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Joanna N. Lahey
Marianne H. Wanamaker

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ABSTRACT

Recent studies based on 20th century US data conclude that abortion access raises children's average socioeconomic outcomes. We generalize a model of fertility, highlighting assumptions under which these abortion predictions can be reversed. Using 19th century abortion restrictions, we empirically demonstrate these points. Despite a more than 5 percent increase in birth rates among abortion-restricted cohorts, we find little evidence of negative selection at birth. Longevity was affected nevertheless; in the first ten years of life, children in these larger cohorts died of infectious disease more frequently. These mortality effects diminish with age, potentially reversing at older ages as a result of disease immunity or other offsetting factors.

Joanna N. Lahey
The Bush School
Texas A&M University
Mailstop 4220
College Station, TX 77843
and NBER
jlahey47@gmail.com

Marianne H. Wanamaker
Department of Economics
University of Tennessee
524 Stokely Management Center
Knoxville, TN 37996
and NBER
wanamaker@utk.edu

I. Introduction

A well-known theoretical and empirical literature connects abortion legalization in the 1970s to higher socioeconomic status of children at birth and improvements in long-run outcomes for affected birth cohorts (Charles and Stephens 2006; Donohue and Levitt 2001; Gruber, Levine, and Staiger 1999; Pabayo et al. 2020). Similar studies for other contraceptive methods and family planning programs also tend to find an improvement in children's living conditions accompanying expanded birth control and abortion access (Ananat and Hungerman 2012; Bailey 2010; Bailey 2012).

The findings of these studies, based on the experience of the United States in the last six decades, may not hold out of sample. In particular, the relationship between fertility control and both socioeconomic status at birth and long-run outcomes may depend on the initial level of development. That the "marginal child" in the 1970s U.S. was of lower socioeconomic status reflects general income levels, the availability of substitutes for abortion, such as contraception, and other context-specific factors. These considerations are important for understanding the impact of abortion availability for average child outcomes earlier in U.S. history, as well as for understanding the relationship between abortion availability and child outcomes in other countries in the modern era. For example, Pop-Eleches (2006) finds that the unexpected ban on abortions in 1966 Romania resulted in better schooling and education outcomes for children born after the ban, suggesting abortion was being primarily used by high socioeconomic status Romanians prior to the ban.

In this paper, we develop a more general model of marginal child selection in the presence of abortion and show that predictions about the socioeconomic status of the marginal child are weakened, or even reversed, under a number of alternative assumptions. Specifically, a high marginal utility of income, high time discount rates, and high returns to child labor could all portend marginal children of *above* average socioeconomic status. Notably, each of these alternative assumptions may hold in settings with lower levels of economic development.

We examine these theoretical predictions by exploiting variation in 19th century United States abortion laws and assembling new data to track their impact. Abortion-restricting legislation in the latter part of the century varied in timing, as well as severity and comprehensiveness, across U.S. states. Lahey (2014a, 2014b) assembles a dataset of these laws and documents that they were associated with increases in the child-woman ratio of at least 5 percent. We utilize the same law dataset to measure the relationship between these laws and the socioeconomic status of children at birth, their health (measured by rates of mortality), and the causes of death driving that mortality. To do so, we generate a new panel of cause of death data from 1850 through 1940, as described in more detail below.

To estimate the impact of abortion-restricting laws on socioeconomic outcomes, we define birth cohorts by year and state of birth, and use complete count U.S. Census of Population returns for 1850 through 1947 to calculate average child circumstances. A simple difference-in-differences estimator compares the outcomes of (larger) abortion-restricted cohorts to (smaller) cohorts without abortion restrictions.[1] On many metrics, we find little measurable difference in socioeconomic status at birth among individuals in the birth cohorts born in states and years with laws outlawing abortion, despite increases in the child-woman ratio documented in Lahey (2014a). Although children in these cohorts appear somewhat more likely to be born into households with fathers present, less likely to live in an urban location, and less likely to live in group quarters, these results are sensitive to sample selection choices and lose economic and statistical significance under more narrow identification specifications. We conclude that marginal children in the 19th century U.S. were, if anything, positively selected relative to the population at large, a reversal of the 20th century result.

