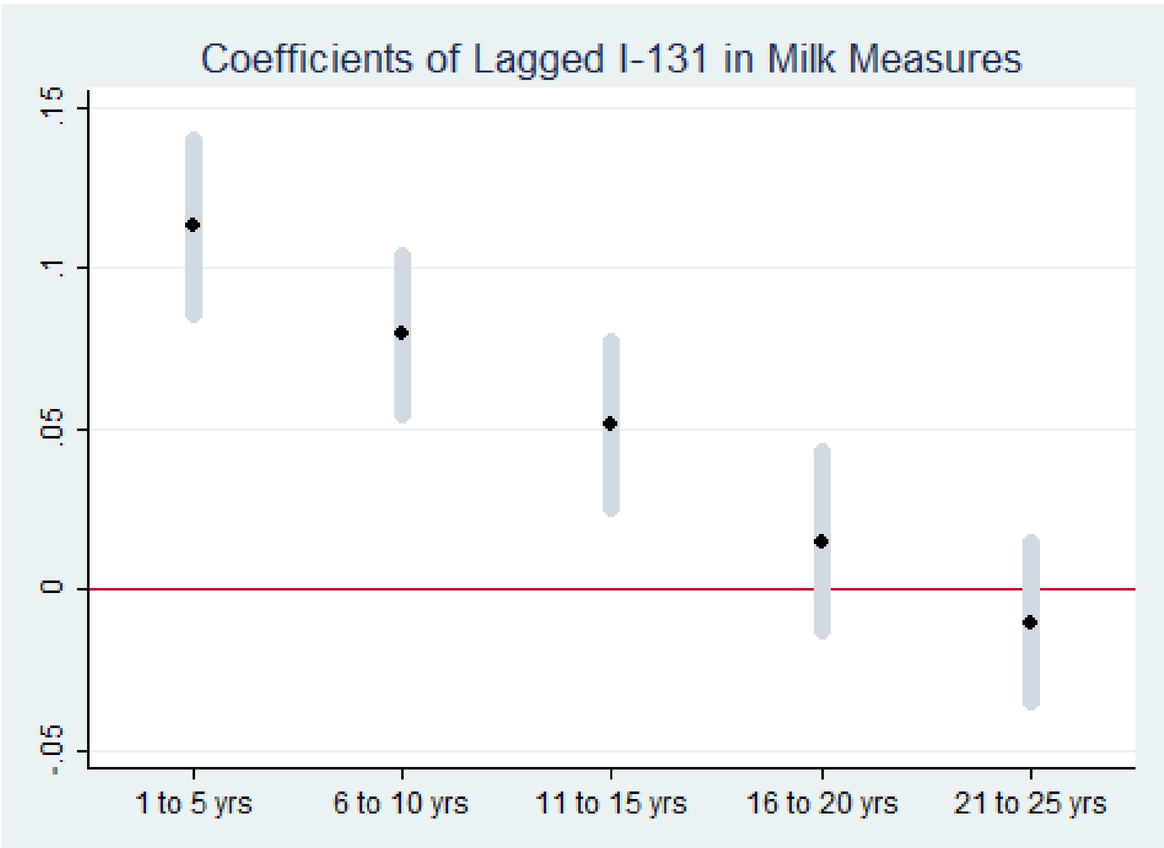

Figure 10: Coefficients on Lagged Milk Measures on Crude Deaths, 95% CI

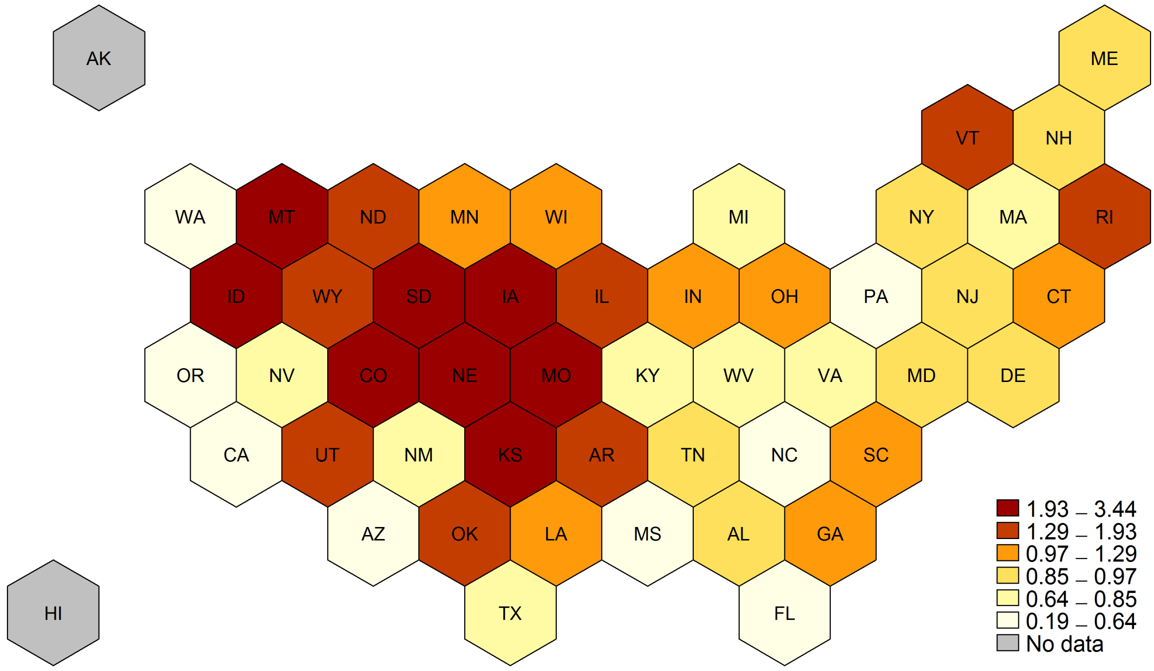

Figure 11: Average Increase in Crude Deaths Per 