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The siren song of cicadas: Early-life pesticide exposure and later-life male 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 the eastern half of the United States as a shock that raises the pesticide use among tree crop growing farmlands. We implement a difference-in-difference framework and employ Social Security Administration death records over the years 1975–2005 linked to the complete count 1940 census. We find that males 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 examine 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.

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 a 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

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generally document negative impacts on infants' health outcomes through the adverse impacts on environment, air quality, and water quality (Bell et al., 2001a, 2001b; 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 scores. 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 that occurred to male individuals between 1975-2005[1] linked to the 1940 census to infer the county of birth and explore the impact of being born in places experiencing cicada exposure 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 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. There is a relatively large literature that documents the strong associations between in-utero exposure to water and airborne pollution and birth outcomes (Currie et al., 2009, 2015; Currie and Neidell, 2005; Currie and Schmieder, 2009; Currie and Walker, 2011; Dave and Yang, 2022). In addition, a strand of research suggests long-term and life-cycle effects of health endowment at birth (Behrman and Rosenzweig, 2004; Black et al., 2007; Cook and Fletcher, 2015; Maruyama and Heinesen, 2020; Royer, 2009). However, fewer studies examine the long-term effects of fetal experience of pollution and long-term outcomes (Bharadwaj et al., 2017; Currie et al., 2014; Rosales-Rueda and Triyana, 2019). The evidence is scarce for old-age health and mortality outcomes. Studies that assess this long-term link usually rely on cross-sectional analyses to compare people who were born in more/less polluted areas and fail to account for the fact that there are inherent differences in these comparisons that are difficult to control for by including observable measures (Phillips et al., 2017). Our study departs from this literature by evaluating the impacts of rises in pesticide use as a result of a plausibly exogenous influencer. We implement various tests to add to the validity of our identification strategy.

Second, we add to the literature documenting the human health impacts of pesticide use and insecticide use by providing empirical evidence on its unexplored long-term effects. Following the introduction of agrichemicals during the 19th and 20th century, there has been public debates and policy concerns over potential health effects of their applications (Barnes, 1950; Biggs et al., 1945; Guiteras, 1909; Journal and Gear, 1921). There are still numerous reports of health impacts of agrichemical use, specifically in developing countries with fewer regulations and lower requirements of monitoring (Brainerd and Menon, 2014; Lai, 2017; Maumbe and Swinton, 2003; Negatu et al., 2021). This paper adds to these discussions by showing long-lasting effects among exposed infants. 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.

We should note that although we look at exposure to agrichemicals of the early decades of the 20th century in a wholly unregulated environment with limited information, the results of this paper have important policy implications with contemporary relevance. While the primary pesticides of the early 20th century evolved rapidly and changed composition over the decades, chemicals such as arsenic and lead arsenic remained in place for several decades afterwards. As we discuss in section 2.1, arsenic was not banned until 1988. Therefore, the findings of the paper have important implications for many cohorts post-1940 that could be exposed during early-life to sets of agrichemicals containing arsenic and lead. Further, many countries in the world still use a variety of these chemicals

[1] The data source of this paper covers substantially wider death years than alternative data (e.g., Numident). The drawback is that it contains only males. We should acknowledge that the results could be different for women and also their policy implications. For instance, there is evidence that male infant mortality is higher in the presence of negative shocks under the sol-called *fragile male* hypothesis (Clark et al., 2021; Drevenstedt et al., 2008; James and Grech, 2017; Rosa et al., 2019; Weinberg et al., 2008). Therefore, surviving male individuals are likely carry a *selection effect* and the observed effects could be larger for females who experienced lower mortality rates during infancy as a result of a negative shock.

either because of limited regulation or due to costly elimination and substitution of these products. For instance, a relatively large literature points to the arsenic crisis in Bangladesh with millions of exposed people (Ahmad et al., 2018; Caldwell et al., 2003; Chakraborti et al., 2015). In addition, the results of the paper point to effects that can be detected many decades later. Therefore, it raises critical questions regarding the safety and regulation of present-day pesticide use.

The rest of the paper is organized as follows. Section 2.2 provides a background on pesticide use in the sample period and 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 8.

2. Background

2.1. Pesticide use in the early 20th century

During the late 19th and early 20th centuries, developed countries experienced an unprecedented demographic transition following population growth, rises in income, and migration to urban areas (Lee and Reher, 2011). This fact changed the demand for agricultural products in ways that low-yield conventional methods would not meet growing demand. Pest management and the rise of pest specialists became the front line of attempts to increase agricultural productivity both in the US and Europe. Innovative pest management specialists started experimenting with various formulations of inorganic pesticides, including arsenic (As), copper (Cu), Lead (Pb), and Sulfur (S). On the other end, apple production had become widespread, specifically in eastern states of the US during the early 20th century. For instance, in 1925, roughly 300,000 acres of land were allocated for apple production, roughly 1 percent of the state's land area (Taylor and Parsons, 1926). This resulted in a harvest of more than 15 million bushels of apples per year (Mattice, 1927). Virginia's apple production today is less than 5 million bushels per year (Schooley et al., 2009). To raise yield-per-acre, cropland farmers started to add extensive amounts of inorganic compounds, including Bordeaux mixture, lime-sulfur, calcium arsenate, and specifically lead arsenate. Total agrichemicals containing arsenic increased from 72,000 (lb) in 1900 to roughly 12.8 million (lb) in 1920 and 39.2 million (lb) in 1940 (Murphy and Aucott, 1998). Later regulations following health concerns and introductions of new insecticides limited their use to below 1 million per year in the late 20th century (Murphy and Aucott, 1998). Arsenic was finally banned in 1988 (Peryea, 1998). An important disadvantage of these agrichemicals is they can easily penetrate through the soil to groundwater and are easily soluble in water, hence resulting in local water pollution. Several studies using data from different countries and time periods provide evidence of negative health impacts of lead and arsenic, specifically among infants and children, with a long-lasting legacy for life-cycle outcomes (Dave and Yang, 2022; Gazze and Heissel, 2021; Gilbert-Diamond et al., 2016; Gustin et al., 2020; Jelliffe-Pawlowski et al., 2006; Laine et al., 2015; Needleman et al., 2010; Perkins et al., 2014; Xie et al., 2013). For instance, Liao et al. (2018) show that an increase of one percent in maternal urinary arsenic levels is associated with roughly 50–70 g lower birth weight of infants.

