The Long Shadow of the Past: Early-Life Disease Environment and Later-Life Mortality*

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Abstract

A recently growing literature evaluates the influence of early-life conditions on life-cycle health and mortality. This paper extends this literature by estimating the associations between birth-state infant mortality rates experienced during early life (as a proxy for general disease environment, health-care access, and nutrition) and later-life old-age mortality rates. Using the universe of death records in the US over the years 1979-2020 and implementing two-way fixed effect models, we find positive and significant associations. Back-of-the-envelope calculations suggest that the sharp reductions in infant mortality rates across cohorts of the final sample (1915-1940) are associated with a roughly 26% drop in mortality rates during old age, conditional on survival up to age 55. Further, we find that these associations are larger for older ages and are larger in magnitudes at higher levels of infant mortality rates. Further, we provide empirical evidence to argue that deteriorations in education-income profile and increases in disability are likely mechanism channels.

Keywords: Mortality, Infant Mortality, Early-Life Exposures

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1. Introduction

There is a recently growing literature that examines the role of early-life environment in explaining the disparities in later-life outcomes. Empirical studies in various settings provide suggestive evidence that policy and environmental exposures experienced by infants and children may have a lingering influence across their life course (Almond et al., 2018; Almond & Currie, 2011a, 2011b; Barker, 1994, 1995; Currie & Rossin-Slater, 2013). For instance, studies point to the relevance of disease environment experienced during in-utero, early-life, and childhood for children's health and their later-life outcomes. Case & Paxson (2009) document that those born in census regions with higher infant mortality rates are more likely to reveal impaired cognitive functioning during old age. Bozzoli et al. (2009) employ data from several developed countries and show that individuals who were born in countries with higher infant mortality reveal lower height across the life course. Although the extent of the outcomes explored in these studies is limited, they point to declining health outcomes for individuals with higher exposure to adversities in early-life with potential spillover and effects on other outcomes.

Mortality outcomes offer an extreme but precise measure of health. Empirical studies link mortality outcomes to early-life conditions and childhood environment (Aizer et al., 2016; J. M. Fletcher, 2018a, 2018b; J. Fletcher & Noghanibehambari, 2024; Hayward & Gorman, 2004; Lindeboom et al., 2010; Van Den Berg et al., 2006). For instance, Fletcher (2018a) provides evidence that cohorts who were in utero during the infamous 1918 influenza reveal higher mortality rates later in life. Similarly, Mazumder et al. (2010) show that cohorts exposed to the 1918 influenza during the prenatal development period have higher cardiovascular diseases during adulthood and old age.

The current study extends this literature by evaluating the correlations between area level infant mortality rates at the level of the birth-state-year, as a proxy for the general disease environment, undernutrition, and lack of healthcare access, and later-life old-age age-specific mortality rates. We find that those born in states with higher infant mortality rates reveal higher mortality rates in ages past 54. Further, we evaluate the heterogeneity of these correlations across cohorts and find that the scarring influence of early-life disease environments is more pronounced for earlier cohorts who were exposed to higher infant mortality regimes. We also find that these

correlations are significant for several causes of death in old age, including infectious diseases, malignant neoplasm diseases, and cardiovascular diseases.

We make two important contributions to the literature. First, to our knowledge, this is the first study to examine the links between the infant mortality rate in early-life and later-life mortality in the US. While previous studies focus on the link between early-life postnatal mortality and other outcomes (Almond et al., 2012; Bozzoli et al., 2009; Saavedra, 2017) or other disease exposures and later-life mortality links (Beach, Clay, et al., 2022; Fletcher, 2018a, 2018b; Mazumder et al., 2010), fewer studies have directly explored the associations between infant mortality rate in early-life and later-life mortality. One exception is the study of Cormack et al. (2024) that explores the effects of disease exposure in the first year of life on later life mortality using the sample of cohorts born between 1905-1929 in Sweden. They proxy disease exposure with post-neonatal mortality rates and document heterogenous impacts by gender. They document scarring impacts on life expectancy of females but no effects on survival of males. Second, we also add to the small but growing literature that evaluates the relevance of early life conditions and adversities on later-life mortality and longevity (Aizer et al., 2016; Cutler et al., 2006; Noghanibehambari & Engelman, 2022; Noghanibehambari & Fletcher, 2023, 2024; Schmitz & Duque, 2022; Vu et al., 2023).

The rest of the paper is organized as follows. Section 2 introduces data sources. Section 3 discusses the econometric method. Section 4 reviews the results. Finally, we conclude the paper in section 5.

2. Data Sources

The primary source of data is vital statistics death certificates extracted from NCHS (2020). The NCHS data covers the universe of deaths in the US. Starting from 1979, the NCHS reports the state-of-birth of decedents, a required identifier in our setting. The data also contains the age, race, ethnicity, and state-year of death. We use age-at-death and year-of-death to infer birth cohort. We use the restricted version of this data which covers death records up to 2020. The initial sample contains roughly 88 million death records.

Infant mortality data is extracted from Bailey et al. (2016). The data contains infant death data at the county level from 1915 to 2007. We aggregate the data at the state-level since the NCHS sample reports birth-states. To calculate mortality rates, we collapse the NCHS data by birth-year, birth-state, current-year, current-state, and age. We then calculate mortality rates as follows:

$$MR_{bcsta} = \frac{N_{bcsta}}{POP_{sta}} \times 100{,}000$$

Where MR is the mortality rate of cohort c born in state b died at age a in state s in year t. The denominator is the aggregate population by state, year, and age taken from SEER (2019).[4] We then merge this data with the infant mortality data of Bailey et al. (2016) based on birth-state and birth-year. We implement four main sample selections. First, since our main focus is exploring the latent effects of early-life environment on old-age mortality, we focus on mortality rates of ages 55 and beyond. We also restrict the cohorts to those born between 1915-1940 as these decades experienced high mortality rates as well as sharp reductions in infant mortality rates. Indeed, the infant mortality rate dropped by roughly 60% from the baseline of about 97 infant deaths per 1,000. Third, not all states participated in the death registration area and reported infant mortality rates across all years. For instance, only 10 and 24 states reported the data in the years 1915 and 1920, respectively. These numbers increased to 47 and 49 states for the years 1930 and 1940, respectively. This unbalanced panel raises the concern that the results could partly pick up on the changes in mortality due to differential longevity of later cohorts in states that joined the death registration area in later years. To address this concern, we restrict the sample to states with available infant death data from 1920 onward, i.e., focusing on 24 states. However, in Appendix B, we report the results without this selection and discuss its implications in interpreting the findings. Table 1 reports the list of birth-states in the final sample for the years 1915 and 1920. Fourth, since SEER (2019) reports state-year-level population data of single ages up to age 89 and aggregate for all post-89 ages (the denominator values of mortality rates), we exclude deaths after age 89. After these selections, the final sample covers roughly 24.2 million deaths that occurred between 1979-2020 to birth cohorts of 1915-1940 in the age range of 55-89.

We also use decennial census data from 1910-1940 to calculate a series of birth-state covariates. We linearly interpolate these covariates for inter-decennial years. Moreover, in the section that explores mechanism channels, we use decennial census data for the years 1980-2000

[4] In the formula for calculating mortality rate, the denominator and numerator have different levels of aggregation. The reason is that the SEER (2019) data does not contain information of population based on birth state and birth year and only reports population estimates based on current year and current state. In Appendix C, we examine the robustness of the results using the population estimates from decennial censuses combined with the American Community Survey data to replace the denominator. The disadvantage is that the data does not cover inter-decennial years and the population estimates are based on random samples rather than the full population as in SEER (2019). The results, reported in Appendix Table C-1, almost identical to the main results of the paper.

combined with the American Community Survey data for the years 2001-2020, years that overlap with the NCHS data. These data are extracted from Ruggles et al. (2020).

Summary statistics of the final sample are reported in the left and right panels of Table 2 for birth cohorts of 1915-1927 and 1928-1940, respectively. The average age-specific mortality rate of the 1915-1927 (1928-1940) cohorts is 142.5 (98.3) per 1,000. Similarly, the average infant mortality rate is 78.4 and 55.9 per 1,000 for the 1915-1927 and 1928-1940 cohorts, respectively.

Figure 1 depicts the geographic distribution of states in the final sample based on the quartiles of infant mortality rate over the years 1915-1940 (top panel) and quartiles of age-specific death rates over the years 1979-2020 (bottom panel). The time-series evolution of mortality rates and infant mortality rates for nine Census divisions are depicted in the top and bottom panels of Figure 2, respectively.

3. Econometric Method

Our identification strategy compares age-specific death rates of cohorts born in states with higher infant mortality rates to those born in states with lower infant mortality rates, net of fixed effects and controls. Specifically, we implement panel data fixed effect models as follows:

$$MR_{brcst} = \alpha_0 + \alpha_1 IMR_{bc} + \alpha_2 X_{bcst} + \alpha_3 Z_{bc} + \eta_{cr} + \gamma_b + \xi_{st} + \varepsilon_{brcst} \qquad (1)$$

Where b indexes birth-state, r census region of birth, c birth-cohort, s death-state, and t death-year. Variable IMR represents infant mortality rate for cohort c born in state b. In X, we include share of females and nonwhites from the NCHS data. In Z, we include birth-state covariates, including share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate individuals, per capita midwives, and per capita doctors. Parameter γ represents birth-state fixed effects. Parameter η represents birth cohort fixed effects interacted with census region of birth fixed effects. Cohort fixed effects account for secular trends of mortality outcomes across cohorts. The interaction of cohort and region fixed effects accounts for differential convergence in the mortality of cohorts across different census regions (shown in Figure 2). To absorb all contemporaneous unobserved state-level confounders that vary over time, we include the interaction set of death-state and death-year fixed effects, represented by ξ. Finally, ε is a disturbance term.