Finding little evidence of socioeconomic selection at birth, we next examine the effects of larger cohort sizes on population health, motivated by previous literature on the deleterious effects of larger birth cohorts (e.g. Easterlin 1980, 1987; Macunovich 2000, 2002). Despite the weak positive selection at birth, our analysis of cohort survival rates indicate the survival probability of children born into these cohorts was markedly reduced, and that enhanced mortality from infectious disease is largely to blame. Using simple decadal cohort survival rates derived from successive enumerations of the U.S. Census of Population, we find 19th century abortion-restricted cohorts of both sexes were approximately two percentage points less likely to survive each of the first two decades of life. This survival penalty diminishes with age, and survivors among abortion-restricting cohorts may even be more likely to survive at older decades.

In an effort to generate cohort-specific cause of death data for cohorts born in the 19th century, we coded cause of death data for more than 1 million decedents in the 1850, 1860, 1870, and 1880 Censuses of Mortality and transcribed cause of death counts from 20th century U.S. Vital Statistics reports from 1939 through 1947. Together, these data represent a panel of cause of death data by gender, state, and birth cohort. Using the same methodology within a multinomial logit framework, comparing abortion restricted cohorts to unrestricted cohorts within and across states, we document excess mortality from infectious disease at early ages among abortion-restricted cohorts. Yet these

[1] We discuss heterogeneous staggered treatment effects (Callaway and Sant'Anna 2021; de Chaisemartin and D'Haultfoeuille 2020, 2021; Goodman-Bacon 2021; Sun and Abraham 2021) within the context of our mortality results later in the paper.

same cohorts appear to have held some immunity to infectious disease later in life, after age 60, when they were somewhat less susceptible to death from infectious disease causes.

We conclude, then, that the effects of abortion restrictions are nuanced and heavily dependent on context. In the 19th century, limits on abortion availability appear to have induced weak positive selection among affected cohorts, raising the average socioeconomic status of these cohorts marginally at birth. This result is at odds with the finding that 20th century marginal children were of below-average socioeconomic status. At the same time, cohort size effects counteract these positive selection effects in the first decades of life when deaths from infectious disease rise among larger cohorts. These effects diminish with age; children of these larger cohorts appear to experience an immunity effect that limits mortality from infectious disease after age 60, suggesting a long horizon for positive immunity responses.

II. Historical Background and Previous Literature on Cohort Selection

We first provide a detailed contraceptive and legal history for the 19th century United States before summarizing the relevant economics literature on marginal children in the presence of abortion.

a. 19th Century Abortifacients and the Legal Environment

The nineteenth century U.S. had an active market for technologies to limit fertility, although these methods did not produce the high rates of efficacy that modern technology has and were more likely to decrease total fertility than to stop it entirely.[2] Devices, herbs, and medical procedures were prominently advertised in the many available 19th century newspapers, while pamphlets (for the literate) and popular lecture circuits (for the illiterate and others) explained practices such as the rhythm method and sexual techniques (e.g. *coitus interruptus*) that reduced the probability of pregnancy. Herbal abortifacients were thought by contemporaries to be effective in early pregnancy, though modern scholars are not certain of their actual effectiveness. Certainly, some herbal medications resulted in spontaneous abortions from making the pregnant woman dangerously sick. (See, for example, Ernst 2002; Madari and Jacobs 2004.)

[2] There is some debate on stopping versus spacing behavior in the literature that is beyond the scope of this paper. In addition to the abortion methodologies we discuss, birth control technologies included withdrawal; douching, which could lead to higher fertility if done with water or to permanent sterilization if done with certain types of acids; condoms, which became inexpensive after the vulcanization of rubber in 1844 but had little quality control; diaphragms and cervical caps, which needed to be fit properly and had higher failure rates than those today; intrauterine devices (now known as IUDs) which were effective but potentially dangerous; and the rhythm method which required instruction and generally gave incorrect timing. (See Degler 1980; Gordon 1976; or McLaren 1990 among others for more information.)

Surgical abortion was common throughout the 19th century and increased in frequency after the modern dilation and curettage, or "D and C," method was popularized in mid-century. Later in the century, abortions were also performed via membrane rupture, which, as today, could be dangerous to the health of the woman (Gordon 2002; King 1992). The frequency of 19th century surgical abortion is the subject of debate (see, *inter alia*, David and Sanderson 1986; Gordon 2002; Lahey 2014a; McFarlane and Meier 2001; McLaren 1990; Sanderson 1979; Smith-Rosenberg 1985 and Tribe 1990), but estimates are as high as one in four pregnancies ending in abortion in this period (McFarlane and Meier 2001; McLaren 1990; Tribe 1990).