Another concern of lead arsenate use as a pesticide is its enduring environmental persistence. There are estimates that, although lead arsenate has been eliminated from the list of agrichemicals for decades, millions of acres of land still remain contaminated (Hood, 2006). For instance, Murphy and Aucott (1998) estimate that in New Jersey, between 1900 and 1980, about 49 million pounds of lead arsenate and 18 million pounds of calcium arsenate were added to the orchard soils. We should note that to the extent that the impacts are cumulative and the application of agrichemicals could contaminate the soil and water for the later periods, results using our research design will underestimate the true effects. It is likely that a portion of the reference cohorts born in non-cicada years is also damaged by the negative impacts of persistent soil-water contaminations over the years.

2.2. 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). One important array of toxic chemicals include arsenic and lead, chemicals which have been widely used in agrichemical compounds during the period of the study (refer to section 2.1) (Smith and Steinmaus, 2009). For instance, Milton et al. (2005) use a cross-sectional data from Bangladesh and reports correlations between maternal arsenic exposure in drinking water and risks of stillbirth and neonatal death. Signes-Pastor et al. (2019) examines the effects of in utero exposure to arsenic, lead, and manganese on birth outcomes. They extract postpartum toenail samples of mothers as a biomarker of in utero exposure to these toxic chemicals from a sample of mother-infant pairs in the New Hampshire Birth Cohort Study data. They find reductions in the newborns' anthropometric outcomes as a result of exposure, including head circumference, length, and weight. Chou et al. (2014) use data from Taiwan and extract maternal urine samples during third trimester to measure inorganic arsenic concentration. They find associations between concentration and DNA damage biomarkers, indicators of changes in epigenetic regulation which affects health of newborns. They also report significant

reductions in infants' Apgar scores as a result of a higher arsenic concentration.[2]

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 scores 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 g lower birth weight.

These adverse impacts on infants and children continue to be seen in later-life outcomes. The negative in-utero and early-life exposures and lower initial health endowment are detected in developmental outcomes (Boardman et al., 2002; Chatterji et al., 2014; De 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, Butler et al. (2023) employ data from the New Hampshire Birth Cohort Study and show that arsenic exposure during pregnancy is associated with lower motor functioning and overall motor proficiency.[3] Billings and Schnepel (2018) employ linked administrative data from Charlotte, NC to examine the effects of early-life intervention to alleviate blood lead level on a wide array of long-term outcomes. They find that interventions resulted in mitigating or reversing the negative effects on outcomes previously documented in the literature, including test scores, educational outcomes, and juvenile crime. Fletcher and Noghanibehambari (2022) examine the effects of in-utero and early-life exposure to lead in drinking water and later-life longevity. They exploit the staggered water-pipe installation across several US cities during the early 20th century in combination with the pipe materials (lean/non-lead). They find significant reductions of about 1.6 months as a result of intent-to-treat effects of being born post-waterwork in a city with lead pipes.

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; Montez and Hayward, 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, 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 males. 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

[2] One strand of research examines the effects of toxic and industrial chemicals on infants' health outcomes. 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 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.

[3] Motor proficiency tests aim at examining a range of physical abilities and progressions in the control and coordination of body movements.

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 1038 counties born between the years 1925–1940 and died between the years 1975–2005. Fig. 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 Fig. 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 rise 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 to 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 measurable 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.[4] 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. Therefore, the treated population is those born during a cicada event in high-intensity production counties. We operationalize this method using the following difference-in-difference estimation method:

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

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.[5,6] In X, we include individual and family controls including race,

[4] The focus of the empirical method on exploiting variations in tree crop intensity is important to identify the treatment group. In Appendix D, we exclude this term from regressions of Equation (1) and focus on variations of cicada event across all counties in the sample. We then restrict the sample to those at the top-quartile of apple production and find that the effect of the latter sample is about 10 times larger than the former sample. Moreover, the estimated difference-in-difference effect of the top-quartile sample is quite comparable to the main results presented in Table 5.

[5] In Appendix B, we show quite robust results when we replace this with indicators of above-median, above-6th-decile, above-7th-decile, above-8th-decile, as well as a continuous measure of apple production per area.

[6] The apple production values are measured in year 1967. The detailed county level information on apple production, to our knowledge, is very limited for years closer to the 1930s. For instance, the well-known agricultural data under the "Great Plains Population and Environment Data" by Gutmann (2005) provides information on various croplands on a 5-year interval. However, it does not report any apple or fruit production. For a subset of counties, it reports total sales from all fruits and nuts in dollars. However, after merging with the final sample, we are left with only 8K observations in 100 counties. Using an indicator of top-quartile fruit sale and implementing regressions similar to Equation (1) produces an insignificant difference-in-difference estimate of −6.24 (se = 2.43). Although it implies a negative and much larger coefficient than our main results (discussed in section 5.4), the regression suffers from statistical power and limited source of treatment variation. Overall, we should note that tree crops are considered long-term investments and their value depreciates over several decades. Hence, there is little room for a concern about noticeable change in tree cropland use of a county.

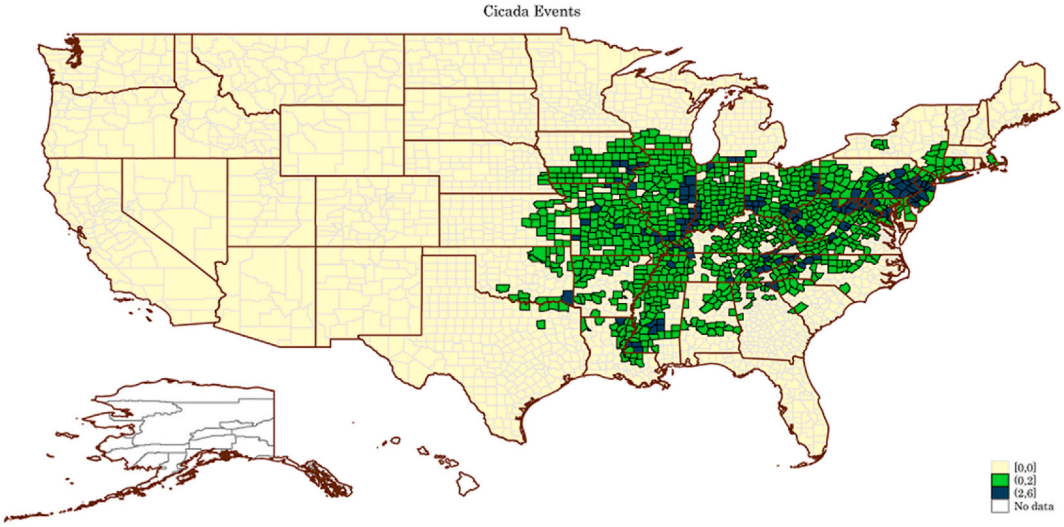

Fig. 1. Geographic distributions of cicada events.