We cluster standard errors at the birth-state level to account for serial correlations in the error terms and also at the birth-cohort level to account for spatial correlations. We weight regressions by the mean state-level age-level population in the 1980 census. To ease the interpretation of coefficients, we standardize both mortality rate and infant mortality rate using the full sample's mean and standard deviation. While in the main results, we focus on a linear relationship and employ continuous variables for both the dependent and independent variables, we show the robustness of the results to alternative functional forms in Appendix A.

4. Results

4.1. Main Results

Before presenting the main results, we examine the visual correlational links between infant mortality rate and later-life mortality rate using Locally Weighted Scatterplot Smoothing (LOWESS) curves. We show the LOWESS curve for different age groups in Figure 3. Three points emerge from this graph. First, across all age groups, exposure to higher infant mortality in early life is associated with higher later-life mortality. Second, this relationship appears to be fairly linear across different age groups, lending to our choice of functional form in equation 1. Third, these age-group-specific curves diverge from each other at higher rates of infant mortality, i.e., we observe that the slopes of curves are higher for older age groups. This implies that the latent impacts of early-life disease environment might appear larger at older ages, a fact that is later confirmed with our formal age-specific analyses.

The main results of the paper are reported in Table 3. We report the parsimonious results of a regression that only includes birth-state and birth-year-by-birth-region fixed effects in column 1. We then add death-state-by-death-year fixed effects into the regression in column 2. In column 3, we add birth-state level controls. The fully parameterized regression suggests that a one-standard-deviation rise in infant mortality at state-year-of-birth is associated with a 0.03 standard deviation higher age-specific mortality rate later in life (past age 54). To put this number into perspective, we use the reduction in infant mortality rate between the years 1920-2000. During this period, infant mortality dropped from 84.1 to 5.3 per 1,000, a decrease of about 4.4 times its standard deviation in the final sample. Therefore, the reduction in later-life age-specific mortality rate associated with this reduction is about 14.6 percent of a standard deviation change, roughly 75.6 lower age-specific deaths per 1,000.

These results are in line with several studies that suggest the disease burden in early-life is associated with adverse life-cycle health outcomes (Bozzoli et al., 2009; Case & Paxson, 2009). These findings are possibly in contrast with the results of Haywarda et al. (2016) who use Finnish data and show that child mortality rate during the early years of life has no association with mortality rates between ages 15-50, however, we focus on deaths occurring past age 54.

As suggested by the LOWESS curves of Figure 3, the documented relationships could reveal heterogeneity based on the age at which we observe mortality outcomes. To investigate such heterogeneities, we replicate the main results for each single age-at-death group and report the estimated coefficients in Figure 4. The coefficients are relatively small and insignificant up to age 59. They rise in magnitude from age 60 and become statistically significant. There is an almost constant rise in the magnitude of coefficients from age 60 to age 72. They stabilize in magnitude from around age 73 up to age 80. The set of coefficients for the age range 81-89 reveals the largest impacts, although in several cases they are statistically insignificant.

Next, we examine the correlations across different age-cohort groups. We divide the sample into two cohorts: 1915-1927 and 1928-1940 cohorts (high and low infant mortality rate regimes in the sample). The earliest of these cohorts (born in 1915) is aged 64 at the time of starting the death window (i.e., the year 1979). The latest of these cohorts (born in 1940) are aged 80 at the time that the death window closes (i.e., the year 2020). Therefore, the age range 64-80 provides a comparable set of age-cohort sets to assess the heterogeneity by birth year. We split this age range into two similar interval groups: 64-72 and 73-80. Overall, we replicate the main findings using the following four categories: cohorts of 1915-1927 aged 64-72, cohorts of 1928-1940 aged 64-72, cohorts of 1915-1927 aged 73-80, and cohorts of 1928-1940 aged 73-80. These results are reported in Table 4. For the age range 64-72, both cohort groups of 1915-1927 and 1928-1940 provide quite comparable coefficients (columns 1 and 2). For the age range 73-80, on the other hand, the high infant mortality regime (i.e., born 1915-1927) provides a relatively larger coefficient compared to the low infant mortality regime (i.e., born 1928-1940).

4.2. Heterogeneity by Race and Gender

Early-life environment, nutrition, healthcare access, and especially exposure to the disease burden may have heterogeneous impacts on later-life outcomes across different genders and races (Almond et al., 2012; Case & Paxson, 2010). To explore such sources of heterogeneity, we

replicate the main results in different subsamples based on race and gender. The results are reported in Table 5. Contrary to previous empirical research that suggests higher impacts of exposure to disease environments for minorities and the disadvantaged population (Almond, 2006; Beach, Brown, et al., 2022), we find negative coefficients among nonwhites (columns 2). One explanation is that nonwhite subpopulations experience a considerably larger infant mortality rate which pushes selection effects to dominate the scarring effects, hence the observed reduction in their later life mortality. Further, this argument is in line with the findings of Almond et al. (2012). They examine the association between birth-state infant mortality rate and later-life maternal birth outcomes. They find that, among white mothers, higher infant mortality rates result in a higher probability of having a low-birth-weight child. In contrast, among black mothers, exposure to higher infant mortality rates is associated with a lower probability of having a low-birth-weight child. They argue that for black children, exposure to a significantly high infant mortality regime results in selection pressures outweighing the scarring impacts.

We also observe a larger impact among males and a relatively smaller coefficient for females (columns 3-4). The respective coefficient of female mortality is about 34 percent smaller in size compared with that of males, although both coefficients are statistically and economically significant. We also report the coefficients of these subsamples (female-male and white-nonwhite samples) for each age-at-death in Figure 5 and Figure 6.

4.3. Heterogeneity by Cause-of-Death

Disease exposure during early-life and specifically during prenatal development could result in later-life mortality risks through several channels, including changes in fetal programming. For instance, disease exposure may lead to malnutrition and alterations in the metabolism of glucose and insulin in the body with a permanent and long-lasting stamp. This adaptation could be advantageous in an environment with limited nutrition during later stages of life but can elevate the risks of cardiovascular disease and diabetes once the nutritional burden is removed (Barker, 1997; Myrskylä et al., 2013). Furthermore, early-life disease exposure may lead to infections that suppress the immune system, making it more susceptible to later-life infections. Alternatively, it may stimulate the immune system in a way that affects chronic conditions. Ochoa et al. (2004). To examine these channels, we examine the correlations across causes of death. The results are reported in Table 6.

We find mixed evidence across different causes of death. For instance, we find significant associations for mortality due to infectious diseases (column 1), malignant neoplasm diseases (column 2), cardiovascular diseases (column 4), and influenza, pneumonia, peptic ulcer, nephritis (column 5). On the other hand, we do not find significant coefficients for death due to diabetes and the category of other diseases (columns 3 and 6, respectively).

Early-life conditions, particularly the disease environment, have been shown to influence susceptibility to certain diseases in later life, including infectious and cardiovascular diseases. (Boekelheide et al., 2012; Moore et al., 2006; Van Den Berg et al., 2011). This literature supports our findings in columns 1-5.

Further, we also observe significant associations with mortality due to external causes. One explanation is that exposure to a higher disease burden during infancy changes the life-course trajectory in a way that increases the prevalence of disability (documented in section 4.4) which is shown to influence accident-injury-related mortality (Landes et al., 2021; Patja et al., 2001). Moreover, given the growing evidence on the role of early-life conditions on later-life mental health outcomes (Zheng et al., 2021), it is not surprising to observe a significant association between early-life disease environment and suicide mortality later in life (column 8). Finally, we document the associations across different causes of death for each single age-at-death in **Error! Reference source not found.** and Figure 8.

4.4. Mechanism Channel

Several studies suggest that improvements in education, income, and socioeconomic status lead to increases in longevity and are associated with mortality gains (Chetty et al., 2016; Fletcher, 2015; Lleras-Muney, 2005; Meghir et al., 2018). On the other end, several studies document a link between early-life factors and later-life education-income profile (Almond, 2006; Fletcher, 2018c; Parman, 2015). Therefore, one might expect that exposure to disease environments during early-life affects later-life mortality through changes in education, income, and measures of socioeconomic status. Since the NCHS data does not report these outcomes, we use alternative data sources to explore these potential mechanism channels. In so doing, we use decennial censuses between the years 1980-2000 combined with the American Community Survey for the years 2001-2020. The coverage of these selected years matches with the death window coverage of the NCHS data.

We implement similar sample selection and regressions as equation 1. The results are reported in Table 7. We observe significant reductions in socioeconomic score (column 1), increases in the probability of low education (columns 2-3), reductions in total personal income (column 4), and increases in disability outcomes (columns 5-8). For instance, a one-standard-deviation rise in birth-state infant mortality rate is associated with a 1.8 percent drop in personal income. It also increases the probability of having less than high school and less than 12 years of education by 1.1 and 1.8 percentage points, equivalent to roughly 14.5 and 16.8 percent change from the mean of the outcomes, respectively.

