The Siren Song of Cicadas: Early-Life Pesticide Exposure and Later-Life Mortality*

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Abstract

This paper studies the long-term effects of in-utero and early-life exposure to pesticide use on adulthood and old-age longevity. We use the cyclical emergence of cicadas in eastern half of the United States as a shock that raises the pesticide use among tree crop growing farmlands. We implement a triple-difference framework and employ Social Security Administration death records over the years 1975-2005 linked to the full-count 1940 census. We find that individuals born in top-quartile tree-crop counties and exposed to a cicada event during fetal development and early-life live roughly 2.2 months shorted lives; those with direct farm exposure face a reduction of nearly a year. We provide empirical evidence to rule out mortality selection before adulthood, endogenous fertility, and differential data linkage rates. Additional analyses suggests that reductions in education and income during adulthood are potential mechanisms of impact. Our findings add to our understanding of the relevance of early-life insults for old-age health and mortality.

Keywords: Mortality, Longevity, Pesticide, Agrichemicals, Fetal Development, Early-Life Exposures, Historical Data

JEL Codes: I18, J18, Q15, Q18, Q53

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* The authors claim that they have no conflict of interest to report. The authors would like to acknowledge financial support from NIA grants (R01AG060109, R01AG076830) and the Center for Demography of Health and Aging (CDHA) at the University of Wisconsin-Madison under NIA core grant P30 AG17266. We are extremely grateful to Charles Taylor for sharing his data on cicada events.

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1. Introduction

Studies in many settings document the adverse effects of environmental hazards such as pollution, radiation, toxic chemicals, agrichemicals, organic pollutants, and pesticides on human health outcomes (Agarwal et al., 2010; Aizer et al., 2018; Alharbi et al., 2018; Azizullah et al., 2011; Billings and Schnepel, 2018; Bove et al., 1995; Cachada et al., 2012; Currie et al., 2014; Currie and Schmieder, 2009; Currie and Schwandt, 2016; Lai, 2017; Lee et al., 2013; Nicolopoulou-Stamati et al., 2016; Rani et al., 2021; Sabarwal et al., 2018). As infants and children are vulnerable population who are at higher risks of negative exposures, a strand of this literature focus on fetal/childhood exposure to toxic pollutants and several short-term and long-term outcomes including birth weight, gestational age, neonatal mortality, infant mortality, cognitive measures, and academic outcomes (Bharadwaj et al., 2017; Billings and Schnepel, 2018; Chay and Greenstone, 2003; Currie et al., 2009; Currie and Schmieder, 2009; Guxens et al., 2018; Knittel et al., 2016; Margolis et al., 2021; Sanders, 2012). A narrower line of research evaluates the effects of pesticide and insecticide use and generally document negative impacts on infants' health outcomes through the adverse impacts on environment, air quality, and water quality (Bell et al., 2001, 2001; Bharadwaj et al., 2020; Brainerd and Menon, 2014; Mettetal, 2019; Regidor et al., 2004; Sonnenfeld et al., 2001; Syafrudin et al., 2021; Taylor, 2022). For instance, Taylor (2022) shows that increases in pesticide use during prenatal development is associated with reductions in gestational age and Apgar score. He also shows that infant mortality rates reveal a strong and robust association with rises in pesticide use in the previous year for recent cohorts. In the longer run, he provides empirical evidence that exposure during the year of birth leads to lower test scores and higher high-school dropout rates. While the focus of this literature is generally on infants' and children's outcomes, we are aware of no studies that explore the in-utero and early life exposure

to toxic pesticide use on old-age health outcomes, and specifically on old-age mortality. Indeed, it is a challenging question to answer due to data limitations—direct measures of pesticide exposure are available only recently, however in order to tie early life exposure to old age mortality, researchers must focus on exposures occurring 60-80 years ago. Our paper enters the literature at this point and aims to fill this gap by exploring fetal and early-life exposure to pesticides on old-age longevity.

We follow the methodology developed by Taylor (2022) and posit that the cyclical emergence of cicadas operate as an external shock followed by sharp increases in pesticide use among farmers, and specifically tree growers, as cicadas damage tree crops and not row crops. Therefore, we take advantage of differences in tree crop land use across counties coupled with county-year variations in cicada emergence. We then employ Social Security Administration death records linked to the 1940 census to infer the county of birth and explore the impact of being born in cicada exposure places on old-age longevity. We find sizeable and robust evidence of a negative impact. Among those born in the top quartile of tree crop counties, cicada exposure in-utero and early-life is associated with 2.2 months lower lifespan, conditional on survival up to age 36. We show the robustness of this effect across a wide array of alternative specification checks and functional from checks. We provide evidence to rule out the concern that endogenous survival of infants confounds the estimates. We also show that selective fertility and differential data linkage do not drive the results. Further heterogeneity analysis suggests effects that are considerably larger among those whose father's occupation is related to farming and are more likely to reside in the vicinity of croplands. Additional analyses using census data suggest that year-of-birth exposure to cicada in high tree crop county-of-birth is associated with reductions in completed education and slight drops in total personal income during adulthood. Although these estimates offer candidate

mechanisms, as several studies document the education-mortality and income-mortality relationships, we cannot rule out alternative mechanisms such as the adverse life-cycle outcomes associated with lower health endowment at birth.

This paper makes contributions to several strands of ongoing research. First, to the best of our knowledge, this is the first study to explore the in-utero and early-life exposure to toxic pollution caused by pesticide use on later-life and old-age longevity. Second, it adds to the literature on human health impacts of pesticide use and insecticide use by providing empirical evidence on its unexplored long-term effects. Third, it adds to the growing body of research on fetal and childhood origins of later-life outcomes by providing a link between early-life adverse environmental exposures and later-life mortality.

The rest of the paper is organized as follows. Section 2 reviews the relevant literature. Section 3 introduces data sources and sample selection strategy. Section 4 discusses the empirical method. Section 5 provides the empirical results. Section 6 offers empirical evidence for potential mechanism channels. We depart some concluding remarks in section 7.

2. Literature Review

Several studies suggest that prenatal exposure to toxic chemicals either through water or air lead to adverse birth outcomes and higher rates of fetal and infant death (Bove et al., 1995; Currie et al., 2009; Currie and Neidell, 2005; Currie and Walker, 2011, 2019; Hill, 2018; Hill and Ma, 2022; Isen et al., 2017; Knittel et al., 2016; Schlenker and Walker, 2016; Simeonova et al., 2021). For instance, Agarwal et al. (2010) explore the effects of toxic release from manufacturing industries on infants' health outcomes. They find that toxic air pollutants and specifically carcinogens are associated with adverse birth outcomes and higher rates of infant mortality. Hill and Ma (2022) explore the negative consequences of shale gas production under fracturing drilling

process on drinking water quality. They find large reductions in water quality following a new drilled shale gas well. They also show that, through deteriorations in water quality, shale gas development has negative impacts on infants' health outcomes. Currie and Walker (2011) take advantage of the introduction of electronic toll collection that reduced traffic congestion to explore the effects of pollution on infants' health outcomes. They show that the reductions in pollution significantly reduced the incidence of preterm birth and low birth weight among mothers residing in the vicinity of highways compared to those further away.