In the second half of the 19th century, a moral crusade against "vice" led to government limitations on the fertility control market. In the 1860s, states began to pass specific anti-abortion laws that outlawed advertisements for the procedure and that, for the first time, prohibited abortions even before "quickening." (Abortions prior to observable movement of the fetus had traditionally been allowed under English common law.) Many of these laws also, for the first time, provided for punishment not only of abortionists but also of the women seeking abortions. Although the courts were often sympathetic to women and abortionists when violations of these new laws were brought to trial, the publicity could permanently tarnish reputations and in many cases the official investigations and court trials amounted to harassment; in some high-profile cases, the accused committed suicide before the court reached a verdict (Reagan 1991). Lahey (2014a, 2014b) describes the changes to these laws over time in more detail and directly connects their passage to increases in birthrates of between 5 and 15 percent.

b. The "Marginal Child" Literature

In the 1970s, a reversal of these 19th century changes saw abortion re-legalized in the United States, first in five states and then nationwide. Levine et al. (1999) estimate that roughly six percent fewer children were born as a result. Gruber, Levine, and Staiger (1999), in turn, examined the average characteristics of children who were born after legalization. Using the change in the birthrate and the change in the average characteristics of these smaller cohorts, Gruber, Levine, and Staiger then backed out the characteristics of the "missing" or "marginal" children who were *not* born because of the legalization of abortion. They determined that the "marginal child" would have been disadvantaged—more likely than average to have lived in a poor, single parent, or welfare-receiving household, more likely to have been of low birthweight, and more likely to have died in infancy. Bitler and Zavodny (2002) measure the effect of the increase in child wantedness following abortion legalization on

adoption rates, finding that abortion access reduced the rate of adoptions for children born to white women by 34 to 37 percent.

Subsequent research has explored the childhood and young adult outcomes for individuals born to the first post-abortion rights cohorts. These analyses have concluded that the marginal child avoided by abortion liberalization would have experienced higher rates of infant mortality (Gruber, Levine, and Staiger 1999), higher rates of drug use in young adulthood (Charles and Stephens 2006), higher rates of single parenthood, lower rates of college graduation, and higher rates of welfare enrollment (Ananat et al. 2009). Abortion legalization has also been linked, perhaps controversially, to lower rates of crime (Donohue and Levitt 2001). In sum, these projects have found that "wanted" children tended to grow up in better-than-average circumstances and experienced lower-than-average deprivation in early years. Moreover, they have concluded that increased average levels of wantedness after the early 1970s have had positive effects on cohorts into early adulthood.[3] But because cohorts affected by the 1970s abortion legalization are still living, the long-run effects of wantedness (e.g., life expectancy) remain empirically unknown.

In contrast to these results, Pop-Eleches (2006) finds the opposite effect of abortion access on outcomes of children ever born in 1966 Romania. Children born after an unexpected abortion ban had better education outcomes, implying that marginal children had higher than average education outcomes. Arguably, 1966 Romania had more in common with the 19th century United States than with the 1970s United States, making it an interesting point of comparison.

We contribute to this literature by providing U.S. estimates from a different century and a context of lower development levels. Moreover, our focus on 19th century law changes affords observation of the lifecycle effects of abortion restrictions, including health effects at older ages.

III. Conceptual Model of Socioeconomic Status, Fertility, and the Cost of Abortion

In this section, we build a conceptual model describing the interaction between parental choices, abortion legalization or access, and child socioeconomic status at birth. Our model begins with insights from Gruber, Levine, and Staiger (1999) and from Ananat et al. (2009). Changes in the cost of abortion will affect both decisions about pregnancy and about birth conditional on pregnancy. In the Ananat et al. model, abortion legalization in the 1970s lowered the cost of abortion and, therefore, induced pregnancies that would otherwise have an expected payoff that was too low to proceed to childbirth. Likewise, legalizing abortion induces additional abortions conditional on

[3] Similar results were found with the 2012 legalization of abortion in Uruguay (Antón, Ferre, and Triunfo 2018).

pregnancy by inducing abortion among individuals who otherwise would have chosen a live birth when the cost of abortion was high.