Consistent with the literature linking old-age disability outcomes to early-life experiences and the research documenting disability-related mortality outcomes (Freedman et al., 2008; Laditka & Laditka, 2019; Pongiglione et al., 2016), we observe significant increases in various measures of disability due to exposure to a harsher disease environment in early life. For instance, a one-standard-deviation increase in birth-state infant mortality rate is associated with a 21, 38, and 39 basis-points increase in the likelihood of cognitive disability, ambulatory difficulty, and independent living difficulty, respectively. These can be translated into a change of 7.8, 6.3, and 5.9 percent with respect to the mean of the outcomes, respectively. Although the coefficient of vision-or-hearing difficulty is positive, it is statistically insignificant, which limits further interpretations (column 8).

To understand what portion of correlations can be explained by these channels, we use estimates from previous research. Lindahl (2005) investigates the effects of income on health and mortality. He estimates that an increase of 10 percent in income is associated with a reduction of about 3 percentage-points in 5-year mortality, off a mean of 0.07. This can be translated into a reduction of about 43 percent with respect to the mean. Based on column 4 of Table 7, a one-standard-deviation rise in birth-state infant mortality is correlated with a roughly 2 percent decrease in total personal income. Based on Lindahl (2005)'s estimates, this drop in income suggests an increase in mortality by about 8.6 percent. On the other hand, a one-standard-deviation rise in birth-state infant mortality rate is associated with a 0.033 standard-deviation change in mortality (column 3 of Table 3). The mean and standard deviation of the mortality rate in the final sample are 1.19 and 5.18, respectively (per 1,000). Therefore, a 0.033 standard-deviation change in mortality is roughly a change of 14.3 percent with respect to the mean. The implied change based on Table 7 and Lindahl (2005) is roughly 60 percent of the reduced-form estimates of the

main results ($\frac{8.6}{14.3}$). Therefore, a considerable portion of the effects can be explained by reductions in income (and arguably education).

5. Conclusion

Understanding the determinants of health and mortality outcomes is important for policymakers as they provide solutions to improve these outcomes. A recently developed and growing literature points to the relevance of early-life and childhood environments and conditions for life-cycle health outcomes (Almond et al., 2018; Almond & Currie, 2011a, 2011b). The current study joined this literature and investigated the associations between state-level infant mortality experienced during in-utero and early-life, as a measure of general disease environment, undernutrition, and lack of healthcare access, on later-life old-age age-specific mortality rates. We employed the universe of death records in the US over the years 1979-2020 and implemented two-way fixed effect models to examine these associations.

Conditional on a full set of fixed effects and controls, a one-standard-deviation higher infant mortality rate (roughly equivalent to the difference in infant mortality rate in 1930 and 1940) is associated with an increase of 0.03 standard deviation in age-specific mortality rate, conditional on survival up to age 55 (roughly 13% change with respect to the mean of mortality rate over the sample period and across all ages). However, we observed that these effects are more concentrated in older ages, specifically ages past 70, suggesting delayed influences of early-life exposures. Moreover, the heterogeneity analysis by birth cohort revealed that the scarring influences of early life disease environment are more pronounced for earlier cohorts who were exposed to higher infant mortality regimes than later cohorts. Finally, we provided evidence of negative correlations between early-life infant mortality rate and later-life education, income, and socioeconomic scores. We also found positive correlations with various measures of disability as potential contributors to mortality. We argued that a large portion of the observed associations for mortality can be explained by changes in these mediatory channels.

One way to put the results into perspective is to calculate potential changes in life expectancy associated with changes in infant mortality rates experienced during early life. Between 1915 and 1940, infant mortality rates dropped by about 58% in our final sample. With respect to the mean of the infant mortality rate, this number is equivalent to roughly a two standard deviation change. Based on the main results of Table 3, this change is associated with roughly 0.06

standard deviation change in mortality rate. The mean and standard deviation of mortality in the final sample are 1.19 and 5.18, respectively. Using these statistics, we can deduce that a two-standard-deviation change in infant mortality results in a roughly 26% drop in later-life mortality. Using simplifying assumptions, this can be converted into a change in life expectancy at age 60 of about 6 years. At the beginning of the NCHS data (year 1979), life expectancy at age 60 was 23.3 and 26.1 for males and females, respectively (Social Security, 2024). Therefore, the associated change in life expectancy is roughly 23-25 percent of the baseline life expectancy (conditional on survival up to age 60) for these cohorts.

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Tables

Table 1 - Birth States in the Final Sample for Selective Years

1915	1920
Connecticut	California
District of Columbia	Connecticut
Maine	District of Columbia
Massachusetts	Indiana
Michigan	Kansas
Minnesota	Kentucky
New Hampshire	Maine
New York	Maryland
Pennsylvania	Massachusetts
Rhode Island	Michigan
Vermont	Minnesota
	Nebraska
	New Hampshire
	New York
	North Carolina
	Ohio
	Oregon
	Pennsylvania
	South Carolina
	Utah
	Vermont
	Virginia
	Washington
	Wisconsin

Table 2 - Summary Statistics

	Birth Years 1915-1927		Birth Years 1927-1940	
	Mean	Std. Dev.	Mean	Std. Dev.
Mortality Rate (per 100K)	1.42	6.01	0.98	4.27
Log Mortality Rate	3.38	1.51	3.1	1.43
Infant Mortality (per 100K)	78.39	15.64	55.97	12.02
Log Infant Mortality Rate	4.36	.19	4.02	.21
Number of Deaths (Numerator, 100K)	35.28	179.31	27.64	137.46
Population (Denominator, 100K)	.36	.4	.41	.43
Age	74.33	9.26	71.61	9.24
Female	.46	.31	.43	.32
Nonwhite	.06	.19	.08	.21
Birth-State Covariates:				
Share of White-Collar Workers	.04	.01	.05	.01
Share of Farmers	.17	.12	.14	.1
Share of Other Occupations	.79	.11	.81	.1
Socioeconomic Index	25.98	3.68	27.6	3.65
Share of Literate	.76	.05	.48	.26
Share of Midwives (per 100K)	2.84	2.46	1.15	1.69
Share of Doctors (per 1K)	1.49	.38	1.54	.43
Observations	308,054		336,327	

Table 3 - Birth State Infant Mortality and Later-Life Mortality

	Outcome: Age-Specific Mortality Rate (STD)		
	(1)	(2)	(3)
Birth-State Infant Mortality Rate *(STD)*	.03214***	.03269***	.03274***
	(.00929)	(.00945)	(.0089)
Observations	644,381	644,381	644,381
R-squared	.02834	.06465	.06528
Mean DV	0.000	0.000	0.000
Birth-State FE	✓	✓	✓
Region-of-Birth by Birth-Year FE	✓	✓	✓
Death-State by Death-Year FE		✓	✓
Birth-State Controls			✓

Notes. Standard errors, clustered on birth-state and birth-year, are in parentheses. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate individuals, share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.

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

Table 4 - Heterogeneity in Correlations of Birth State Infant Mortality and Later-Life Mortality across Ages and Cohorts

	Outcome: Age-Specific Mortality Rate (STD)			
	Age 64-72; Birth Years 1915-1927	Age 64-72; Birth Years 1928-1940	Age 73-80; Birth Years 1915-1927	Age 73-80; Birth Years 1928-1940
	(1)	(2)	(3)	(4)
Birth-State Infant Mortality	.00912***	.00831**	.01346***	.00895
Rate *(STD)*	(.0033)	(.00342)	(.00475)	(.00579)
Observations	82628	94334	81332	93456
R-squared	.07447	.06458	.074	.06394
Mean DV	-0.039	-0.077	0.084	0.016

Notes. Standard errors, clustered on birth-state and birth-year, are in parentheses. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. All regressions include birth-state fixed effects, birth-year fixed effects interacted by region-of-birth fixed effects, death-state by death-year fixed effects, and birth-state controls. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate individuals, share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.
*** p<0.01, ** p<0.05, * p<0.1

Table 5 – Heterogeneity in the Correlations by Race and Gender

	Outcome: Age-Specific Mortality Rate (per 100K)			
	Whites	Nonwhites	Females	Males
	(1)	(2)	(3)	(4)
Birth-State Infant Mortality Rate	.02045***	-.01364*	.02564***	.03872***
(per 100K)	(.00661)	(.00712)	(.00661)	(.01061)
Observations	993082	313713	509070	552342
R-squared	.04926	.11767	.06706	.06946
Mean DV	-0.012	0.120	-0.041	0.038

Notes. Standard errors, clustered on birth-state and birth-year, are in parentheses. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. All regressions include birth-state fixed effects, birth-year fixed effects interacted by region-of-birth fixed effects, death-state by death-year fixed effects, and birth-state controls. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate individuals, share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.
*** p<0.01, ** p<0.05, * p<0.1