Currie et al. (2009) explore the effects of airborne pollution on infants' health outcomes using data from New Jersey. They employ mother fixed effect strategy to account for unobserved heterogeneity among infants with differential exposure to pollution and find negative and significant impacts specifically for third trimester exposures. Altindag et al. (2017) explore the health effects caused by pollution of Yellow Dust outbreaks, a natural phenomenon that starts primarily in China and moves to Korea in which a strong surface wind coupled with major storms pick up dry soil particles and builds miles-long dust clouds. They use Korean birth certificate data and show that the resulting pollution is associated with increases in low birth weight and preterm birth. Currie et al. (2013) employ the universe of birth records in New Jersey over the years 1997-2007 and implement family fixed effect models to compare variations in siblings' outcomes to explore the effect of water contamination level on birth outcomes. They find that among low educated mothers, water contamination is associated with lower birth weight and gestational age.

One strand of the literature explores the impacts of pesticide and insecticide use on infants and children health outcomes. For instance, Taylor (2022) explores the effect of pesticide use on infants' health measures exploiting emergence of cicadas as the shock that increases pesticide use in counties with higher concentration of tree crop products. He finds sizeable effects on next year

infant mortality rates. He also documents increases in low Apgar score and premature birth following the cicada-induced rises in pesticides. Brainerd and Menon (2014) explore the effect of fertilizer agrichemicals in water on infants' health outcomes. They use seasonal and spatial variation in plantation across Indian states as their source of variation in agrichemical pesticide use and show that exposure during pregnancy and early life is associated with higher neonatal death and infant mortality. Mettetal (2019) explores the effect of irrigation dam construction in South Africa on water quality and infants' health outcomes. She empirically documents that dam construction brings recycled waste agricultural water pollution back into the water system of the local area and hence reduces water quality. The reductions in water quality as a result of a new dam construction is associated with 10-20 percent rise in local infant mortality rates. Calzada et al. (2021) investigate the impact of aerial fumigation of banana plantations on infants' health outcomes. They show that pesticide exposure during the first trimester is associated with 38-89 grams lower birth weight.

These adverse impacts on infants and children can be translated into their later-life adverse outcomes. The negative in-utero and early-life exposures and lower initial health endowment could then be detected in their developmental outcomes (Boardman et al., 2002; Chatterji et al., 2014; Kieviet et al., 2009), Intelligence Quotient (Varella and Moss, 2015), test scores (Almond et al., 2015; Breslau et al., 2004; Litt et al., 2012; Majid, 2015), cognitive functioning (Løhaugen et al., 2010; Mamluk et al., 2021), completed education (Royer, 2009), adulthood earnings (Behrman and Rosenzweig, 2004; Black et al., 2007), hospitalization (Hummer et al., 2014; Pocobelli et al., 2016), disability (Almond and Mazumder, 2011; Arthi, 2018; Spracklen et al., 2017), chronic conditions (Hack et al., 2011), and old-age cognitive ability (Grove et al., 2017; Shenkin et al., 2009). For instance, Isen et al. (2017) exploit the changes in the 1970 Clean Air Act as the source

of differential improvements in air quality across counties and show that exposure to pollution in the year of birth leads to lower labor force participation and income during adulthood.

Several strands of literature document the association between any of the above-mentioned outcomes and old-age mortality (Cutler et al., 2006; Cutler and Lleras-Muney, 2006; Hayward and Gorman, 2004; Karas Montez et al., 2014; Smits et al., 1999). While later in the paper we directly test for education-income links as a potential mechanism channel, we do not rule out any of these candidate mechanisms as the link between cicada exposure during prenatal development and old-age mortality. Indeed, our paper joins the growing literature that documents the reduced-from effects of health endowment at birth and health accumulation during childhood on old-age mortality and longevity (Baker et al., 2008; Goodman-Bacon, 2021; Risnes et al., 2011; Samaras et al., 2003; Vaiserman, 2014).

3. Data Sources and Sample Selection

The primary source of data is Death Master Files (DMF) extracted from the Censoc Project (Goldstein et al., 2021). The DMF data covers deaths to male individuals reported by the Social Security Administration over the years 1975-2005. The Censoc project implements modern linkage techniques and use information reported in the DMF files to link the data with the full-count 1940 Decennial Census. The linkage is primarily based on name, gender, age, and place of birth. Therefore, the DMF-census linked dataset contains information on exact date of birth, exact date of death, a wide array of childhood family sociodemographic characteristics, and detailed granular geographic data for place of residence during early years of life. The constructed longitudinal data offers two aspects which makes it unique in addressing questions related to early-life conditions and old-age longevity. First, we can observe a wide range of early-life family-level covariates for a relatively large sample of individuals. Second, we have below-state geographic

identifiers, i.e., county, for early childhood that we can use as a proxy for county of birth. Very few other "big" datasets provide information on below-state geographic granularity for place of birth.

To infer county of birth from the 1940 census records and to reduce migration issues as much as possible, we impose three sample selection criteria. First, we exclude individuals whose state of birth is different than their state of residence in 1940. Second, the 1940 census asks for place of residence five years ago. In cases that individuals report that they have moved, they also report the county of residence in 1935. We use county of residence in 1935 as the default location of birth and use county of residence in 1940 as the county of birth in case of people who stayed in the same place over the last five years. Third, we focus on cohorts born between the years 1925-1940. The reason is that children usually leave their family households after age 16, which makes inference of birthplace based on current location more problematic.

The data on county-by-year level cicada events are taken from Taylor (2022). We merge this database with DMF-census data based on county-of-birth and year-of-birth. Since counties that experience cicada events are arguably different than other US counties for reasons that could also be correlated with their health trends, we focus only on counties that had any cicada event over the years that data was available (1915-2016). These sample selections leave us with 203,372 male individuals from 1,038 counties born between the years 1925-1940 and died between the years 1975-2005. Figure 1 depicts the geographic distribution of cicada counties by their number of cicada event experienced between the years 1925-1940. The distribution of age-at-death of individual observations in the final sample is shown in Figure 2. States with higher concentration of cicada events include Pennsylvania, New Jersey, Ohio, Connecticut, New York, West Virginia, Virginia, and Missouri. Summary statistics of the final sample is reported in Table 1. Average age-

at-death is roughly 65 years (779 months). About 6 percent of observations experience a cicada in their year of birth.