To derive predictions over the average living standards or socioeconomic conditions of "marginal" children, Ananat et al. (2009) "assume that children's outcomes are directly linked to the payoff of giving birth" (p. 126). Said another way, "more 'wanted' (i.e., higher payoff) births have better outcomes than less wanted births" (p. 126). The prediction from both selection into pregnancy and into birth conditional on pregnancy is that cohorts under abortion restrictions will exhibit a weaker set of cohort quality indicators, including more single parent households, higher poverty, higher welfare receipt, and higher rates of infant mortality. In other words, the marginal child is of lower socioeconomic status.

A simple graphical model, developed further in Appendix A, makes these points more formally. Suppose the marginal benefits of unborn children within the population can be summarized by a unimodal symmetric curve, as in Figure 1, and that the marginal cost of a child is known with certainty. Households deciding whether to abort a pregnancy or not are assessing whether the net cost of the child (the marginal cost less the marginal benefit), when born, is greater than the cost of an abortion.[4] We assume households know all benefit and cost values of an unborn child with certainty at the time of pregnancy.

The household's decision rule can be depicted in the graphical model. Pregnancies for which the marginal benefit of the child (MB_{chil}) exceeds the marginal cost (MC_{chi}) less the cost of abortion, C_A, will continue to a live birth. Other pregnancies will be aborted. Pregnancies with a marginal benefit in close proximity to the vertical $MC_{child} - C_A$ line are, themselves, considered "marginal", depicted by the shaded area in Figure 1. Populations for which the marginal benefits of children are higher, reflected by a rightward shift of the marginal benefit curve, will experience fewer abortions. Similarly, populations for which the costs of an abortion are higher will see a leftward shift in the vertical decision line and, again, experience fewer abortions.

In this model, socioeconomic-based selection at birth arises when subpopulations within a larger population have different relative positions of the MB_{child} curve and $MC_{child} - C_A$ threshold. In a population where high SES households experience higher marginal benefits of children, on average, the MB_{child} curve for high SES households is shifted to the right and there is a lower density of marginal children among high SES than among low SES households.

[4] The model's decision rule is $MC_{child} - MB_{child} > C_A$ where MC and MB represent marginal costs and benefits, respectively. Said another way, pregnancies for which $MC_{child} - C_A > MB_{child}$ will be aborted.

Abortion legalization can be represented in this schematic by a decrease in the cost of an abortion (a rightward shift in the $MC_{chi} - C_A$vertical decision threshold). When the cost of abortion falls, there will be positive selection into live births as a relatively larger number of additional low SES pregnancies are aborted compared to additional high SES pregnancies. This is the scenario envisioned in the Ananat et al. (2009) model.

The predictions of the model would be reversed if high SES households had reduced marginal benefit (a leftward shift in the MB curve) relative to low SES households, or if abortion costs were higher for low SES households, or both. Gruber et al. (1999) hints at this reversal:

> *A priori, the direction and size of selection is unclear. On the one hand, if women use abortion to avoid bearing children into adverse circumstances, positive selection would result.... On the other hand, negative selection would result if, for instance, the most disadvantaged women are constrained in their abortion access, either geographically or financially.*

Here, we point out that the expected payoff of a live birth, MB_{child} , depends on the context in which children are born. For economies at earlier stages of economic development, evidence suggests that children served as sources of household income such that a rise in the market returns to child labor induced additional births (Wanamaker 2014). The marginal benefit of children would then decline with household income, i.e., child labor is an inferior good. In this case, children from high SES households are more likely to be marginal (the MB_{child} curve is shifted to the left for high SES households), and abortion restricted cohorts of children will have *higher* socioeconomic status, reversing the direction of selection observed in the 20th century U.S.

Ambiguity in the direction of the selection effect may also come from differences in the location of the decision threshold across groups, holding the marginal benefit of children constant. For example, if the marginal utility of income is substantially higher for lower income households, this would result in a higher one-time cost of abortion and fewer marginal children among lower socioeconomic status (SES) individuals. Similarly, if there is a high discount rate among lower SES households (Pepper and Nettle 2017), wherein the future costs that children will impose on household consumption are weighted less than consumption losses today compared to other households, the result will be a leftward shift in the decision threshold among lower SES households. Credit constraints (or

higher borrowing costs) for low-income households will have a similar effect. In all of these cases, a lower density of marginal children among low SES households than among high SES households would imply negative selection from removing abortion restricting legislation and positive selection from enacting abortion restricting legislation.