Table 6 - Heterogeneity in the Correlations by Causes of Death

	Outcome: Mortality Rate (STD), Cause:							
	TB, Syphilis Other Infectious Diseases	Neoplasm Diseases	Diabetes	Cardiovascular Diseases	Influenza, Pneumonia, Peptic Ulcer, Nephritis	All other Diseases	External Causes	Suicide
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Birth-State Infant Mortality Rate (STD)	.03308**	.01899*	.00115	.03498**	.01761*	-.00059	.03105***	.0299**
	(.01584)	(.01049)	(.00759)	(.01357)	(.00923)	(.00683)	(.00812)	(.01195)
Observations	35160	376644	169153	385735	267997	284380	123935	51319
R-squared	.37758	.14567	.16154	.12841	.14593	.1294	.21753	.5134
Mean DV	-0.001	-0.000	-0.000	-0.000	0.000	-0.000	-0.000	-0.000

Notes. Standard errors, clustered on birth-state and birth-year, are in parentheses. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. All regressions include birth-state fixed effects, birth-year fixed effects interacted by region-of-birth fixed effects, death-state by death-year fixed effects, and birth-state controls. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate individuals, share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.
*** p<0.01, ** p<0.05, * p<0.1

Table 7 - Mechanism Channel Using Census Data 1980-2000 and American Community Survey Data 2001-2020

	Outcomes:							
	Socioeconomic Score	Education < High School	Education < 12	Log Total Personal Income	Cognitive Disability	Ambulatory Difficulty	Independent Living Difficulty	Vision or Hearing Difficulty
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Birth-State Infant Mortality Rate (STD)	-.4299633***	.0111587***	.0182048***	-.0182211***	.0021668**	.0038622***	.0039649***	.0009527
	(.1018682)	(.002132)	(.0024008)	(.0027712)	(.0008671)	(.000978)	(.0009832)	(.0008822)
Observations	11951216	21555007	21555007	20212792	17630188	17630188	20530879	17630188
R-squared	.0529232	.0756103	.1063869	.1297212	.026888	.0604788	.0661691	.0619006
Mean DV	46.498	0.062	0.148	10.267	0.078	0.199	0.109	0.118

Notes. Standard errors, clustered on birth-state and birth-year, are in parentheses. The independent variable is standardized (STD) with respect to their sample's mean and standard deviation. All regressions include birth-state fixed effects, birth-year fixed effects interacted by region-of-birth fixed effects, death-state by death-year fixed effects, and birth-state controls. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate individuals, share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census. The income values are in 2020 dollars.
** p<0.01, ** p<0.05, * p<0.1

Figures

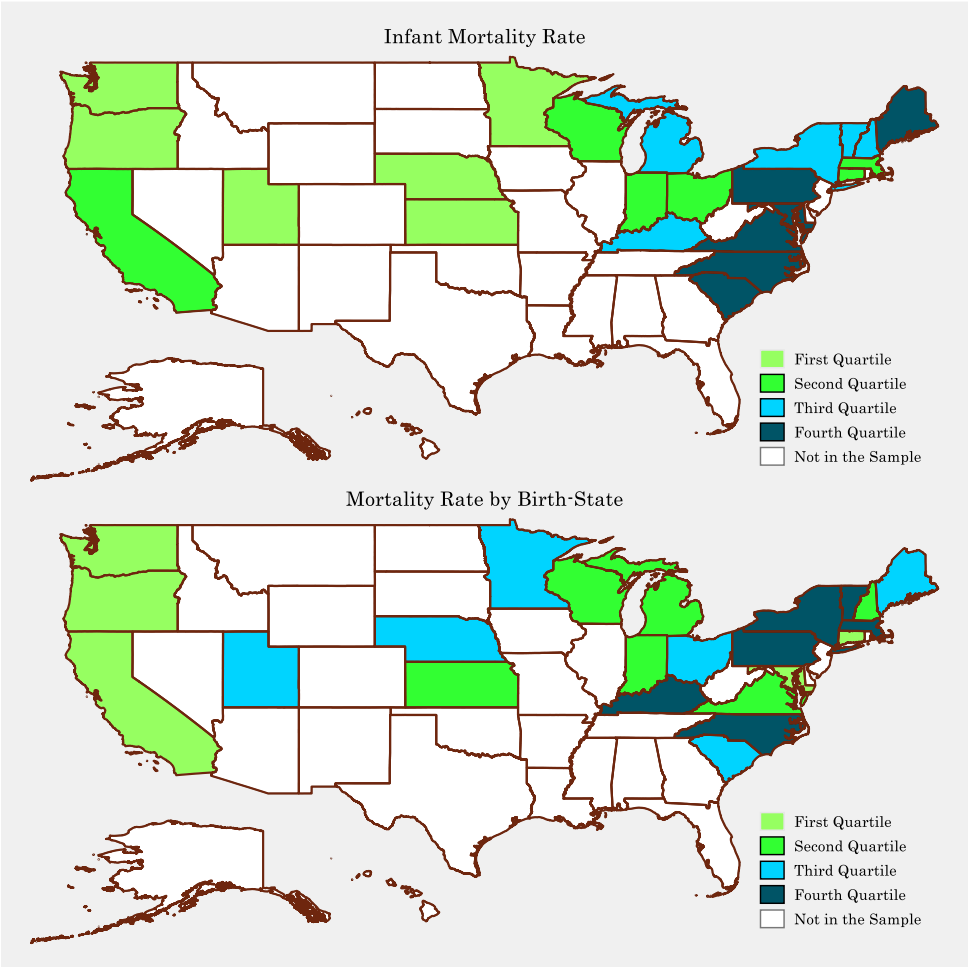


Figure 1 - Geographic Distribution of Infant Mortality Rate by Birth States

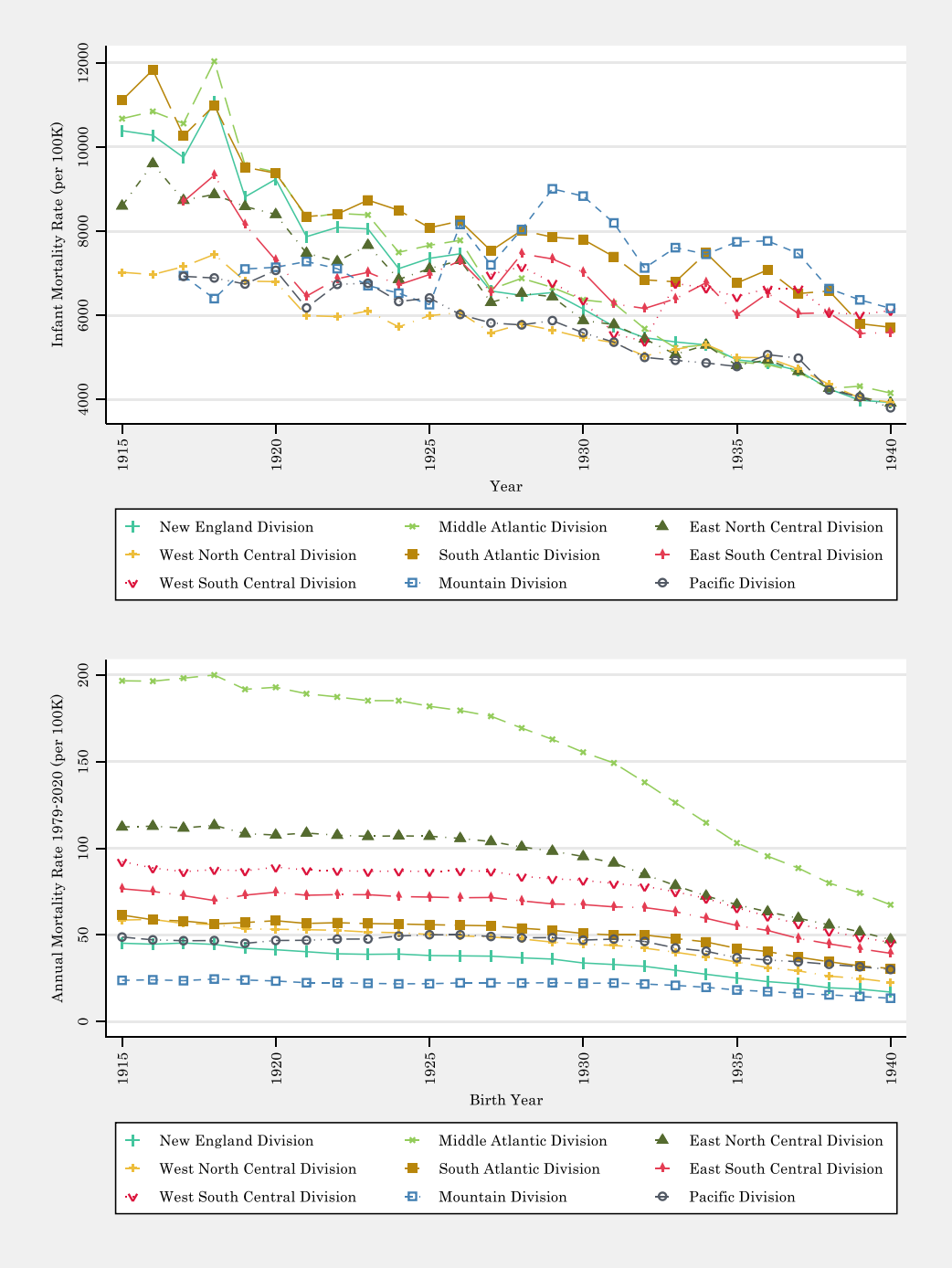


Figure 2 - Evolution of Infant Mortality and Later-Life Mortality Rates across Birth Cohorts and Census Divisions