A cicada event triggers a sharp rises in the use of insecticides and pesticides. Since cicadas only damage tree crops and do not feed from agricultural row crops, the intensity of pesticides applications is much higher in areas with tree growing plants than areas with a higher share of crop production. Therefore, there are variations in pesticide application intensity based on the type of land use. We exploit this source of variation for the intensity of pesticide use by employing each county's concentration of apple production as a proxy. As shown in Table 1, the average intensity of apple production is 0.05 thousand bushels per square kilometer of county area. In addition, roughly 31 percent of observations live in counties that can be categorized as the fourth quartile of apple production per area.

4. Empirical Methodology

The primary purpose of the paper is to investigate the long-lasting effects of pesticide and insecticide use on human health and longevity. However, as we discussed our data and sample selection, we focus on cohorts born between the years 1925-1940. There is virtually no county level cropland data or county level pesticide use data available for this time period. Indeed, the county level measures of insecticide are only available for recent decades (i.e. post-1990). However, since cicada emergence occur in fixed cycles in specific locations, we are able to go back to the first half of the twentieth century and construct cicada exposure measures by county and year. The simple idea is that emergence of cicadas is associated with rises in pesticide use among tree cropland areas. Taylor (2022) uses county-level data from 1990-2016 and shows that pesticide use during cicada events increases by about 6-7 kg/km^2, off a mean of roughly 9 kg/km^2. Several case studies of older cicada emergences and other studies using more recent data also

confirm the sharp rise in pesticide use to control cicada events and protect tree crops (Ahern et al., 2005; Asquitii, 1954; Gaskin et al., 2012; Robinson et al., 2004; Steinhaus, 1957; Zaller, 2020). Therefore, we estimate a reduced-form analysis of cicada events on longevity as a measureable shock to pesticide exposure.

The empirical method takes advantage of variations in county and year of cicada events and the fact that this variation is more concentrated in counties with higher tree crop concentration. Following Taylor (2022), we use apple production intensity as a proxy for tree crop land use. Specifically, we compare the longevity of individuals who were born in county and years that experienced a cicada emergence in places with a higher intensity of apple production. We operationalize this method using the following triple-difference estimation method:

$$y_{ict} = \alpha_0 + \alpha_1 Event_{ct} \times Dosage_c + \alpha_2 X_{ict} + \alpha_3 Z_{ct} + \xi_t \times Event_{ct} + \zeta_c + \varepsilon_{ict} \qquad (1)$$

Where y is age at death for the individual observation i born in county c and year t. The parameter $Event$ refers to a cicada emergence in the respective county and year. It is a dummy variable that equals one if the county experience a cicada emergence in the year and zero otherwise. The parameter $Dosage$ represents the tree crop intensity in the county proxied by an indicator that equals one if per area apple production is at the fourth quartile and zero otherwise. In X, we include individual and family controls including race, ethnicity, paternal socioeconomic index dummies, and maternal education dummies. In Z, we include a series of county controls that are extracted from full-count decennial censuses 1920-1940 and interpolated for inter-decennial years, including share of immigrants, share of literate people, share of married people, and average occupational income score. Year fixed effects, represented by ξ, account for secular trends in longevity and health-related factors that affect individuals in all counties similarly but vary by time. The

parameter ζ represents the set of county fixed effects that absorb all time-invariant features of counties that may influence the long-term mortality outcomes.

Cicada events re-emerge on a periodical basis, specifically in 13 and 17-year cycles. Therefore, counties may anticipate this phenomenon by experience and respond in various ways not just by increasing pesticide use. To account for this unobserved feature change, we allow for time fixed effects to vary by the indicator of cicada event in a specific county-year. Finally, ε is a disturbance term. Following Taylor (2022), we cluster standard errors at the census division level to control for serial autocorrelation in the error term. In the robustness checks, we show that the results are robust to alternative clustering levels and also two-way clustering at the location-year levels. Since concentration of pesticides and their negative effects are expected to be larger in areas with higher population, we allow the regressions to assign more weights to areas with higher county-level population.

5. Results

5.1. Survival into adulthood

Before considering the main results, we explore several possible sources of bias. As shown by Taylor (2022), pesticide use during cicada events could lead to rises in infant mortality rates in the following years. The survival of infants could generate bias in our analyses if it leads to differences in the share of people in the final sample in ways that are correlated with their longevity. For instance, if whites are less likely to be affected by pesticide use during infancy the sample represents higher share of whites. Thus, regressions reveal lower marginal effects of cicada on longevity as whites have higher longevity for unobserved reasons that cannot simply be captured by white dummies in regressions. Similarly, the sample may contain more people with higher maternal education who also live longer lives for unobserved reasons related to their

maternal human capital and the subsequent intergenerational transmissions of human capital. The inclusion of a controls for maternal education does not solve the issue as they fail to absorb the unobserved features related to human capital of mothers. We explore these potential sources of bias by regressing a series of "pre-determined" observable characteristics on the main independent variables of equation 1, conditional on a full set of fixed effects. The results are reported in Table 2. There is no statistically significant association between a cicada event in top quartile apple counties and the individual's race, father's socioeconomics index, father's socioeconomic score being missing, and maternal education. The point estimates are also economically small and insignificant when we compare them with the mean of the outcome reported in row 4. For instance, the marginal effect of white suggests 0.15 percent change from the mean of the outcome. However, we do observe a negative correlation between missing maternal education and cicada exposure. Missing information could refer to the absence of mother for various reasons such as death or divorce. It could also imply that the mother is illiterate and did not reveal the literacy information. These possibilities usually are correlated with adverse outcomes among children (Beegle et al., 2006; Chen et al., 2009). If we believe that the adverse associations continue into old age and appear in longevity outcomes, the negative marginal effect of exposure on maternal education being missing (column 9) suggests that the regressions likely underestimate the true effects of pesticide exposure on mortality. However, we should be aware that only 5 percent of observations have missing maternal education. The subsequent bias is likely modest. Moreover, since there is no consistent pattern among different outcomes and this association is not accompanied by significant changes in other outcomes, it is not concerning.

5.2. DMF-Census Merging

Another concern is possible differential match rates in linking between DMF death records and the 1940 census. Although the linkage rule is primarily based on name, demographic features, birthplace, and age, it could be the case that certain demographic characteristics that are correlated with the likelihood of exposure are also more/less likely to lead to a successful DMF-Census match. Therefore, exposure measures become correlated with the likelihood of DMF-Census linkage. This correlation induces selection into our final sample since the linking rule is, by construction, based on observable features. For instance, if whites are more likely to appear in the linked sample (relative to other groups in the unlinked 1940 census sample) and assuming that exposure is correlated with linking rule, then the regressions of equation 1 underestimate the true effects as white have generally higher longevity. We can empirically examine this sources of bias by using the full sample of 1940 records before linking to the DMF records. We then implement the same sample selection criteria explained in section 3. We link this with DMF records and generate a dummy indicator for successful merging. We then regress the successful linking dummy variable on our measure of exposure, conditional on fixed effects. The results are reported in Table 3 across columns for different subsamples. In column 1 and for the full sample, there is no statistical association between cicada/pesticide exposure and successful merging indicator. The point estimate is economically small and suggests a mere 0.4 percent change from the mean of the outcome. We replicate this result for the subsample of whites, low-educated mothers, and low-socioeconomic status fathers in columns 2-4. We observe a similar insignificant association which rule out the concern over endogenous data linking.