10,000 attributable to I-131 in Milk, 1951 to 1973

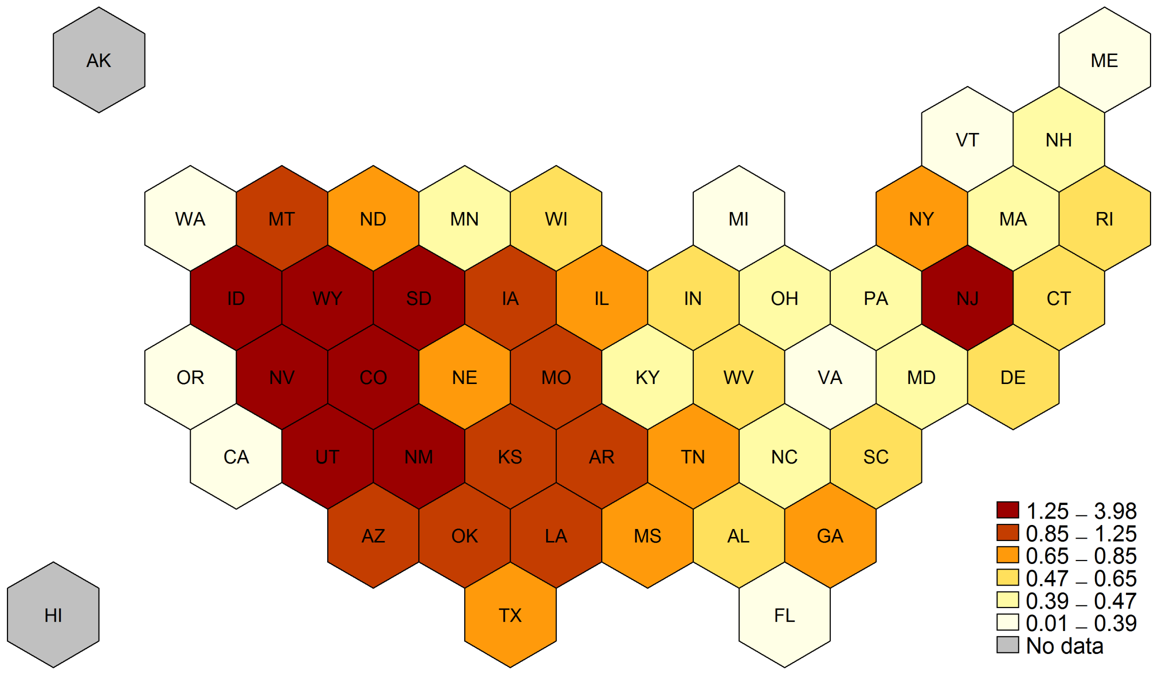

Figure 12: Average Increase in Crude Deaths Per 10,000 attributable to I-131 Ground Deposition, 1951 to 1973