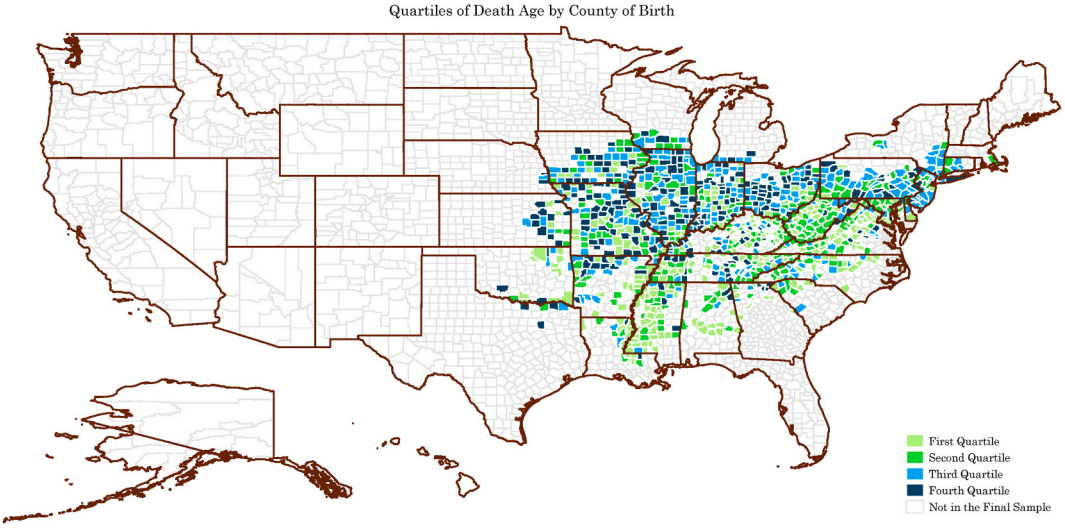

Fig. 2. Distribution of age at death in the final sample.

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 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.

Table 1
Summary statistics.

Variable	Born During Cicada Years		Born in non-Cicada Years	
	Mean	Std. Dev.	Mean	Std. Dev.
Death Age (Months)	772.248	103.546	779.454	103.715
Birth Year	1930.437	2.785	1929.591	2.934
Death Year	1994.772	8.298	1994.528	8.302
Cicada Event × Quartile 4 Apple Production	.393	.488	0	0
Cicada Event	1	0	0	0
Apples Bushels 1967 Per County Area (1000 bushels per km^2)	.068	.156	.051	.185
Quartile 1 Apple	.101	.302	.077	.266
Quartile 2 Apple	.182	.386	.267	.442
Quartile 3 Apple	.323	.468	.353	.478
Quartile 4 Apple	.393	.488	.303	.459
White	.935	.246	.937	.243
Black	.064	.244	.062	.241
Other Races	.001	.033	.001	.032
Father is Farmer	.251	.434	.291	.454
Father's Socioeconomic Index Quartile 1	.285	.451	.275	.446
Father's Socioeconomic Index Quartile 2	.216	.412	.254	.435
Father's Socioeconomic Index Quartile 3	.233	.423	.212	.409
Father's Socioeconomic Index Quartile 4	.266	.442	.26	.439
Father's Socioeconomic Index Missing	.135	.342	.132	.338
Mother's Education < HS	.653	.476	.664	.472
Mother's Education = HS	.238	.426	.229	.42
Mother's Education College-More	.043	.203	.045	.207
Mother's Education Missing	.066	.248	.063	.243
County Population	389124.99	547271.02	533326.83	1017501.3
Average Number of Children < 5 Years Old Nchild5	.366	.097	.389	.12
Share of First-Generation Immigrants	.112	.092	.1	.09
Share of Second-Generation Immigrants	.198	.142	.19	.146
Average Occupational Income Score	24.915	3.581	24.381	3.783
Share of Literate	.818	.196	.851	.173
Share of Married	.608	.027	.612	.028
Observations	12,754		190,618	

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.

Another similar concern is the inherent differences in the final sample's counties and other counties in non-cicada states. This issue may confound the implications as counties may experience different time-varying exposures which could also influence later-life health and longevity. We argue that these differential exposures to other socioeconomic and policy environment is concerning if

they correlate with our cicada exposure measure. Besides, the differential exposures could potentially be reflected in sociodemographic composition of the final sample through channels such as different survival and mortality. These two arguments provide a framework for an empirical evaluation of this concern. Specifically, we regress observables on cicada measures similar to balancing tests of Table 2 but include all counties in the US in our regressions. These results are reported and discussed in Appendix E. We observe no significant differences between pre-determine observables of treated counties/cohorts and other counties/cohorts in the rest of the US.

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 source 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 0.12 percent change from the mean of the outcome. We replicate this result for the subsample of whites and low-educated mothers in columns 2–3. We observe a similar insignificant association which rule out the concern over endogenous data linking. However, we observe small but significant association with low-socioeconomic status father. The negative coefficient suggests reductions of those individuals from low socioeconomic status families. To the extent that socioeconomic status during childhood is relevant for later-life mortality (Hayward and Gorman, 2004; Lee and Ryff, 2019; Montez and Hayward, 2014), this negative coefficient implies underestimation in the results as the positive effects of higher socioeconomic individuals might suppress the negative effects of early-life pesticide exposure.

5.3. Selective fertility

Paternal pre-prenatal pesticide exposure and maternal exposure during pregnancy has been associated with negative consequences for the developing fetus, including preterm birth, low birth weight, congenital anomalies, and fetal death (Dias et al., 2023; Kalliora et al., 2018; Shaw et al., 1999). Moreover, infant mortality is also higher among low birth weight and preterm birth infants (Lau et al., 2013). Since these effects may reveal disparities by sociodemographic factors, pesticide exposure could change the sociodemographic composition of births through their influence on fetal death and selection mortality. If these mortality and fetal death impacts change 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 are correlated with their infants' survival and those characteristics are (in unobservable ways) linked to their infants' later-life longevity. For instance, if exposure to pesticide reveal lower fetal death among white mothers (through mitigating effects of other channels, e.g., better socioeconomic status), then the surviving sample overrepresents whites for whom longevity is higher for many (usually unobservable) reasons. Hence, the fetal death selection adds a bias into the long-term links.