5.3. Selective Fertility

Cicadas emerge on 13 and 17-year cycles. People may observe the cycle, gain knowledge of their re-appearance by experience, and prepare in many ways to face the phenomenon. One way to respond to this is selective fertility. Parents may choose to postpone their fertility either from the fear of the cicada's negative effects or from the knowledge of negative pesticide exposures. If fertility behavior changes by cicada exposure, then the composition of births in the treatment and control group could be different. This is primarily true if certain parental characteristics lead them to postpone their fertility and those characteristics are (in unobservable ways) correlated with their infants' health and later-life longevity. We test this selective fertility concern using county-level fertility data extracted from Bailey et al. (2016). We limit the sample to the same data years and counties as in our final sample. We then merge it with our cicada database and implement regressions that include county fixed effects and birth-year-by-event fixed effects. The results are reported in Table 4. We fail to find any association between cicada event exposure and total birth counts, log of birth counts, fertility rate, share of births to white women, and share of births to black women. The point estimates are economically small as compared to the mean of the outcomes reported in the fourth row. These results fail to provide empirical evidence to support the selective fertility concerns.

5.4. Main Results

The main results of the paper are reported in Table 5. We start with a parsimonious model that only includes county and birth year fixed effects. It suggests that among individuals residing in counties at the top quartile of apple production exposure to a cicada event at the year of birth is associated with 1.3 months lower longevity. We add event-by-birth-year fixed effects in column 2 to control for unobserved differences in year of birth among cicada exposed and unexposed

cohorts. The estimated coefficient rises by 62 percent and becomes statistically significant at the 5 percent level. We add family-level covariates in column 3 and several county controls in column 4. The estimated effect remains virtually constant and statistically significant. These intent-to-treat effects suggest a reduction in longevity by about 2.2 months. These estimated effects are arguably large in comparison with similar early life exposures documented in other studies. For instance, (Noghanibehambari and Fletcher, 2021) investigate the effects of alcohol availability during prenatal development and early life on long-term longevity outcomes. They exploit the early twentieth century prohibition movements across counties and years as the source of reductions in alcohol availability and show that exposed cohorts reveal a treatment-on-treated rise in longevity up to 1.7 months. These results also align with studies that show the adverse exposures during early life are associated with later life negative impacts on health outcomes and reductions in longevity (Hayward and Gorman, 2004; Karas Montez et al., 2014; Phillips et al., 2017; Schellekens and van Poppel, 2016; Zhang et al., 2020).

5.5. Robustness Checks

In this section, we explore the robustness of the main results to alternative specifications. We start by reporting the estimated coefficient of the fully parametrized model (from column 4 Table 5) in column 1 of Table 6. We then continue to test the robustness of the results in subsequent columns.

In the main results, the analysis sample was restricted to counties that experienced any cicada event over the sample period. In column 2, we extend the sample to include all counties in states that any of its counties experienced a cicada events. Therefore, the control group of this sample also includes non-cicada-counties in cicada-states. Furthermore, we extend our sample to

include all US counties and report the results in column 3. The estimated marginal effects in both samples are almost identical to that of column 1.

In column 4, we add more family-level covariates including a dummy to indicate father being a house owner, a dummy to indicate mother's labor force status, an indicator for mother being married, and father's reported wage in 1940. We also add more county-by-birth-year controls extracted from decennial censuses including average homeowners, share of people in blue-collar occupations, share of farmers, share of people in different age groups, average number of children less than 5 years old, share of females, share of whites, share of blacks, and share of Hispanics (column 5). These additional family and county controls do not change the estimated marginal effects.

In column 6, we allow for fixed effects of county to vary by race and parental characteristics. Thus, time-invariant features of counties could flexibly have differential influences in health (and subsequent longevity) of individuals with different parental education and socioeconomic status. That results in only 3 percent reduction in the marginal effect.

A small strand of studies suggests that there are seasonality in food quality and hence birth outcomes, the effects of which can be detected in old-age longevity (Doblhammer, 1999; A. Vaiserman, 2014, 2021). There is also evidence that several cause-specific deaths reveal seasonal patterns (Marti-Soler et al., 2014; Simmerman et al., 2009; Xuan et al., 2014). To account for these potential seasonal confounders, we add month-of-death and month-of-birth fixed effects. The result, reported in column 7, suggests slight increase in the marginal effect with respect to the baseline coefficient.

One concern in interpreting the result is that cicada events co-occur with higher concentration in regions of the country (e.g., east) that are on a path of converging to the health outcomes of other regions. Therefore, we expect that such path-convergence lead the estimates to underestimate the true effects. We add census-division-by-birth-year fixed effects to account for cross-region convergence in health outcomes across different cohorts. The results are reported in column 8. The marginal effect is about 15 percent larger than the main results.

In the next four columns, we check for sensitivity of the functional form of the outcome. In column 9, we replace the outcome with the log of age at death. Exposure in top-quartile counties is associated with a 0.3 percent reduction in age at death, roughly equivalent to the implied percent change from the mean in column 1 (2.2 versus 779 months average age-at-death). In columns 10-12, we replace the outcome with a dummy that indicates the age-at-death is greater than 55, 60, and 65 years, respectively. We observe negative and significant effects for all three alternative outcomes. For instance, column 12 suggests that year-of-birth exposure in top-quartile counties is associated with 1.2 percentage-points lower probability of living beyond age 65.

Since a cicada event has a staggered nature with heterogeneous effect based on the county tree crop production, the conventional OLS estimations are likely to provide biased estimates. We use the imputation technique developed by Borusyak et al. (2021) to re-evaluate the regression-produced estimates. The result is reported in column 13. The estimated marginal effect drops by 16 percent but remain statistically and economically significant.

In the main results, we cluster standard errors at the census-division level. In columns 14-16, we show that the results are robust to clustering at the state level, birth-year level, and two-way clustering at the census-division and birth-year level.