IV. Empirical Evidence on Socioeconomic Selection Resulting from Abortion Restrictions

We first describe the data available for measuring the effect of abortion-restricting laws on child socioeconomic characteristics. We then use those data to estimate the degree of selection among abortion-restricted cohorts by measuring the difference in household characteristics in early life between abortion restricted and unrestricted cohorts.

a. Data on abortion laws and child outcomes

To assess the early life circumstances of marginal children, we use proxies for socioeconomic status contained in the decennial Censuses from 1850 through 1900, excluding the 1890 returns which are not available for analysis (Ruggles et al. 2015). Table 1 provides both summary statistics and census year availability for each outcome of interest.[5] Our analysis is comprised of individuals aged 0 to 9 in each census year, and our strategy effectively captures each child born after 1840 once in the 1850 through 1900 census manuscripts. Individual-level data from each census year is collapsed into a cell average for each birthyear-birthstate-sex combination.[6] The number of individuals represented by each cell average is used as a sample weight in the analysis to follow. We include only states that were admitted to the union prior to 1880 to ensure availability of census and legal information during most of the time period studied.

Information on statutes restricting abortion were originally published in Lahey (2014a, b), and additional details on their collection are available in those publications. In short, Lahey used archived state legal codes to compile a comprehensive dataset of the introduction and amendment of laws restricting activities related to birth control and abortion. These dates were confirmed using contemporary secondary sources.[7]

[5] We use full count population census data for these results whenever possible. Values for literacy and school attendance in 1880 and labor force participation and school attendance in 1900 are not available in the 100 percent samples; we use values from the largest microsample available in each year instead.
[6] The analysis here focuses solely on outcomes for white children due to limitations in early censuses.
[7] These original data are generally consistent with other *ex post* compilations and studies of these laws for various time periods (for example, Dellapenna 2006; Dennett 1926; Lader 1966; Mohr 1978; Quay 1961; Storer 1860; Storer and Heard 1868), and any inconsistencies were resolved by comparing to the original state legal codes.

Figure 2 contains the year of abortion-restricting law adoption for U.S. states. Although Southeastern states were generally late adopters while the Northeast adopted these laws earlier in the century, there remains substantial heterogeneity across and within regions in the timing of passage. In the analysis to follow, we use the first incidence of abortion-restricting laws as the effective date of treatment, although multiple laws may have been passed and the strictness may have changed over time.[8] Because very early abortion restriction laws were often part of omnibus malpractice or anti-poison laws that were only later used as anti-abortion laws (Degler 1980; Lader 1966; Mohr 1978; Polsky 1970), we drop states that passed their laws before 1840 from the analysis sample.[9] Dropping these "always treated" states also limits identification to states which are treated during the sample period.

b. Empirical Strategy and Results

To understand the socioeconomic-based selection implications of abortion restrictions in the 19th century, we estimate a series of difference-in-differences regressions in the form of Equation 1:

$$y_{scgt} = \alpha + \beta HAVELAW_{sc} + \gamma_{sg} + \gamma_{tc} + \varepsilon_{sct} \quad (1)$$

where y_{scgt} is the average value of a particular characteristic for a cohort of gender *g* born in year *c* in state *s* and measured in census year *t*. The indicator variable $HAVELAW_{sc}$ is a measure of the presence of abortion restriction legislation in state *s* in cohort *c*'s year of birth and the γ variables are state of birth by gender and census year of measurement by year of birth fixed effects. ε_{sct} is an error term that is assumed to be uncorrelated with the other independent variables in Equation 1. All estimates reflect weighted OLS where the sample weight is the number of individuals from which mean values of y_{scgt} are derived. Because all but one state passed a law by 1900, we restrict the sample to include only children born before that date. Standard errors are clustered by state of birth.

[8] In results available from the authors, we rerun our hazard specifications from Figure 3 controlling for forms of legal strictness such as medical exemptions, only a crime if the fetus dies, female fault, bans on advertising or selling via the mail, felony, any stage illegal, and quickening distinction. In general, the magnitudes remain approximately the same although there are some significance changes when controlling for various forms of legal strictness.