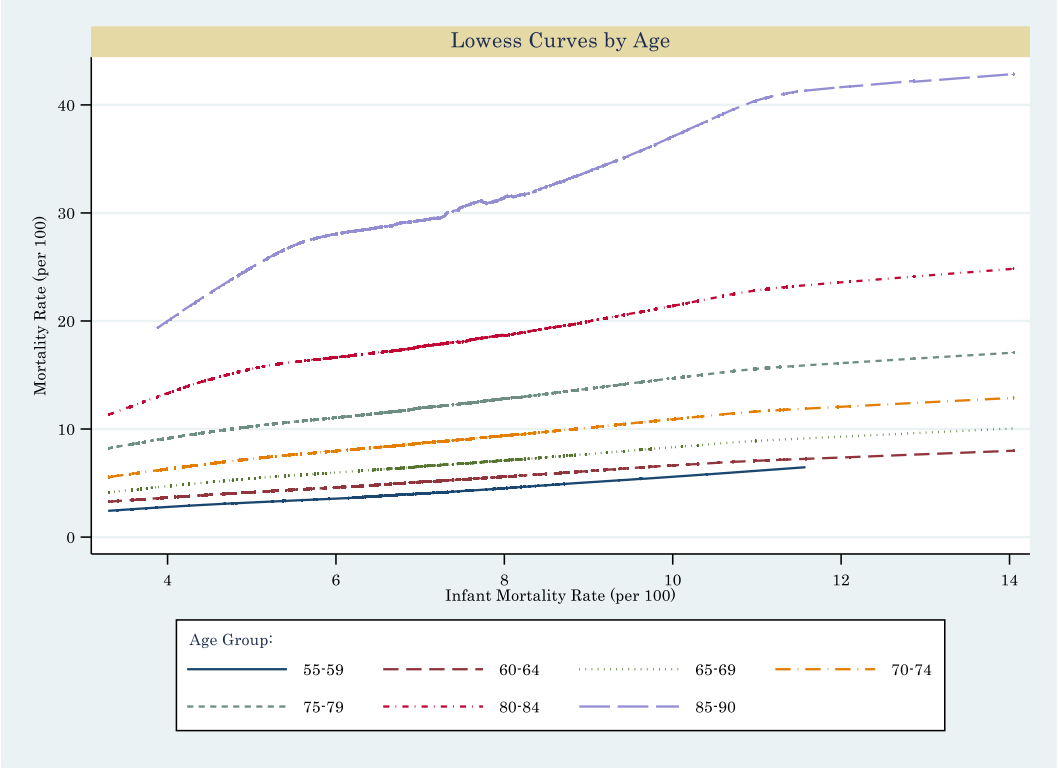


Figure 3 - Locally Weighted Scatterplot Smoothing (LOWESS) Curves of Correlation between Infant Mortality Rate and Age-Specific Mortality Rate

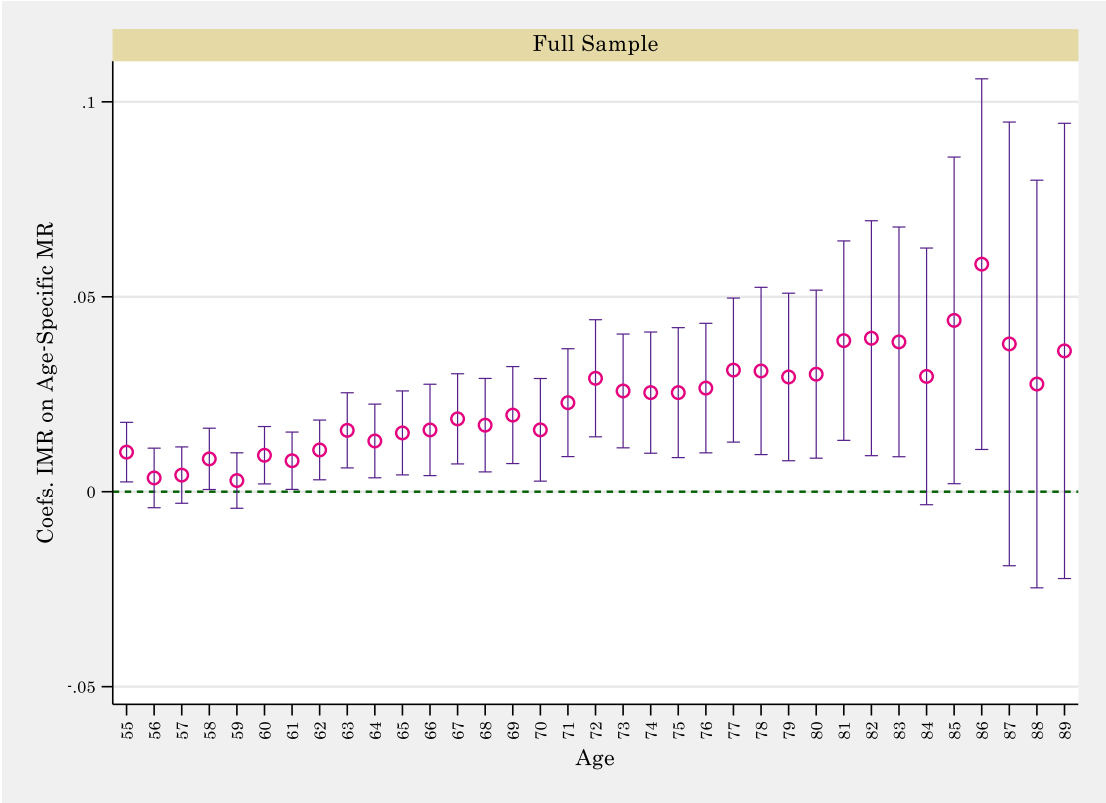


Figure 4 - the Association between Birth-State Infant Mortality Rate and Age-Specific Mortality Rate across Different Ages

Notes. Regression coefficients and 95% confidence intervals are illustrated. Standard errors are clustered on birth-state and birth-year. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. All regressions include birth-state fixed effects, birth-year fixed effects interacted by region-of-birth fixed effects, death-state by death-year fixed effects, and birth-state controls. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.

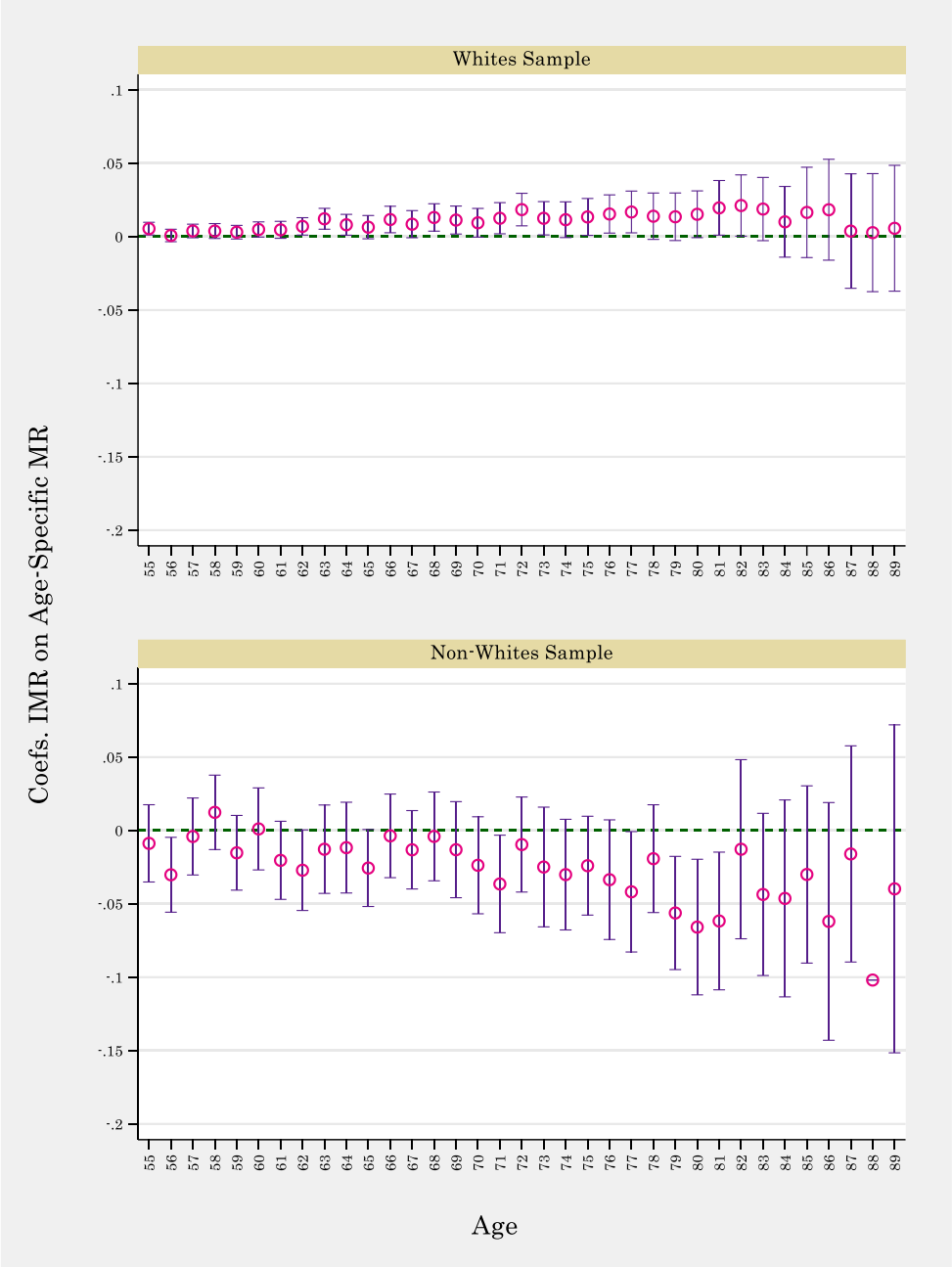


Figure 5 - the Association between Birth-State Infant Mortality Rate and Age-Specific Mortality Rate across Different Ages and Races

Notes. Regression coefficients and 95% confidence intervals are illustrated. Standard errors are clustered on birth-state and birth-year. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. All regressions include birth-state fixed effects, birth-year fixed effects interacted by region-of-birth fixed effects, death-state by death-year fixed effects, and birth-state controls. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.