5.6. Exposure at Pre-prenatal and Postnatal Ages

In the main results, we merge cicada and DMF data at the birth-year (and county) level under the assumption that the in-utero period is a critical period during which exposures influence initial health endowment (Almond and Currie, 2011; Barker et al., 2002). However, air-water pollution could also affect postnatal age health outcomes. Moreover, if the prenatal and postnatal influences of pesticide pollution exposure is the channel, then we should observe no effect for periods before pregnancy. We explore these tests by including exposure measures at different ages in the regressions. Specifically, we assign exposure measures (and their interaction with the dosage variable) at ages -2 through +2. The results are reported in Table 7 across columns for different specifications. The fully parametrized model of column 3 shows that the effect on age -2 (i.e., exposure two years prior to birth) is quite small in magnitude and statistically insignificant. This can be considered a placebo test as we do not expect the cicada exposure and subsequent air-water pollution to affect those who have not yet been conceived. The effect starts to rise for age -1, those who were in-utero at the cicada event year. However, the effects reach the maximum (in magnitude) impact for year of birth exposure and become statistically significant. We should note that the control groups in this formulation consists of different cohorts than the main results and hence the estimated marginal effects. For instance, in the main results, we compare the outcome of exposure at year of birth with all other cohorts including those who were exposed at age -1, for whom column 3 implies an effect of -2.2. Therefore, it is not surprising to observe relatively smaller effects than the case in which the control groups exclude exposure at age -1.

In addition, we also observe some negative effects for postnatal ages. Column 3 suggests an effect of -0.4 for exposure at age 1 and -2.5 for exposure at age of 2. Therefore, although the

largest effects appear for in-utero exposure, we also find negative impacts for exposure during early years of life.

5.7. Heterogeneity across Subsamples

Several studies suggest that effects of exposure to air and water pollution varies across subpopulations with larger effects on minorities, low educated parents, and families of low socioeconomic status (Brainerd and Menon, 2014; Currie et al., 2013; Gray et al., 2013). In Table 8, we explore this potential heterogeneity by replicating the fully parametrized model equation 1 across subsamples based on sociodemographic characteristics. We observe substantially larger effects among nonwhites, though the effects are statistically insignificant partly due to much smaller sample size (column 1). The effects on whites is roughly half of the estimated coefficient in the main results and is statistically significant. We observe larger effects for those with low socioeconomic status fathers (-4.7 months) versus those with high socioeconomic status fathers (-1.1 months). However, both estimated coefficients are statistically insignificant, which restricts us from providing additional interpretations. We also observe larger effects among those with maternal education less than high school (column 6). The implied coefficient (-2.8 months) is statistically significant. However, we observe a positive and statistically insignificant coefficient among those whose maternal education is greater than or equal to high school (i.e., years of schooling≥9). Overall, these results provide suggestive evidence that the adverse effects are more pronounced for minorities and for children raised in poorer families.

5.8. Heterogeneity by Father Farmer Status

Studies that explore health impacts of exposure to pollution and specifically pesticide pollution highlight the heterogeneity in the effect by the dosage of exposures and the locality of exposure measures with largest effects among people who live/work in the vicinity of the source

of pollution (Agarwal et al., 2010; Hill and Ma, 2017). Therefore, one would expect to observe larger effects among people in the vicinity of tree croplands in general and apple orchards specifically. However, the public use 1940 census does not report geographic identifier below the county level. To infer the household's location of residence relative to farmland, we use information on father's occupation and focus on those who report working on a farm. The idea is that these individuals are more likely to reside in places close to a tree cropland than the general population. To explore this potential heterogeneity, we interact with the primary independent variables in equation 1 a dummy that indicates father's farmer status. The results are reported in Table 9. The triple interaction term suggests a reduction in longevity of about 10 months among those whose father's occupation is farmer. This effect is roughly 4.5 times that of the main effect for the general population. The coefficient is statistically significant at the 10 percent level.

6. Potential Mechanisms

Pesticide exposure leaves newborns and infants with lower health endowment at birth (Berkowitz et al., 2004; Nougadère et al., 2020; Taylor, 2022). A strand of the literature documents the association between measures of health at birth, e.g., birth weight, with later-life longevity (Baker et al., 2008; Belbasis et al., 2016; Risnes et al., 2011; Samaras et al., 2003; A. M. Vaiserman, 2018; Watkins et al., 2016). The primary argument of these studies relies on the Developmental Origins of Adult Health and Disease and changes in fetal programming due to adverse environmental shocks and external stressors. Other studies explore several other later-life outcomes that are influenced by health at birth. These studies show that a higher health capital at birth is associated with higher education and labor market outcomes during adulthood (Behrman and Rosenzweig, 2004; Bharadwaj et al., 2018; Black et al., 2007; Maruyama and Heinesen, 2020; Royer, 2009). Improved education and labor market outcomes in turn could affect longevity as

they can be translated into a healthier environment, better access to health-related information, better health insurance, and safer occupations (Chetty et al., 2016; Cutler and Lleras-Muney, 2010; Demakakos et al., 2015; Fletcher, 2015; Fletcher and Noghanibehambari, 2021; Halpern-Manners et al., 2020; Lleras-Muney, 2005; Lleras-Muney et al., 2020).

We add to our understanding of pesticide exposure and later-life mortality by exploring some potential mechanisms in line with this literature. The disadvantage of using 1940 census data is that children are at most 15 years old and have not completed their education. Moreover, the public-use 1950-onward censuses do not provide county identifiers for all counties. Ruggles et al. (2020) de-identify county identifier for a subset of counties (about 450 counties) based on other geographic variables and reports of population counts. In addition, from 1960-onward, census bureau provides Public Use Microdata Area (PUMA) geographic identifier. PUMA is a geographic boundary that depends on the population and hence vary over time. It contains several counties in rural areas with lower population density and becomes a sub-area of a county in urban areas with higher population density. We use 1960 census and construct a geographic variable that is the largest of county and PUMA. In urban areas where counties surpass PUMAs and the county identifier is available, our geographic measure equals county. In low population density areas where PUMAs cover several counties, our geographic variable equals PUMA. We then aggregate cicada database at the PUMA-county level and merge it with the 1960 census based on PUMA-county and year of birth. We then impose similar sample selection as the main results and implement regressions similar to equation 1 but replace the outcome with education-income profile of individuals. We do not include parental covariates in the regressions of this section as they are not available in the 1960 census.

The results are reported in Table 10 across columns for different outcomes. Cicada exposure for individuals at the top-quartile of PUMA-county apple production is associated with roughly 0.1 fewer years of schooling, 1.5 percentage-points lower probability of having any college education, $57 lower annual total personal income, and 1 unit lower socioeconomic index. These coefficients can be compared with the mean of the outcome and be translated into the percent change from the mean: 1.2, 6.7, 1.7, and 3.3 percent reduction from the mean of their respective outcomes. Except for income, the effects on other outcomes are statistically significant at 10 percent.