[9] Lahey (2014a,b) did not remove states with laws prior to 1840—in that paper these states are coded as always having laws given the universe and outcome variables used— but results for the effect of anti-abortion laws on fertility are nearly identical when removing these states. Moreover, the main results in this paper are not substantially different if we include early law states. Lahey (2014a) also determined that law passage is not related to the medical movement, immigration patterns, previous child/woman ratios, percent urban, or literacy.

We report equation (1) results for three specifications: a baseline specification including all census years and birth cohorts, a specification that includes only census years after 1860, and a specification that restricts the sample to cohorts born within a 20-year window centered on anti-abortion law adoption. The restriction to census years after 1860 is motivated by census quality concerns (with more recent census years arguably having better coverage) as well as the fact that abortion laws passed later in the century were more likely to be stand-alone laws.

We measure the effect of abortion law changes on a host of socioeconomic conditions of children at the time of their birth, including the presence of a father in the household, paternal labor force participation, parents' education as proxied by literacy, whether the household reports positive real or personal property, urban location, residence in group quarters, and whether the child's parents were immigrants. We also report results for the age of the parents. Results for additional outcomes are reported in Appendix Table A1.[10] Our sample restrictions imply that Table 2 reflects outcomes for birth cohorts born between 1841 and 1899.

Results in Table 2 suggest either no changes in the home environment of children, or a slightly improved home environment in the years following the law changes. In the baseline specification, we include all census years and all cohorts. In that specification, a modestly improved home environment is apparent in a 1 percent increase in the age of fathers from an average of 33.5, a 1 percent increase in the probability of living with both parents from an average of 83.5 percent, and a 20 percent reduction in the probability of living in group quarters from an average of 0.007 percent.[11] The reduction in paternal labor force participation is the only indicator of negative selection, but the implied change represents only a small variation from the mean.[12]

Some of these results are strengthened with a restriction to post-1860 census year observations (the second row of Table 2), with slightly larger magnitudes, and the post-1860 results also indicate a decline in the probability of urban residence of 26 percent from an average of 20.8 percent and a 3.0 percent rise in maternal literacy, from an average of 88.9 percent.

In combination, we view these results as evidence of, if anything, weak, positive socioeconomic selection among the additional children induced by abortion restrictions, a result in contrast to the findings of 20th century empirical studies.

[10] Additional outcomes in childhood are father's occupational category, mother's share of parental property, number of children less than 5 in the household, and an indicator for whether an individual was adopted.
[11] Single mother and group quarters estimates survive a Bonferroni correction for multiple inference testing. Paternal age survives at the 10 percent level but not at the 5 percent level.
[12] A 0.56 percentage point reduction in paternal labor force participation is only 0.6 percent of the mean of 0.958.

V. Previous Literature on Health-Based Selection, Scarring and Disease Immunity

Based on this evidence of weak socioeconomic based selection at birth following abortion restrictions, we now assess implications for post-birth health outcomes. Our thinking is guided by the existing literature on diseases and cohort size, and on health-based selection, scarring, and theories of long-run disease immunity.

A substantial literature examines the infectious disease implications of higher population levels.[13] The conclusion of this literature is that higher birth rates (cohort sizes) are generally accompanied by increases in infectious disease, both among that cohort and population-wide. For example, Liu et al. (2014) show that China's 1979 one-child policy reduced the annual incidence of influenza. The proposed mechanisms for the Chinese influenza case are that "children have an increased susceptibility due to lower immunity" and that "increased viral shedding and longer infectious periods in children lead to more influenza among susceptible populations" (p.2). In other words, a larger number of children among the population will lead to higher rates of influenza contraction among the broader population both because children are more likely to contract influenza, and because the duration of illness (and its contagion) is longer in children. Other studies have reached similar conclusions, including Cummings et al. (2009). Siblings of these cohort members are also likely to be affected by changes in cohort size, and in the same direction. As a result, the difference-in-differences strategy we pursue for identification is likely to underestimate the effects of larger cohort sizes on health and socioeconomic outcomes.

Mortality at older ages, however, may have a different relationship to cohort size. A traditional health-based "selection effect" suggests that early insults, both pre- and post-natal, may result in increased life expectancy conditional on survival past childhood. For abortion-restricted cohorts, an enhanced disease environment early in life may result in surviving children who are stronger on average than would otherwise be expected, and the surviving cohort may then exhibit higher conditional life expectancies.