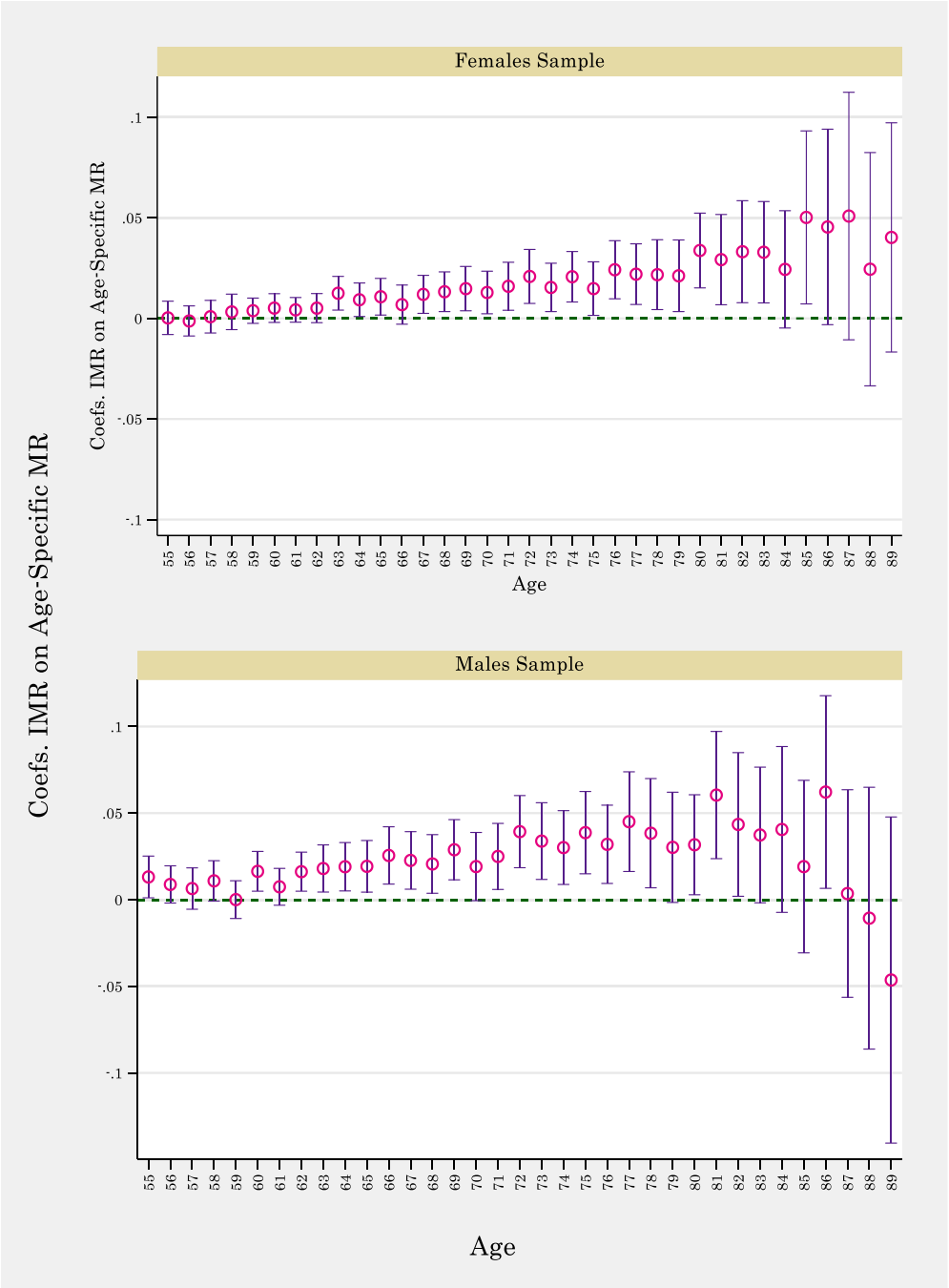


Figure 6 - the Association between Birth-State Infant Mortality Rate and Age-Specific Mortality Rate across Different Ages and Genders

Notes. Regression coefficients and 95% confidence intervals are illustrated. Standard errors are clustered on birth-state and birth-year. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. All regressions include birth-state fixed effects, birth-year fixed effects interacted by region-of-birth fixed effects, death-state by death-year fixed effects, and birth-state controls. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.

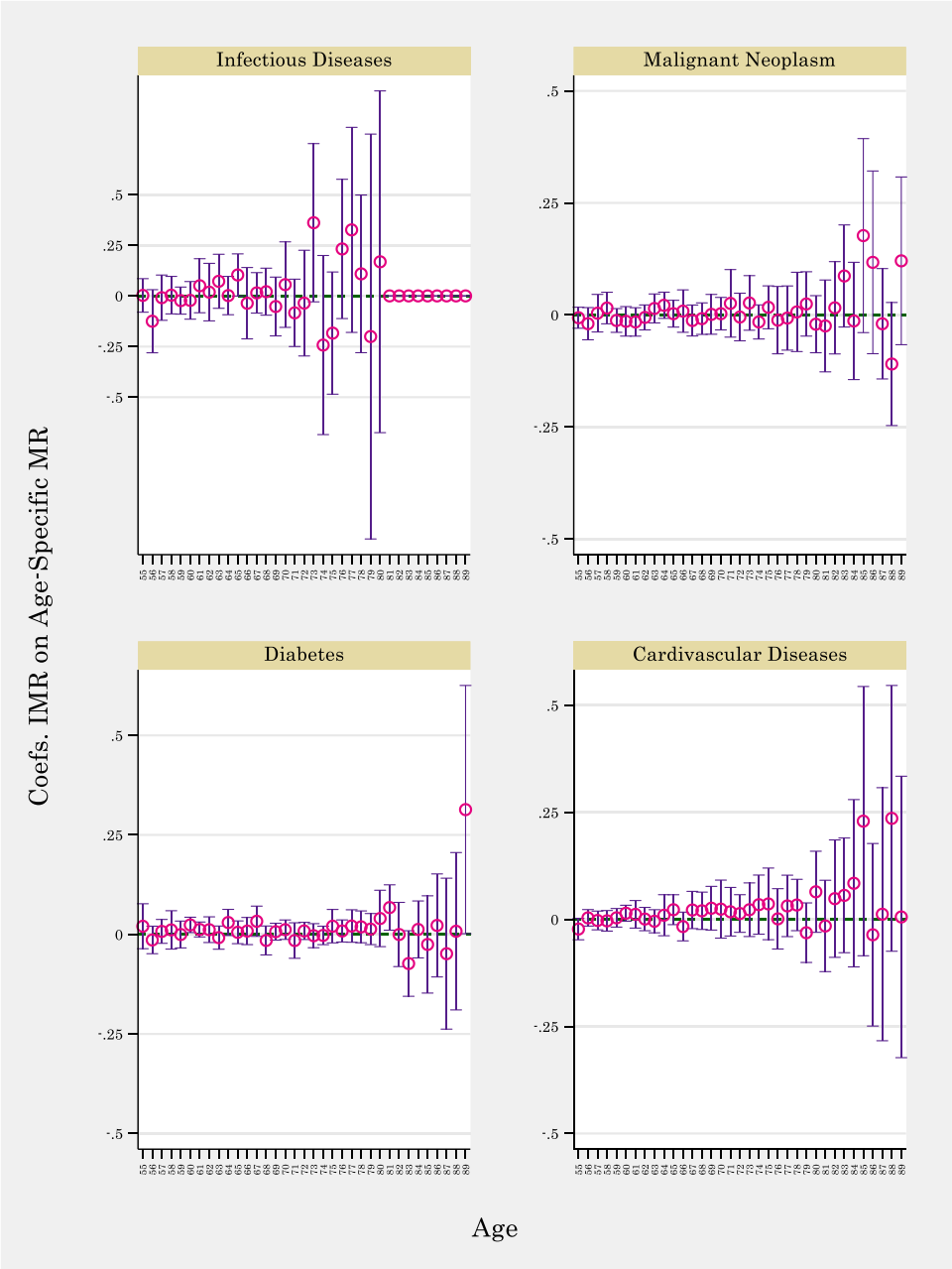


Figure 7 - the Association between Birth-State Infant Mortality Rate and Age-Specific Mortality Rate across Different Ages and Cause-of-Death

Notes. Regression coefficients and 95% confidence intervals are illustrated. Standard errors are clustered on birth-state and birth-year. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. All regressions include birth-state fixed effects, birth-year fixed effects interacted by region-of-birth fixed effects, death-state by death-year fixed effects, and birth-state controls. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.