There are two notes that we should consider in interpreting these results. First, apple orchards cover a relatively small area of a given county. Exposed people (treated people versus those intent-to-treat observations) constitute a smaller fraction of all people in the county. Aggregating the data at the county-PUMA level exacerbates this issue. In Appendix A, we explore this error in measurement of the treatment. In so doing, we replicate the main results (reported in Table 5) in a sample that exploits the variation at the county-PUMA level instead of county level. We implement regressions similar to equation 1 but replace the county fixed effects with county-PUMA fixed effects. The results suggest slight drops in marginal effects compared to those of Table 5. Therefore, we may expect that the true intent-to-treat effects of Table 10 were slightly larger had we had the county identifier in the 1960 census. Second, we should also note that the PUMA-county of observation in the 1960 census does not fully reveal the PUMA-county of birth. The subsequent migration issues add measurement errors into analyses of this section and we should exercise caution in interpreting these results. However, the observed negative effects provide suggestive evidence that reductions in education and income could be potential mechanism channels.

7. Conclusions

While pesticides are necessary tools to increase productivity of the agricultural sector, they come at a cost to the environment and human health. Understanding these negative externalities are important for policymakers in the agriculture industry to regulate agrichemical use in farm lands. The literature so far has provided evidence of the effects on short-run and medium-run outcomes for recent exposures. The current study is the first to evaluate exposure to pesticide on old-age mortality, based on pesticide practices in the early 20th century. We provide empirical evidence that fetal and early-life exposure to rises in pesticide use as a response to cyclical emergence of cicadas are associated with lower lifespan during adulthood and old ages. The estimated intent-to-treat effect suggests a reduction of 2.2 months of longevity for those born in top-quartile apple production counties and during a cicada event year. However, this effect is driven by a very small portion of the population (i.e., those in the vicinity of an apple orchard) in a small subset of counties (i.e., those at the top quartile and exposed to cicada) and under a relatively uncommon event (i.e., 13 and 17-year cycles of cicada). In our heterogeneity analysis, we show a 10 month reduction in longevity for those whose father's occupation is farmer and are more probable to live in the vicinity of tree croplands. In addition, apple orchards account for less than 0.1 percent of US croplands and apple growers' use of pesticides add up to roughly 4.5 percent of all pesticides (Fernandez-Cornejo et al., 2014). Therefore, we would expect larger effects among populations at risk of other pesticide use and other croplands.

To gauge the potential life-years lost due to pesticide exposure during in-utero, we estimate a back-of-an-envelope calculation with simplified assumptions. Our final sample is drawn from specific cohorts born in specific counties. In 1940, these cohorts born in cicada event years in counties at the top quartile apple production add up to a population of roughly 300K individuals.

Assigning the intent-to-treat effects of 2.2 months to the population, we reach 55,000 life-years lost only for those cohorts and only for cicada-induced rises in pesticide exposure. We can also extrapolate this number for all croplands and pesticides while still working with the same cohorts. Using the share of pesticides use in apple croplands (Fernandez-Cornejo et al., 2014), we can also scale this number up and reach a value of 1.2 million life-years lost due to in-utero exposure to pesticide use.

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Tables

Table 1 - Summary Statistics

Variable	Mean	Std. Dev.	Min	Max
Death Age (Months)	779.002	103.719	434	959
Birth Year	1929.644	2.932	1926	1940
Death Year	1994.543	8.302	1975	2005
Cicada Event × Quartile 4 Apple Bushels	0.025	0.155	0	1
Cicada Event	0.063	0.242	0	1
Apples Bushels 1964 Per County Area (1,000 bushels per Km^2)	0.052	0.184	0	3.182
Quartile 1 Apple	0.078	0.269	0	1
Quartile 2 Apple	0.262	0.439	0	1
Quartile 3 Apple	0.352	0.477	0	1
Quartile 4 Apple	0.308	0.462	0	1
White	0.937	0.244	0	1
Black	0.062	0.242	0	1
Other Races	0.001	0.032	0	1
Father is Farmer	0.289	0.453	0	1
Father's Socioeconomic Index Quartile 1	0.275	0.447	0	1
Father's Socioeconomic Index Quartile 2	0.251	0.434	0	1
Father's Socioeconomic Index Quartile 3	0.213	0.41	0	1
Father's Socioeconomic Index Quartile 4	0.26	0.439	0	1
Father's Socioeconomic Index Missing	0.132	0.338	0	1
Mother's Education <HS	0.663	0.473	0	1
Mother's Education =HS	0.229	0.42	0	1
Mother's Education College-More	0.045	0.206	0	1
Mother's Education Missing	0.063	0.243	0	1
County Population	524283.55	995181.05	3769	4068453
Average Number of Children<5 Years Old Nchild5	0.387	0.118	0.17	0.994
Share of First-Generation Immigrants	0.101	0.091	0	0.319
Share of Second-Generation Immigrants	0.19	0.146	0	0.46
Average Occupational Income Score	24.414	3.773	14.033	29.62
Share of Literate	0.849	0.175	0	0.997
Share of Married	0.611	0.028	0.507	0.697
Observations	203,372			

Table 2 - Balancing Test

	Outcomes:								
	White	Black	Other	Father's SEI	Father's SEI Missing	Mother's Education<HS	Mother's Education=HS	Mother's Education>HS	Mother's Education Missing
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Cicada Event × Quartile 4 Apple Bushels	-0.00148	0.0015	-0.00002	0.44773	-0.00378	0.00213	0.00711	0.00137	-0.01061**
	(0.00213)	(0.00226)	(0.00024)	(0.30631)	(0.00336)	(0.00296)	(0.00558)	(0.00242)	(0.0035)
Observations	203372	203372	203372	203372	203372	203372	203372	203372	203372
R-squared	0.07862	0.0788	0.01747	0.02486	0.00708	0.02053	0.02029	0.00629	0.0094
Mean DV	0.964	0.035	0.001	27.552	0.125	0.682	0.227	0.040	0.051
County and Birth-Year FE	✓	✓	✓	✓	✓	✓	✓	✓	✓
Event-by-Birth-Year FE	✓	✓	✓	✓	✓	✓	✓	✓	✓

Standard errors, clustered at the census division of birth level, are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 3 - Endogenous Merging

	Outcome: Successful Merging with the Original Population (Dummy), Subsamples:			
	Full Sample	Whites	Mother Education<HS	Father SEI<Median
	(1)	(2)	(3)	(4)
Cicada Event × Quartile 4	0.00022	-0.00043	-0.00002	-0.00157
Apple Bushels	(0.00129)	(0.00114)	(0.00111)	(0.00134)
Observations	3685314	3346527	2276045	1695551
R-squared	0.00509	0.00506	0.0055	0.00569
Mean DV	0.050	0.051	0.053	0.050
County and Birth-Year FE	✓	✓	✓	✓
Event-by-Birth-Year FE	✓	✓	✓	✓

Standard errors, clustered at the census division of birth level, are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 4 - Endogenous Fertility