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 (2023) 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

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 Production	−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	203,372	203,372	203,372	203,372	203,372	203,372	203,372	203,372	203,372
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. All regressions are weighted using county population. The outcomes are shown in columns. All regressions include county, birth-year, and event-year by birth-year fixed effects.
***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 Apple Production	−.00006	−.00078	−.00027	−.00199**
	(.00097)	(.00098)	(.00086)	(.00075)
Observations	3,684,502	3,345,775	2,275,414	1,694,963
R-squared	.00479	.00477	.00526	.00545
Mean DV	0.050	0.052	0.054	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. All regressions are weighted using county population. All regressions include county, birth-year, and event-year by birth-year fixed effects.
***p < 0.01, **p < 0.05, *p < 0.1.

early life are associated with later life negative impacts on health outcomes and reductions in longevity (Hayward and Gorman, 2004; Montez and Hayward, 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.

To account for potential family-level and county-level confounders, we add additional covariates to our baseline model. 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.

There are intrinsic and time-invariant features of counties that structurally make county effects have differential influences on outcomes of different subpopulations. For instance, the place-based effects could be different among blacks in less segregated counties with potential long-term legacies (Böhlmark and Willén, 2020). To address this concern, 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

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 Production	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. All regressions are weighted using county population. The outcomes are shown in columns. All regressions include county, birth-year, and event-year by birth-year fixed effects. County controls include share of immigrants, share of literate people, share of married people, and average occupational income score.
***p < 0.01, **p < 0.05, *p < 0.1.

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 patterns in food quality and hence birth outcomes, the effects of which can be detected in old-age longevity (Abeliansky and Strulik, 2020; Buckles and Hungerman, 2013; Currie and Schwandt, 2013; 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. For instance, a cluster of states may initiate economic and social policies that provide health benefits and affect life expectancy at birth. Hence, these exposures may lead to a convergence in life expectancy across different state-clusters. Co-moving of cicada events with this convergence trend might result in biased estimates (Goodman-Bacon, 2021). 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 column 9, we add birth-state by birth-year fixed effects to limit the variation to come from cohorts born in the same state-year across treated versus control counties. Since the final sample has a limited set of counties with reported apple production and experience of cicada during the period of 1925–1940, the inclusion of state-year fixed effects largely absorbs all variations based on which the estimates are driven. Hence, it is not surprising that we observe relatively smaller effects.

In the next four columns, we check for sensitivity of the functional form of the outcome. In column 10, 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 11–13, 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 13 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 the death window of DMF data is limited (1975–2005), it does not provide the full longevity history of individuals, i.e., the data is truncated and censored. This truncation is especially concerning if the cicada-induced pesticide impacts result in deaths at younger ages (i.e., prior to 1975) or appear with delays at older ages (i.e., post-2005). In other words, DMF death selection does not follow a random process and may add bias to our regressions. There are two models that are designed for handling truncated data and implement survival analysis, which we implement in columns 14–15 as additional robustness checks. First, we employ the Accelerated Failure Time (AFT) model which is useful for truncated data and especially for the outcome of under study (Aizer et al., 2016). We assume an exponential functional form which can be interpreted as a logarithmic outcome. The results, reported in column 14, provides quite similar effects compared with the OLS effect on log age-at-death of column 10. Further, we implement Heckman two-step model using the original cohorts of the 1940 linked with the final sample (see section 5.2) (Heckman, 1979). This model estimates the probability of merging between DMF observations (in the final sample) and their respective original population in the full-count 1940 census as a function of observables. It then calculates the Inverse Mills Ratio (IMR) and adds to the second stage regression as an additional covariate. This new control accounts for the influence of selection bias due to non-random merging. The results are reported in column 15. We find the effects that are 22 percent larger than column 1, suggesting that the truncation may results in underestimation of true effects.

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) and the difference-in-difference technique of Sun and Abraham (2021) to re-evaluate the regression-produced estimates. The results are reported in columns 16–17. The estimated marginal effects drop by 16 and 13 percent suggesting small overestimation due to potential

Table 5
Main results for exposure at year of birth.

	Outcome: Age at Death (Months)			
	(1)	(2)	(3)	(4)
Cicada Event × Quartile 4 Apple Production	−1.33764	−2.1646**	−2.12428**	−2.22169***
	(0.70062)	(0.66248)	(0.67217)	(0.54418)
Observations	203,372	203,372	203,372	203,372
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. All regressions are weighted using county population. Family controls include maternal education dummies and paternal socioeconomic status dummies. County controls include share of immigrants, share of literate people, share of married people, and average occupational income score.
***p < 0.01, **p < 0.05, *p < 0.1.

Table 6
Robustness checks.

	Column 4 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 Production	−2.22169***	−2.21768***	−2.20768**	−2.22447***
	(0.54418)	(0.57553)	(0.66266)	(0.54973)
Observations	203,372	361,751	441,909	203,356
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 Production	−2.21436**	−2.16265***	−2.27114***	−2.56644**
	(0.668)	(0.51689)	(0.58322)	(0.78312)
Observations	203,372	203,184	203,372	203,372
R-squared	0.07739	0.07902	0.07907	0.07764
	Adding State-Year of Birth FE	Outcome: Log Age at Death	Outcome: Death Age >55	Outcome: Death Age >60
	(9)	(10)	(11)	(12)
Cicada Event × Quartile 4 Apple Production	−1.68117***	−0.00301**	−0.00826***	−0.01618***
	(.40581)	(0.00082)	(0.00087)	(0.0031)
Observations	203,365	203,372	203,372	203,372
R-squared	.07836	0.07404	0.01818	0.01645
	Outcome: Death Age>65	Outcome: Log Age at Death, Method: Accelerated Failure Time (AFT) Model	Heckman (1979) Estimate	Borusyak et al. (2021) Estimate
	(13)	(14)	(15)	(16)
Cicada Event × Quartile 4 Apple Production	−0.01237**	−.0028,045***	−2.698442	−1.86458***
	(0.00462)	(.0000376)	(1.980004)	(0.4560855)
Observations	203,372	203,372	3,684,502	203,372
R-squared	0.04322	–	–	–
	Sun and Abraham (2021) Estimate	Clustering SE at State -Level	Clustering SE at Birth-Year-Level	Clustering SE at Division-Year
	(17)	(18)	(19)	(20)
Cicada Event × Quartile 4 Apple Production	−1.93529	−2.22169*	−2.22169**	−2.22169***
	(1.182928)	(1.16959)	(1.00588)	(0.84528)
Observations	203,372	203,372	203,372	203,372
R-squared	0.0868	0.07732	0.07732	0.07732

Standard errors, clustered at the division of birth level (except for column 14–16), are in parentheses. All regressions are weighted using county population. All regressions include county, birth-year, and event-year by birth-year fixed effects. Family controls include maternal education dummies and paternal socioeconomic status dummies. County controls include share of immigrants, share of literate people, share of married people, and average occupational income score.
***p < 0.01, **p < 0.05, *p < 0.1.

bias in OLS-produced estimates.