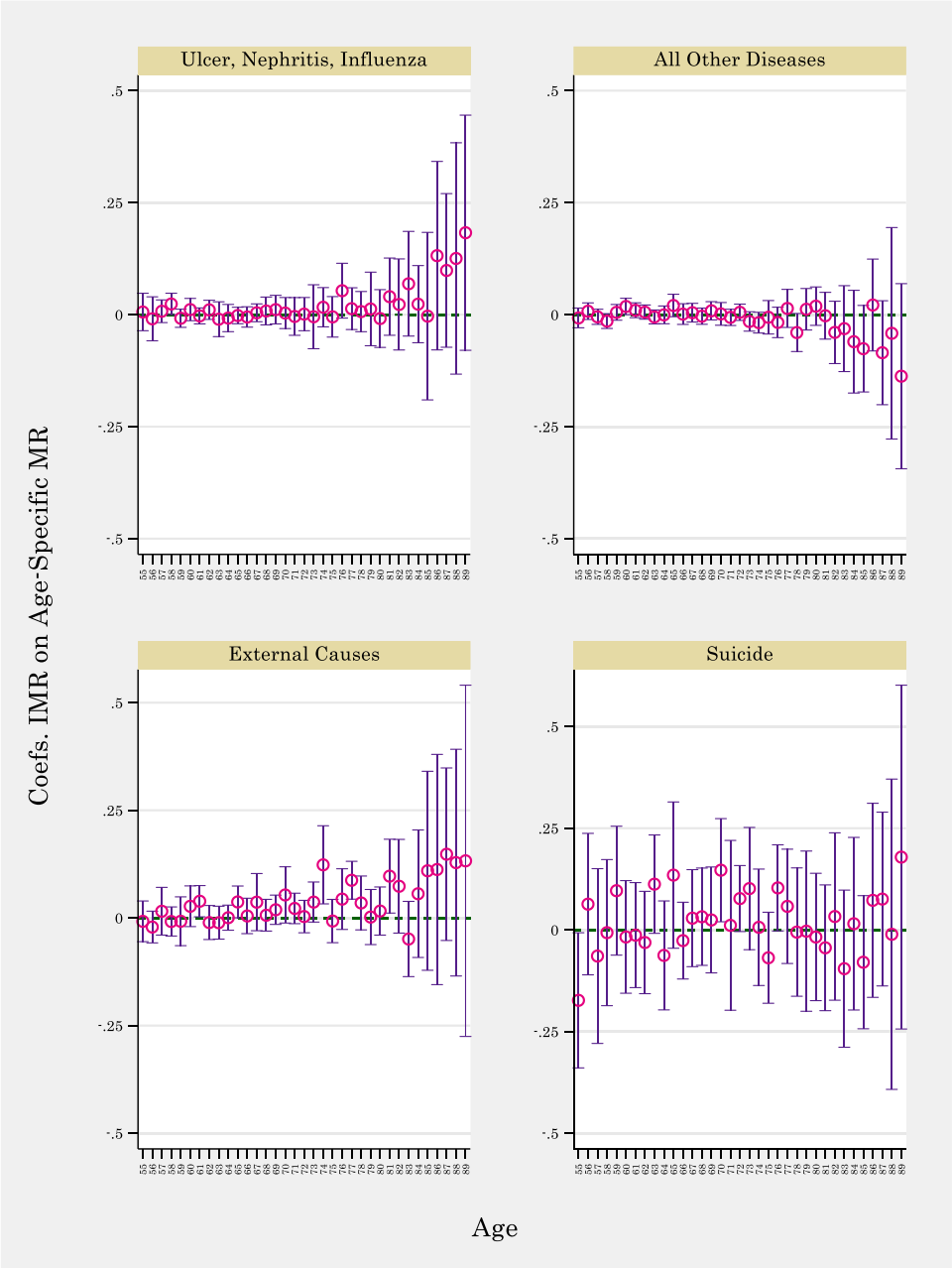


Figure 8 - the Association between Birth-State Infant Mortality Rate and Age-Specific Mortality Rate across Different Ages and Cause-of-Death

Notes. Regression coefficients and 95% confidence intervals are illustrated. Standard errors are clustered on birth-state and birth-year. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. All regressions include birth-state fixed effects, birth-year fixed effects interacted by region-of-birth fixed effects, death-state by death-year fixed effects, and birth-state controls. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.

Appendix A

In this appendix, we examine the robustness of our results to different functional forms. In Appendix Table A-1, we replace the continuous measure of infant mortality rate with two dummies indicating the second and third terciles of birth-state infant mortality rate (leaving the first tercile as the reference group). Both terciles provide positive and significant coefficients, i.e., higher mortality rates due to exposure to higher infant mortality regimes. Further, the coefficient of the third tercile is roughly 70% larger than the second tercile, suggesting that the influence of early-life disease environment is higher as we move to state-years with higher infant mortality rates.

In Appendix Table A-2, we examine the sensitivity of the results to using logarithmic functional forms. In column 1, we use the log of birth-state infant mortality rate while keeping the outcome continuous. In column 2, we keep the birth-state infant mortality rate continuous and use a logarithmic outcome. In column 3, we use a log-log specification. In all cases, we observe positive and significant coefficients. For example, a 100% percent increase in birth state infant mortality rate is associated with a 14.2% rise in later-life mortality (column 3).

As a further check, we include an interaction term of the infant mortality rate with itself in the regression model, i.e., using a polynomial regression model. If the relationship is not linear and the magnitude of effects varies significantly at different levels of infant mortality rate, then we expect to observe a positive and significant coefficient for the interaction term. The results, reported in Appendix Table A-3, only weakly support this. Although the interaction terms are positive, the fact that the interaction term is comparably small in magnitude and statistically insignificant lends support to our choice of functional form. However, the fact that the main effect is smaller in size compared to those of Table 3 and the interaction term is positive implies a small

degree of nonlinearity, i.e., the impacts are larger as we move to higher levels of infant mortality rates.

Appendix Table A-1 - Linearity of Birth State Infant Mortality and Later-Life Mortality

	Outcome: Age-Specific Mortality Rate (STD)		
	(1)	(2)	(3)
Second Tercile of Birth-State Infant Mortality	.01477***	.01624***	.02252***
	(.00557)	(.00584)	(.00508)
Third Tercile of Birth-State Infant Mortality	.02118***	.02333***	.03858***
	(.0073)	(.00759)	(.00809)
Observations	644381	644381	644381
R-squared	.0283	.06461	.06527
Mean DV	0.000	0.000	0.000
Birth-State FE	✓	✓	✓
Region-of-Birth by Birth-Year FE	✓	✓	✓
Death-State by Death-Year FE		✓	✓
Birth-State Controls			✓

Notes. Standard errors, clustered on birth-state and birth-year, are in parentheses. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate individuals, share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.

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

Appendix Table A-2 - Birth State Infant Mortality and Later-Life Mortality: Robustness to Functional Form

	Outcomes:		
	Age-Specific Mortality Rate (STD)	Log *Age-Specific Mortality Rate*	Log *Age-Specific Mortality Rate*
	(1)	(2)	(3)
Log Birth-State Infant Mortality Rate	.08362***		.14215***
	(.03054)		(.04355)
Birth-State Infant Mortality Rate *(STD)*		.02755***	
		(.00954)	
Observations	644381	644381	644381
R-squared	.06525	.44754	.44755
Mean DV	0.000	2.783	2.783

Notes. Standard errors, clustered on birth-state and birth-year, are in parentheses. Variables labeled with *STD* are standardized with respect to their sample's mean and standard deviation. All regressions include birth-state fixed effects, birth-year fixed effects interacted by region-of-birth fixed effects, death-state by death-year fixed effects, and birth-state controls. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate individuals, share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.
*** p<0.01, ** p<0.05, * p<0.1

Appendix Table A-3 - Birth State Infant Mortality and Later-Life Mortality: Examining the Nonlinearity Using a Polynomial Regression Model

	Outcome: Age-Specific Mortality Rate (STD)		
	(1)	(2)	(3)
Birth-State Infant Mortality Rate *(STD)*	.02605***	.02706***	.02023**
	(.00992)	(.01017)	(.00817)
Birth-State Infant Mortality Rate *(STD)*× Birth-State Infant Mortality Rate *(STD)*	.00292	.0027	.00487
	(.00421)	(.00424)	(.00387)
Observations	644381	644381	644381
R-squared	.02834	.06465	.06529
Mean DV	0.000	0.000	0.000
Birth-State FE	✓	✓	✓
Region-of-Birth by Birth-Year FE	✓	✓	✓
Death-State by Death-Year FE		✓	✓
Birth-State Controls			✓

Notes. Standard errors, clustered on birth-state and birth-year, are in parentheses. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate individuals, share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.