	Outcomes:				
	Total Birth Counts	Log Total Births	Total Births per Women	Share of Births to Whites	Share of Births to Blacks
	(1)	(2)	(3)	(4)	(5)
Cicada Event × Quartile 4 Apple Bushels	36.10604	-0.00524	-0.07618	0.00673	-0.0005
	(195.69181)	(0.01664)	(0.47973)	(0.01655)	(0.00301)
Observations	6297	6297	6297	6297	6288
R-squared	0.99778	0.99556	0.83559	0.95352	0.9854
Mean DV	7055.400	7.560	38.846	0.712	0.286
County and Birth-Year FE	✓	✓	✓	✓	✓
Event-by-Birth-Year FE	✓	✓	✓	✓	✓
County Controls	✓	✓	✓	✓	✓

Standard errors, clustered at the year level, are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 5 - Main Results for Exposure at Year of Birth

	Outcome: Age at Death (Months)			
	(1)	(2)	(3)	(4)
Cicada Event × Quartile 4	-1.33764	-2.1646**	-2.12428**	-2.22169***
Apple Bushels	(0.70062)	(0.66248)	(0.67217)	(0.54418)
Observations	203372	203372	203372	203372
R-squared	0.07706	0.07712	0.07731	0.07732
Mean DV	779.353	779.353	779.353	779.353
County and Birth-Year FE	✓	✓	✓	✓
Event-by-Birth-Year FE		✓	✓	✓
Family Controls			✓	✓
County Controls				✓

Standard errors, clustered at the census division of birth level, are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 6 - Robustness Checks

	Column 5 Table 5	Sample: All States with Ever-Any Cicada Event	Sample: All US Counties	Additional Family Controls
	(1)	(2)	(3)	(4)
Cicada Event × Quartile 4 Apple Bushels	-2.22169***	-2.21768***	-2.20768**	-2.22447***
	(0.54418)	(0.57553)	(0.66266)	(0.54973)
Observations	203372	361751	441909	203356
R-squared	0.07732	0.07849	0.0792	0.07754
	Additional County Controls	Adding County-by-Race and County-by-Parental-Covariates Fixed Effects	Adding Birth-Month and Death-Month FE	Adding Division-Birth-Year FE
	(5)	(6)	(7)	(8)
Cicada Event × Quartile 4 Apple Bushels	-2.21436**	-2.16265***	-2.27114***	-2.56644**
	(0.668)	(0.51689)	(0.58322)	(0.78312)
Observations	203372	203184	203372	203372
R-squared	0.07739	0.07902	0.07907	0.07764
	Outcome: Log Age at Death	Outcome: Death Age>55	Outcome: Death Age>60	Outcome: Death Age>65
	(9)	(10)	(11)	(12)
Cicada Event × Quartile 4 Apple Bushels	-0.00301**	-0.00826***	-0.01618***	-0.01237**
	(0.00082)	(0.00087)	(0.0031)	(0.00462)
Observations	203372	203372	203372	203372
R-squared	0.07404	0.01818	0.01645	0.04322
	Borusyak et al. (2021) Diff-in-Diff	Clustering SE at State -Level	Clustering SE at Birth-Year-Level	Clustering SE at Division-Year
	(13)	(14)	(15)	(16)
Cicada Event × Quartile 4 Apple Bushels	-1.86458***	-2.22169*	-2.22169**	-2.22169***
	(0.4560855)	(1.16959)	(1.00588)	(0.84528)
Observations	173874	203372	203372	203372
R-squared	---	0.07732	0.07732	0.07732

Standard errors, clustered at the division of birth level (except for column 14-16), are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 7 - Exposure to Cicada at Different Ages

	Outcome: Age at Death (Months)		
	(1)	(2)	(3)
Interaction Coefficient of Cicada Event × Quartile 4 Apple Bushels for Assignment of Event at Age:			
-2	-0.65186	-0.62595	-0.84431
	(3.18059)	(3.17803)	(3.34794)
-1	-2.27681	-2.20945	-2.50567
	(2.0967)	(2.09032)	(1.97028)
0	-3.01703***	-2.97999***	-3.40893***
	(0.60554)	(0.60087)	(0.53452)
1	0.06574	0.03929	-0.45
	(1.81873)	(1.79811)	(1.64134)
2	-1.95303**	-1.99592**	-2.51563**
	0(.73679)	(0.72234)	(0.89659)
Observations	203372	203372	203372
R-squared	0.07733	0.07752	0.07753
Mean DV	779.353	779.353	779.353
Birth-year-by-Event FE	✓	✓	✓
County and Birth-Year FE	✓	✓	✓
Individual Controls	✓	✓	✓
Family Controls		✓	✓
County Controls			✓

Standard errors, clustered at the census division of birth level, are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 8 - Heterogeneity across Subsamples

	Outcome: Age at Death (months) *Subsamples:*					
	Nonwhite	Whites	Father's SEI<Median	Father's SEI>Median	Mother's Education<HS	Mother's Education≥HS
	(2)	(3)	(4)	(5)	(6)	(7)
Cicada Event × Quartile 4	-25.55464	-1.03409**	-4.72658	-1.07049	-2.78665**	1.14394
Apple Bushels	(17.65692)	(0.36952)	(2.71057)	(1.41543)	(1.09385)	(2.00036)
Observations	12776	190496	107086	96276	147658	55704
R-squared	0.0999	0.07537	0.07565	0.07962	0.07559	0.0849
Mean DV	755.114	780.259	778.598	779.732	780.631	775.840
Birth-year-by-Event FE	✓	✓	✓	✓	✓	✓
County and Birth-Year FE	✓	✓	✓	✓	✓	✓
Individual-Family-County Controls	✓	✓	✓	✓	✓	✓

Standard errors, clustered at the census division of birth level, are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 9 - Heterogeneity by Father Farmer Status

	Outcome: Age at Death (months)		
	(1)	(2)	(3)
Cicada Event × Quartile 4 Apple Bushels × Father Farmer	-10.28714*	-10.33739*	-10.44854*
	(4.7794)	(4.86611)	(4.95677)
Cicada Event × Quartile 4 Apple Bushels	-1.44452*	-1.42645*	-1.5201**
	(0.72874)	(0.73398)	(0.57972)
Father Farmer × Cicada Event	3.46267	3.43314	3.54316
	(5.02332)	(5.10882)	(5.13535)
Father Farmer × Quartile 4 Apple Bushels	-3.53741	-3.68501	-3.67412
	(3.32306)	(3.39276)	(3.40208)
Father Farmer	8.36673**	8.98033***	8.96225***
	(2.57643)	(2.40127)	(2.40816)
Observations	203372	203372	203372
R-squared	0.07728	0.07748	0.0775
Mean DV	779.353	779.353	779.353
County and Birth-Year FE	✓	✓	✓
Event-by-Birth-Year FE	✓	✓	✓
Family Controls		✓	✓
County Controls			✓