In the main results, we cluster standard errors at the census-division level. In columns 18–20, 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.

Another primary concern is the confounding influence of time-varying unobserved covariates at the county level. One notable example is income. Cicada events may damage crops and have a negative effect on county-level economic measures and the effects pick up on this income effect rather than the pesticide exposure effect. In Appendix C, we posit that the income effects might have a more devastating effects on farmland owners than farm laborers. In this case, one may observe larger effects when focusing on those whose father's occupation is farmland owner than those whose father's occupation is farm laborer. We explore this potential difference empirically and find no evidence to support it. On the contrary, we find substantially larger effects for farmland laborers, which further lends to the influence of pesticides rather than alternative explanations. Another related response to this concern is the proactive behavior of farmers (both laborers and farm owners). Since cicada events are cyclical and easily predictable, the income changes could have been mitigated by other channels (e.g., temporarily change of occupation, saving ahead) and not have the effects of similar income shocks, documented in the literature (Aizer et al., 2016). Moreover, we should also note that the pesticide use, while causing pollution, also increases farm income and longevity. Therefore, our findings represent the net effect of pollution and income changes.

Another argument, in line with the previous income effect, is that the interaction term of Equation (1) compares longevity of high versus low apple producing counties. The income differences in these two types of counties might add to the observed negative effects and bias the estimates. However, the average socioeconomic score of high apple intensity counties is about 1.4 units higher than low apple concentration counties. Therefore, if this socioeconomic score (as a proxy for income) confounds the estimate, it results in underestimating the true effects as the evidence suggests benefits of socioeconomic measures of birth-place and childhood-place on later-life mortality (Almond et al., 2018; Hayward and Gorman, 2004).

A remaining concern is that cicadas might permanently damage apple trees (and in general fruit trees) which results in substitution between tree and row crops with its consequences for income of farmers, hence adding endogeneity to the results. However, there are two facts that makes this scenario less plausible. First, the potential damage of cicadas to apple trees even in the absence of pesticides is not permanent. One concerning damage is through feeding on tree sap and cutting trees' vascular system. Although it makes the apple trees weaker, a healthy tree can recover relatively fast from this damage. Moreover, pesticides largely limit the negative damages of cicadas, leaving little room for permanent damage. Second, fruit trees (and specifically apple trees) are long-term investments with large fixed costs and benefits that mature across several decades. It is unlikely for farmers to switch to row crops in the presence of temporary damage from cicadas.

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 -3 through $+6$.[7] The results are reported in top panel of Fig. 3.[8] The effects of exposure during several years prior to birth is close to zero. This set of coefficients 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. However, the effects reach the maximum (in magnitude) impact for year of birth exposure and become statistically significant. We observe a comparable pattern when we replicate these results using Sun and Abraham (2021) estimate. We observe insignificant coefficients for negative age-at-exposure and for postnatal ages. We find a negative and significant reduction for age-at-exposure of zero, suggesting the relevance of in-utero and early-life impact of pesticide for long-term health and mortality in contrast with other ages at exposure.

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 5–6, for whom we observe negative though insignificant impacts. Therefore, it is not surprising to observe relatively smaller effects than the case in which the control groups exclude other ages at exposure.

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 7, 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

[7] Hence, the control group consists of those born 7-more years before or 4-more years after the cicada emergence.

[8] Since some cohorts may be exposed twice (e.g., at birth and age 13 if exposed to a 13-year cycle cicada brood), we also add a cicada cycle length (a dummy for whether the emerged cicada is 13- or 17-year cycle). However, the coefficients are almost identical to the case where we do not add this variable. Moreover, in Appendix F, we show the robustness to excluding in the subsample of 17-year-cycle cicada counties, suggesting little concern for the influence of receiving treatment in later ages of childhood.

statistically insignificant partly due to much smaller sample size (column 1). The effects on whites are 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 8. The 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. This larger effect may suggest various means of exposure, including local water pollution, airborne pollution from pesticide sprays, or even a lower migration among farmers which results in a higher cumulative exposure.

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; 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 9 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