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

Appendix B

In the main results of the paper, we restrict the sample to include birth states that consistently report infant mortality rates from the year 1920 onward. This selection eliminates the inclusion of states that joined the death registration area after 1920. In Appendix Table B-1, we show the results without this sample selection and include all states with available infant mortality rates regardless of how many years they are included in the sample. The point estimate of the full specification of column 3 suggests a smaller effect compared with that of the main results, 0.02 versus 0.03. In Appendix Table B-2, we implement a stricter sample selection and include states that have infant mortality data throughout the birth years in the final sample, i.e., 1915-1940. We observe a considerably larger coefficient size compared with that of the main results. Appendix Figure B-1 reports the associations across different ages at death for the main results as well as the two subsamples we used in Appendix Table B-1 and Appendix Table B-2.

One interpretation of these different coefficients relates to two facts that we documented in the paper. First, the impacts are relatively larger at higher levels of infant mortality. Second, the coefficients rise in magnitude at higher ages at death. The inclusion of later-joining states mechanically adds more later cohorts to the sample who are exposed to lower levels of infant mortality rates (since infant mortality drops sharply across cohorts in this period). Furthermore, these cohorts will appear at younger ages in death records compared with earlier cohorts. This is primarily because the death window closes in 2020 in our sample. Both of these elements push the coefficients downward as we include more states that joined the data in later birth years.

Appendix Table B-1 – Replicating the Main Results without Restricting the Sample to a Balanced Panel of States

	Outcome: Age-Specific Mortality Rate (STD)		
	(1)	(2)	(3)
Birth-State Infant Mortality Rate (STD)	.01367**	.01346**	.01844***
	(.00587)	(.00605)	(.00666)
Observations	1026477	1026477	1026477
R-squared	.02651	.04694	.04724
Mean DV	0.000	0.000	0.000
Birth-State FE	✓	✓	✓
Region-of-Birth by Birth-Year FE	✓	✓	✓
Death-State by Death-Year FE		✓	✓
Birth-State Controls			✓

Notes. Standard errors, clustered on birth-state and birth-year, are in parentheses. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate individuals, share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.

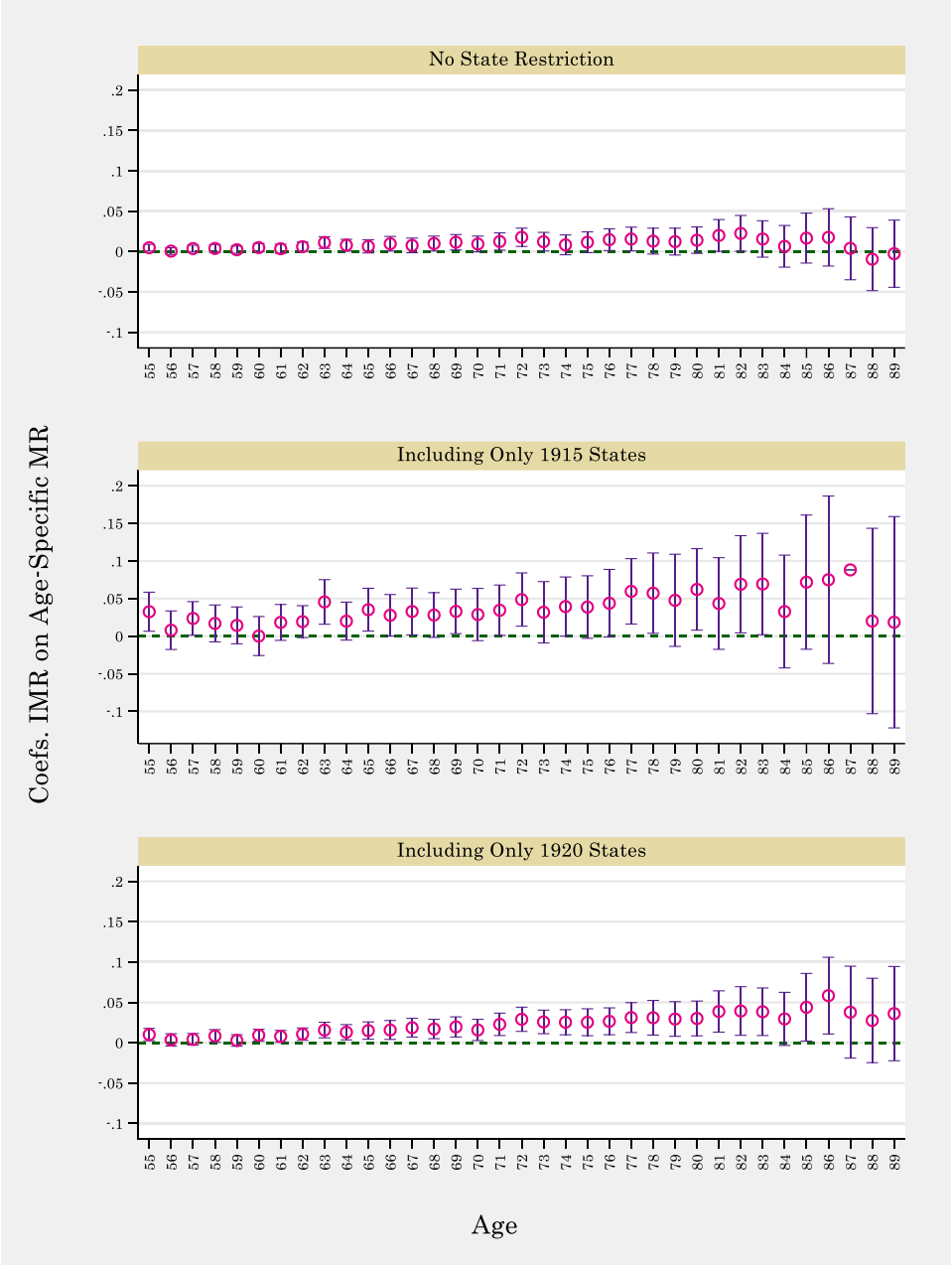
*** p<0.01, ** p<0.05, * p<0.1

Appendix Table B-2 - Replicating the Main Results with Restricting the Sample to a Balanced Panel of 10 States Reporting Infant Death across All Years of 1915-1940

	Outcome: Age-Specific Mortality Rate (STD)		
	(1)	(2)	(3)
Birth-State Infant Mortality Rate (STD)	.054***	.05557***	.05863***
	(.01967)	(.01983)	(.01792)
Observations	301077	301077	301077
R-squared	.04261	.122	.12385
Mean DV	-0.000	-0.000	-0.000
Birth-State FE	✓	✓	✓
Region-of-Birth by Birth-Year FE	✓	✓	✓
Death-State by Death-Year FE		✓	✓
Birth-State Controls			✓

Notes. Standard errors, clustered on birth-state and birth-year, are in parentheses. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate individuals, share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.

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



Appendix Figure B-1 - the Association between Birth-State Infant Mortality Rate and Age-Specific Mortality Rate across Different Sample Restrictions

Notes. Regression coefficients and 95% confidence intervals are illustrated. Standard errors are clustered on birth-state and birth-year. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. All regressions include birth-state fixed effects, birth-year fixed effects interacted by region-of-birth fixed effects, death-state by death-year fixed effects, and birth-state controls. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.

Appendix C

The denominator of mortality rates in the paper is calculated based on current state-year population counts. The reason is that our data source regarding population estimates does not report population based on the level of mortality estimates in the numerator, i.e., birth state, current state, birth year, current year, and age. While we acknowledge this data limitation in our mortality estimates, we further examine the robustness of the results using an alternative data source for the denominator. Specifically, we use population estimates based on random samples of decennial censuses combined with the American Community Survey data. The disadvantage of this data source is that it does not cover inter-decennial years 1981-1989 and 1991-1999. Further, the estimates are based on random samples rather than the full population. The main advantage, however, is that we can estimate the population at the numerator level (i.e., birth state, current state, birth year, current year, and age). Therefore, for the years 1980, 1990, and 2000-2020, we can calculate mortality rates using numerators and denominators at the same level of aggregation. The results are reported in Appendix Table C-1. The fully parameterized regression of column 3 is almost identical to the main results.

Appendix Table C-1 - Replicating the Main Results Using Census-Based Denominator

	Outcome: Age-Specific Mortality Rate (STD)		
	(1)	(2)	(3)
Birth-State Infant Mortality Rate *(STD)*	.02231*	.01778	.03114***
	(.01271)	(.0116)	(.01141)
Observations	234812	234812	234812
R-squared	.05413	.15519	.15549
Mean DV	-0.000	-0.000	-0.000
Birth-State FE	✓	✓	✓
Region-of-Birth by Birth-Year FE	✓	✓	✓
Death-State by Death-Year FE		✓	✓
Birth-State Controls			✓

Notes. Standard errors, clustered on birth-state and birth-year, are in parentheses. Both independent variable and dependent variable are standardized (STD) with respect to their sample's mean and standard deviation. Controls include birth-state share of white-collar workers, share of farmers, share of other occupations, socioeconomic index, share of literate individuals, share of midwives, and share of doctors. Regressions are weighted using the average number of state-level age-level population in the 1980 census.

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