Notes. Standard errors, clustered at the census division of birth level, are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Table 10 - Potential Mechanisms Using 1960 Census (PUMA Level)

	Outcomes:			
	Years of Schooling	Education≥College	Total Personal Income	Socioeconomic Index
	(1)	(2)	(3)	(4)
Cicada Event × Quartile 4 Apple Bushels	-0.09785*	-.01477*	-56.67625	-1.0806*
	(0.04617)	(0.00721)	(55.89558)	(0.50105)
Observations	318057	318057	318057	304708
R-squared	0.12047	0.05511	0.2738	0.12573
Mean DV	8.024	0.231	3339.514	33.613
%Change	-1.219	-6.393	-1.697	-3.215
Birth-year-by-Event FE	✓	✓	✓	✓
County-PUMA and Birth-Year FE	✓	✓	✓	✓
Individual Controls	✓	✓	✓	✓

Notes. Standard errors, clustered at the division of birth level, are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Figures

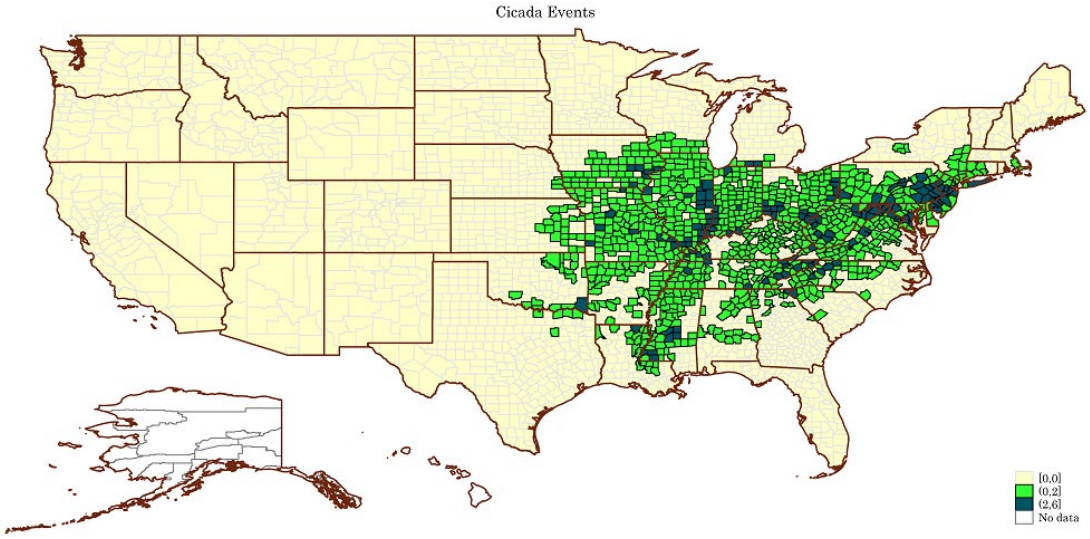

Figure 1 - Geographic Distributions of Cicada Events

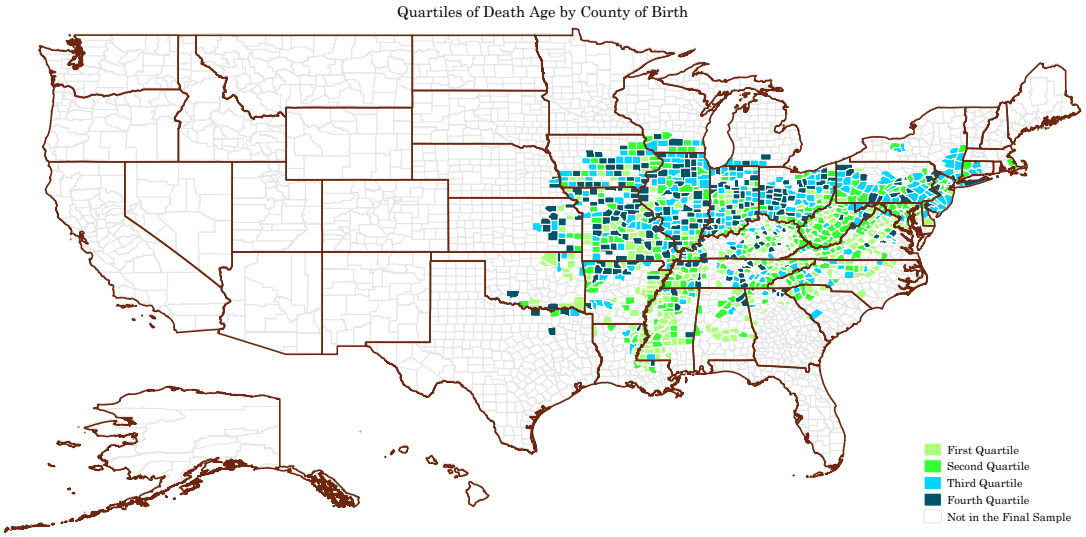

Figure 2 - Distribution of Age at Death in the Final Sample

Appendix A

In the paper, we explored the mechanisms of impact using the 1960 census data and aggregating the data at the county-PUMA level since the census suppresses the county identifier in the public-use data releases. One concern in interpreting those results (reported in Table 10) is measurement error due to aggregation. Since the new geographic identifier is (in many instances) larger than the county, the assignment of the treatment contains error since fewer people are treated in relatively larger geographic boundary. We gauge the relevance of this measurement error by aggregating the main data at the PUMA-county level and implement regressions similar to those reported in Table 5 and replace the county fixed effect with county-PUMA fixed effects. We report the results in Appendix Table A-1. The estimated marginal effects are slightly lower than those reported in Table 5. For instance, the full specification of column 4 suggests a drop in the coefficient of about 5 percent. However, the effect is still statistically significant. This relatively robust estimation suggests that the effects presented in Table 10 are likely robust to aggregation. The estimated coefficients could be even slightly larger had we had county identifier in the census rather than aggregating at the county-PUMA level.

Appendix Table A-1 - Cicada Exposure at Year of Birth at the PUMA Level

	Outcome: Age at Death (Months)			
	(1)	(2)	(3)	(4)
Cicada Event × Quartile 4	-1.29841	-2.11442**	-2.07517**	-2.08306**
Apple Bushels	(0.7163)	(0.67398)	(0.68379)	(0.60813)
Observations	203182	203182	203182	203182
R-squared	0.07695	0.077	0.0772	0.07721
Mean DV	779.302	779.302	779.302	779.302
County-PUMA and Birth-Year FE	✓	✓	✓	✓
Event-by-Birth-Year FE		✓	✓	✓
Family Controls			✓	✓
County Controls				✓

Standard errors, clustered at the census division of birth level, are in parentheses.
*** p<0.01, ** p<0.05, * p<0.1