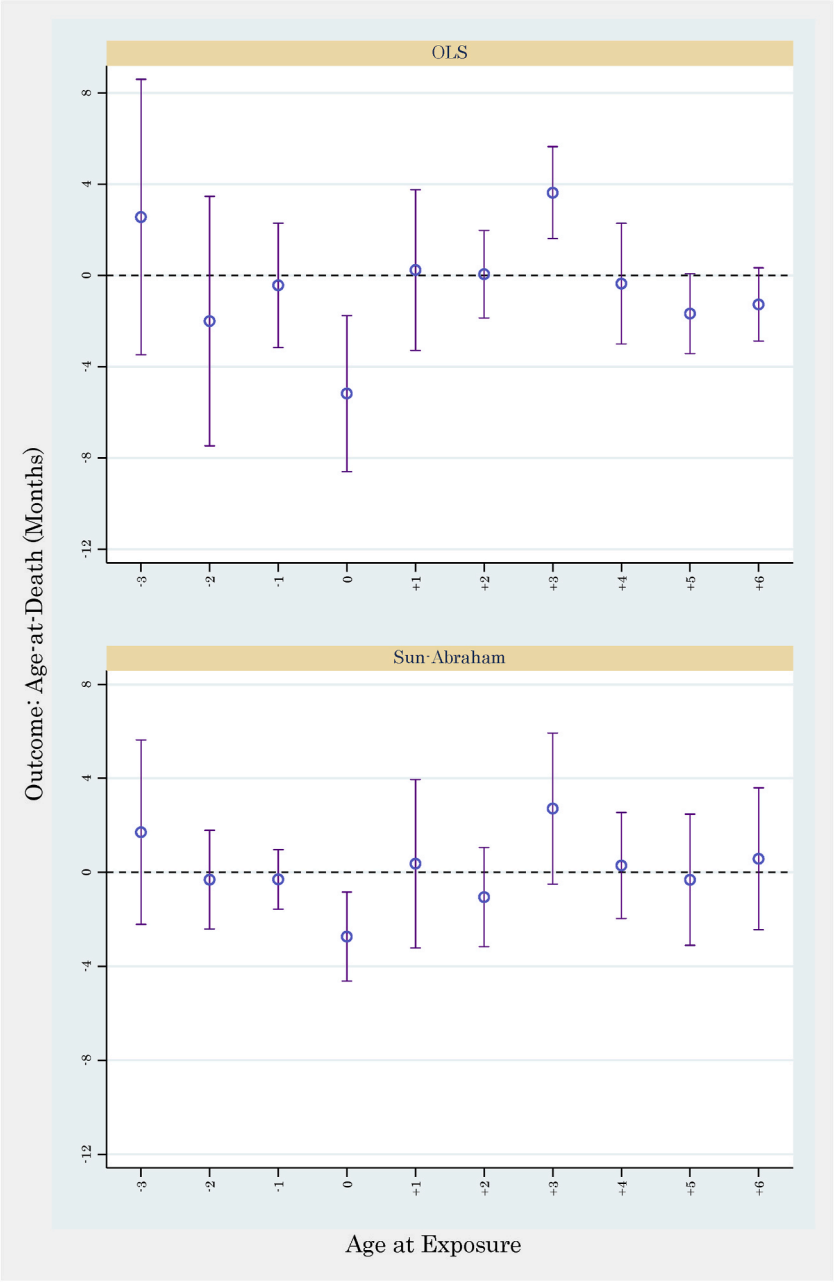

Fig. 3. The effects of cicada exposure across different ages.
Notes. Point estimates and 95 percent confidence intervals are reported. The coefficients are from a regression of age at death on exposure to cicada at various ages interacted by apple production dummy. The regression includes county and birth year fixed effects and events interacted with birth year. The regression is weighted by population and standard errors are clustered at the division level. Family controls include maternal education dummies and paternal socioeconomic status dummies. County controls include share of immigrants, share of literate people, share of married people, and average occupational income score.

Table 7
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 Apple Production	−25.55464	−1.03409**	−4.72658	−1.07049	−2.78665**	1.14394
	(17.65692)	(0.36952)	(2.71057)	(1.41543)	(1.09385)	(2.00036)
Observations	12,776	190,496	107,086	96,276	147,658	55,704
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. All regressions are weighted using county population. All regressions include county, birth-year, and event-year by birth-year fixed effects. Family controls include maternal education dummies and paternal socioeconomic status dummies. County controls include share of immigrants, share of literate people, share of married people, and average occupational income score.
***p < 0.01, **p < 0.05, *p < 0.1.

Table 8
Heterogeneity by father farmer status.

	Outcome: Age at Death (months)		
	(1)	(2)	(3)
Cicada Event × Quartile 4 Apple Production × Father Farmer	−10.28714*	−10.33739*	−10.44854*
	(4.7794)	(4.86611)	(4.95677)
Cicada Event × Quartile 4 Apple Production	−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 Production	−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	203,372	203,372	203,372
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. All regressions are weighted using county population. All regressions include county, birth-year, and event-year by birth-year fixed effects. Family controls include maternal education dummies and paternal socioeconomic status dummies. County controls include share of immigrants, share of literate people, share of married people, and average occupational income score.
***p < 0.01, **p < 0.05, *p < 0.1.

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 9 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 to 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. Discussion

To gauge the potential life-years lost due to pesticide exposure during in-utero, we estimate a back-of-the-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 of apple production added up to a population of roughly 300K individuals. Assigning the

Table 9
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 Production	−0.09785*	−.01477*	−56.67625	−1.0806*
	(0.04617)	(0.00721)	(55.89558)	(0.50105)
Observations	318,057	318,057	318,057	304,708
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. All regressions are weighted using county population. All regressions include county-PUMA, birth-year, and event-year by birth-year fixed effects. Individual controls include dummies for race and ethnicity.
***p < 0.01, **p < 0.05, *p < 0.1.

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 monetize this value by incorporating Value of Statistical Life (VSL) estimates. While VSL estimates vary widely by agency, study, and sample, we assume an average VSL of $10 million (in 2020 dollars) (Kniesner and Viscusi, 2019; Viscusi, 2018). Given the difference of about 13 years in average age-at-death in the final sample and the average life expectancy in the US, and assuming a discount rate of 3 percent, we reach a Value of Statistical Life Year (VSLY) of about $940K (Colmer, 2020). Using our back-of-the-envelope calculation on life-years lost, we reach a total loss of about $51.7 billion. We can also extrapolate this number for all croplands and pesticides while still working with the same cohorts. Using the share of pesticides used 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. We can also use this number to extrapolate the VSLY for our back-of-an-envelope estimate of total life-years lost due to pesticide exposure for the whole US. We reach a total loss of about $1.1 trillion.[9]

Moreover, we can understand the effects of this paper using the equivalent income required to offset the negative effects of pesticide use. Chetty et al. (2016) examine the income-longevity relationship using tax records linked to social security administration death records over the years 1999–2014. They find that an additional income percentile is associated with about 1.9 months increases in longevity. For a household at the sample's median, an additional income percentile is equivalent to a rise of about $8K (in 2020 dollars). Therefore, to offset the early-life pesticide exposure effect, individuals' income would need to increase by about $9260 annually. Adding up all potentially affected cohorts in the treated counties, this is equivalent to an annual transfer of about $2.8 billion. We can also use the ratio of pesticide use in apple tree croplands to the total pesticide use in the US to extrapolate this number for non-cicada years and non-apple-producing counties. We reach an estimate required transfer of about $60.7 billion to offset the negative impacts of pesticide exposure during in-utero and early-life.[10]

8. Conclusions

While pesticides are necessary tools to increase the 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 male individuals 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-months 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.

The long-term negative impacts documented in this paper seem to be primarily driven by in-utero and early-life exposures, with

[9] Assuming that pesticides in apple tree croplands account for about 2.18 percent of all pesticide use in the US (Fernandez-Cornejo et al., 2014), total treated individuals of about 300K, a marginal effect of 2.2, a VSLY of about $940K, we can calculate total loss as $\frac{(300{,}000*2.2)/12}{0.0458} \times 940K$.

[10] We use the same scaling as footnote 9, assuming 300K treated cohorts, and a monetary equivalent of $9260 for 2.2 months rise in longevity. We calculate the number as follows: $\frac{300{,}000*9{,}260}{0.0458}$.

only small changes observed for other ages at exposure. As a result, one policy implication is to acknowledge the population at risk and implement initiatives to protect this subpopulation, rather than focusing solely on eliminating or reducing pesticide use. Furthermore, the long-term effects operate through various mediatory channels and dynamically interact with many later-life exposures and conditions (Cunha et al., 2010; Cunha and Heckman, 2007). Therefore, another policy path to reduce these long-term negative impacts is to recognize the affected population and understand factors that alleviate the effects throughout life course.

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 9) 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 9 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.

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 Apple Production	−1.29841	−2.11442**	−2.07517**	−2.08306**
	(0.7163)	(0.67398)	(0.68379)	(0.60813)
Observations	203,182	203,182	203,182	203,182
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. All regressions are weighted using county population. All regressions include county, birth-year, and event-year by birth-year fixed effects. Family controls include maternal education dummies and paternal socioeconomic status dummies. County controls include share of immigrants, share of literate people, share of married people, and average occupational income score.
***p < 0.01, **p < 0.05, *p < 0.1.

Appendix B

In the paper, we focused on the top-quartile of apple production concentration as the dosage of the cicada exposure. In this appendix, we examine the robustness of the results to alternative thresholds. In column 1 of Appendix Table D-1, we replace the apple production dummy with a dummy that equals one if the apple production per county area is above the median of counties in the final sample and zero otherwise. In columns 2–4, this measure is replaced with dummies indicating apple production being above 6th, 7th, and 8th decile of the respective variable's distribution. Across these columns, we observe a quite comparable coefficient to that of the main results of Table 5. Finally, in column 5, we replace this variable with a continuous measure of apple production per county land area (in bushels/km^2). The difference in this continuous variable in top-quartile and bottom-three quartile counties is about 0.15 bushels/km^2. Therefore, this difference results in a 1.9 months reduction in longevity (0.15 × 12.4), comparable to the main results although smaller and insignificant.

Table B-1
Replicating The Results for Alternative Measures of Apple Production Counties

	Outcome: Age at Death (Months)				
	(1)	(2)	(3)	(4)	(5)
Cicada Event × (Above-Median) Apple	−2.51794**				
	(.84382)				
Cicada Event × (Above-6th Decile) Apple		−2.35225***			
		(.54907)			

(*continued on next page*)

Table B-1 (*continued*)

	Outcome: Age at Death (Months)				
	(1)	(2)	(3)	(4)	(5)
Cicada Event × (Above-7th Decile) Apple			−2.25233***		
			(.32918)		
Cicada Event × (Above-8th Decile) Apple				−2.13961**	
				(.62504)	
Cicada Event × (Continuous) Apple Bushels per Area (1000 bushels/km^2)					−12.38082
					(8.22366)
Observations	203,372	203,372	203,372	203,372	203,297
R-squared	.07733	.07732	.07732	.07732	.07733
Mean DV	779.353	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. All regressions are weighted using county population. Family controls include maternal education dummies and paternal socioeconomic status dummies. County controls include share of immigrants, share of literate people, share of married people, and average occupational income score.
***p < 0.01, **p < 0.05, *p < 0.1.

Appendix C

One concern in interpreting the main results of the paper is the influence of time-varying county-specific confounders. One important confounding influence comes from potential changes in income as a result of damages cicada impose on counties and specifically farmlands, which results in relatively larger negative effects of Table 8. If this concern is valid, one expects to observe relatively larger effects for farmland owners who are more affected by the loss of crops compared with other laborers in farmlands. Another note is that cicada events and the damage to tree croplands may induce a disincentive among farmers (both laborers and owners) to continue working in agriculture. To avoid this, and to examine the effects by farmer-type status, we employ cross census linking rules provided by Abramitzky et al. (2020) to find fathers in the full-count 1930 census and extract their occupation in 1930. We then replicate an interaction model similar to the one in Table 8 in which the father's farmer status is defined based on whether they are farm owner or farm laborer. We report the results in Appendix Table C-1. We find that the interaction term is substantially larger for laborers than owners, a fact that does not support the income-effect concern.

In our response to the above-mentioned concern, we should acknowledge two limitations. First, if farm owners decrease their hiring because of production losses, it can result in income reduction for the hired workers. However, farmers may respond proactively to these types of income shocks (owners or laborers) as cicada events were easily predictable. Therefore, the income changes could have been mitigated by other channels (e.g., temporarily change of occupation, saving ahead) and not have the effects of similar unexpected income shocks. Second, we should note that the pesticide serves as an essential factor contributing to farm output and the overall increase in farm income. While pesticide pollution can reduce longevity, the application of pesticides raises the growth of farm income, thus increasing longevity. Therefore, the findings represent the (negative) effect of pollution net of (positive effect of) income.

Table C-1
Replicating the Effects based on Father Farmer Type in 1930

	Outcome: Age at Death (Months)	
	Father's Farmer Occupation: Laborer	Father's Farmer Occupation: Owner
	(1)	(2)
Cicada Event × Quartile 4 Apple × Father Farmer	−8.47421	−.39896
	(28.36277)	(7.03156)
Cicada Event × Quartile 4 Apple Production	−.50821	−.16186
	(.82916)	(.92342)
Father Farmer × Cicada Event	21.44855	−4.67729
	(28.30015)	(5.52966)
Father Farmer × Quartile 4 Apple Production	−6.53345	3.20404
	(5.01529)	(3.11684)
Father Farmer	7.57589	3.35209*
	(5.95805)	(1.52694)
Observations	84,373	84,373
R-squared	.08098	.08094
Mean DV	780.710	780.710
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. The columns show the definition of father farmer which is interacted by the right-hand side variables (and do not represent subsamples). All regressions are weighted using county population. Family controls include maternal education dummies and paternal socioeconomic status dummies. County controls include share of immigrants, share of literate people, share of married people, and average occupational income score.
***p < 0.01, **p < 0.05, *p < 0.1.

Appendix D

While the identification strategy of the paper exploits the cross-sectional differences in apple-production concentration, we examine the effects to a difference-in-difference method in which the primary treatment variable is cicada emergence exposure measure. We report the results in Appendix Table D-1. The main effect on the full sample is −0.2 (column 1). However, when we focus on the subsample of counties that are in the top-quartile of apple production, we observe an effect that is about 10 times larger (column 2). Moreover, this effect is quite similar to the effect reported in Table 5, although statistically insignificant. In column 3, we find small and positive coefficient when we focus on the subsample of low apple production counties.

Table D-1. The Difference-in-Difference Results without Employing the Dosage of the Treatment by Apple Orchard Concentration

	Outcome: Age at Death (Months)		
	Full Sample	Top Quartile Apple Counties	Bottom-Three Quartile Apple Counties
	(1)	(2)	(3)
Cicada Event	−.20783	−2.01353	.31428
	(1.13711)	(1.64866)	(1.21555)
Observations	203,372	62,678	140,693
R-squared	.07724	.08343	.07627
Mean DV	779.353	780.660	779.127
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. All regressions are weighted using county population. Family controls include maternal education dummies and paternal socioeconomic status dummies. County controls include share of immigrants, share of literate people, share of married people, and average occupational income score.
***p < 0.01, **p < 0.05, *p < 0.1.

Appendix E

Table 2 reports a series of balancing tests to explore the correlation between receiving the treatment and several other pre-determined observables as potential confounders. The results of that table rule out the sociodemographic compositional change to bias the estimates. Another concern in line with the sociodemographic compositional change is the clustering of cicada counties in the final sample. These counties are primarily located in Northeast and Midwest and have characteristics and experience exposures that are systematically different than other counties in the country. However, we provide three pieces of evidence to argue that this clustering does not impact the generalizability of the results. First, in columns 2–3 of Table 6, we show that the effects are virtually identical if we include observations from other counties of cicada states and also observation from all states. Therefore, cicada events are not accompanied with other shocks and exposures in other states. Second, many of the geographic differences that influence later-life life expectancy are captured by county fixed effects. The fact that we are working with a relatively narrow set of birth cohorts (1926–1940) further mitigates time-variant differences. Third, the primary concern is that there are time-varying differences in characteristics and exposures that correlates with cicada events and appear differently in cicada counties/states versus non-cicada counties/states. If this is the case, then we would observe differential trends in characteristics of the final sample from those who were treated (cicada event) and all other observations from all other counties. We can empirically test this using a version of balancing test of Table 2 that includes all the US counties. The results of these balancing tests are reported in Appendix Table E-1. We do not observe a robust, consistent, and significant association between several observables and cicada events. Since we do not find associations with observables, we conclude that there are no correlations with unobservables as well (Altonji et al., 2005; Fletcher et al., 2021). We conclude that there are no differences in the treated population (in a clustered region) and other non-treated population in ways that bias the estimates.

Table E-1
Exposure to Cicada Events and Observable Characteristics Using the Full Sample of All Counties in the US

	Outcomes:									
	White	Black	Other	Father's SEI below Median	Father's SEI above Median	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 Production	−.00135	.00132	.00003	−.00074	.00074	−.00298	.00235	.00701	.00115	−.01052**
	(.00265)	(.00304)	(.00045)	(.00489)	(.00489)	(.00392)	(.00301)	(.0047)	(.00266)	(.00335)
Observations	441,909	441,909	441,909	441,909	441,909	441,909	441,909	441,909	441,909	441,909
R-squared	.08424	.09041	.03819	.05602	.05602	.00971	.04055	.03427	.01533	.01352
Mean DV	0.963	0.034	0.003	0.329	0.671	0.133	0.662	0.238	0.042	0.058
County and Birth-Year FE	✓	✓	✓	✓	✓	✓	✓	✓	✓	
Event-by-Birth-Year FE	✓	✓	✓	✓	✓	✓	✓	✓	✓	

Standard errors, clustered at the census division of birth level, are in parentheses. All regressions are weighted using county population. The outcomes are shown in columns. All regressions include county, birth-year, and event-year by birth-year fixed effects.
***p < 0.01, **p < 0.05, *p < 0.1.

Appendix F

One concern in the results of Fig. 3 is the confounding effect of exposure to future cicada events. For instance, those in counties that experienced broods of cicadas with 13-year cycle and were exposed during in-utero will again be exposed at age of 13. Since different broods might appear in a given county, county fixed effects do not absorb their influence. One solution is to include type of cicada brood (13-year cycle versus 17-year cycle) in our regressions. We did this in the analysis of Fig. 3 And observed almost identical coefficients with or without this additional control. Another solution is to examine the effects across 13-year and 17-year cycle cicada counties and compare the effects. However, very few counties experience 13-year cycles. In our final sample, roughly 1800 observations are born in 13-year-cycle cicada counties. The inclusion of county and cohort fixed effects reduce the degree of freedom in a way that the effects of exposure cannot be estimated. Instead, we focus on counties with 17-year cycle and replicate the regressions of Fig. 3 and report them in Appendix Figure F-1. We observe very similar pattern and coefficients. Further, we replicate the main results for this restricted sample and report the estimates in Appendix Table F-1. Similarly, we find similar coefficients to those reported in Table 5.

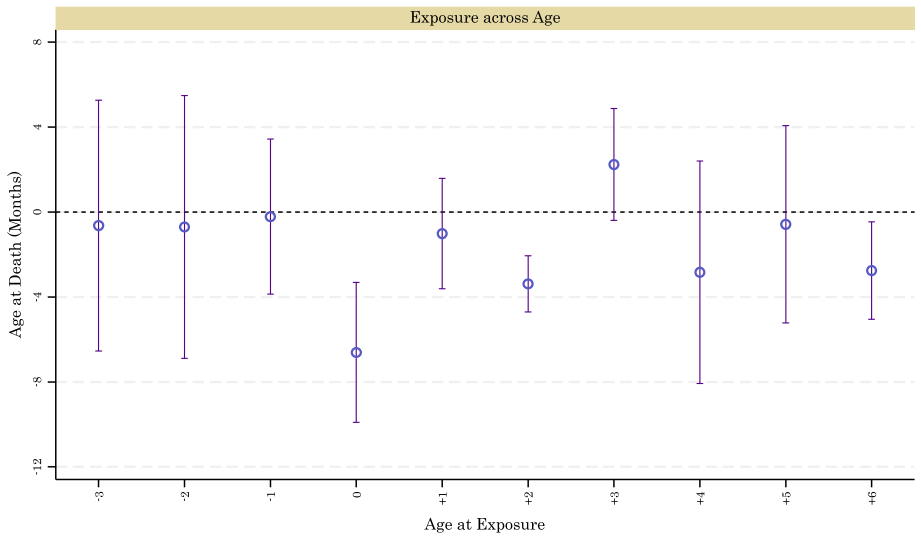

Fig. F-1. The Effects of Cicada Exposure across Different Ages Excluding Counties with Broods with Emergence Cycle of 13
Notes. Point estimates and 95 percent confidence intervals are reported. The coefficients are from a regression of age at death on exposure to cicada at various ages interacted by apple production dummy. The regression includes county and birth year fixed effects and events interacted with birth year. The regression is weighted by population and standard errors are clustered at the division level. Family controls include maternal education

dummies and paternal socioeconomic status dummies. County controls include share of immigrants, share of literate people, share of married people, and average occupational income score.

Table F-1

Replicating Main Results for Exposure at Year of Birth Excluding Counties with Broods with Emergence Cycle of 13

	Outcome: Age at Death (Months)			
	(1)	(2)	(3)	(4)
Cicada Event × Quartile 4 Apple Production	−1.74805***	−2.34223***	−2.30212***	−2.42902***
	(.40657)	(.54748)	(.55584)	(.43659)
Observations	201,491	201,491	201,491	201,491
R-squared	.07694	.07698	.07718	.07719
Mean DV	779.414	779.414	779.414	779.414
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. All regressions are weighted using county population. Family controls include maternal education dummies and paternal socioeconomic status dummies. County controls include share of immigrants, share of literate people, share of married people, and average occupational income score.

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

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