On the other hand, a "scarring effect" may emerge if, even conditional on survival through childhood, an enhanced disease environment in childhood or an elevated level of "unwantedness" endows children with biological tradeoffs that lead to shorter life expectancies. A large body of literature implies that adverse prenatal conditions, including malnutrition, disease environment, and stress, result in reduced life expectancy even conditional on survival past infancy (e.g., Barker 1992,

[13] See, *inter alia*, Behrman et al. (1980); Cummings et al. (2009); Ernst and Angst (1983); Finkenstädt and Grenfell (2000); Gao and Hethcote (2006); Geard et al. 2015; Liu et al. (2014); Manfredi and Williams (2004); Marziano et al. (2015); McDonald et al. (2012); Merler and Ajelli (2014); Williams and Manfredi (2004).

see McEniry 2013 for a literature review). These adaptations may lead to chronic conditions, including cardiovascular disease and type 2 diabetes, and reduced life expectancy. Thus, if larger cohort size or unwantedness are associated with poor prenatal or early life conditions, they may also be associated with lower longevity.

Finally, if childhood mortality is increased because of infectious disease burden, there may be a direct immunity effect on survivors who caught the infectious disease and are inoculated against this disease in later life. Thus, we might expect surviving cohorts to be less likely to die of infectious diseases at older ages than in a counterfactual childhood disease environment.

These patterns are apparent in other historical settings. For example, children born during the 1918 influenza epidemic (Almond 2006) or during the summer (Costa and Lahey 2005) appear to exhibit scarring behavior and reduced life expectancy at older ages. Klemp and Weisdorf (2012) measure scarring effects through an increased mortality risk at all ages for individuals born during the English famine of the late 1720s where the treatment is measured by famine intensity in the year of birth. Costa (2012) measures heterogeneous effects in Union Army Civil War prison survivors depending on the age at imprisonment; younger prisoners experienced higher later life mortality rates than expected (scarring) while older prisoners displayed lower mortality than would have otherwise been the case (selection or disease immunity). Measuring the long-term effects of the Finnish famine (1866-1868), Kannisto, Christensen, and Vaupel (1997) find short-run mortality spikes for exposed cohorts, but no effect on mortality rates after age 16.

VI. Empirical Evidence on the Health Impacts of Abortion Restrictions

In our sample of 19th century birth cohorts, we find that abortion restricting laws appear to have had negative mortality and disease consequences early in life, and that these effects may have reversed direction at older ages. We first describe the empirical approach and results for measuring overall mortality and survivorship effects, which relies on consecutive Census enumerations. We then use available cause-of-death data to document heightened infectious disease mortality among abortion-restricted cohorts early in life, especially from diseases transmitted by humans, counteracted by reduced mortality in later decades from these same causes.

a. Evidence on Mortality

We first ask whether cohorts affected by abortion restrictions exhibited differential mortality relative to unaffected cohorts. To measure mortality --- and its inverse, longevity or survivorship --- we compiled data from the 1850 through 1980 decennial Censuses on the number of living individuals

born in a given state and decade (Ruggles et al. 2015), applying the same race and sample restrictions as in Section IV.[14] Note that all survival outcomes are conditional on being alive in the 1850 or later censuses.

To understand these survival patterns, we calculate decade-to-decade survival rates -- the number of people born in a given state and year who survived to the time of the census as a share of the number who were observed in the previous census. For example, we calculate the share of a cohort observed during its first decade (when members are between 0 and 9), who remain alive in the cohort's second decade (ages 10 to 19), and so on.[15]

The outcome variable (S_{Dys}) is the forward-looking decade *D* survival rate of each cohort born in year *y* in state *s*, estimated separately by gender:

$$S_{Dys} = \%\ survive\ to\ decade\ D+10|\ birth\ year\ y; birth\ state\ s; survival\ to\ D$$
$$= \frac{count\ in\ D+10\ |\ birth\ year\ y; birth\ state\ s}{count\ in\ D\ |\ birth\ year\ y; birth\ state\ s}$$

In Figures 3a-c we plot coefficients from a simple OLS regressions with $\ln(S_{Dys})$ as the outcome variable, estimating a separate regression for each decade of a cohort's lifespan. As in Section IV, the critical explanatory variable is *HAVELAW,* and estimates also include birth state and sex by birth year fixed effects. All observations are weighted by the number of people in the decade D cell.[16] We report results for all persons, and then for men and women separately (with birth state and birth year fixed effects only in sex-specific regressions).

[14] We use IPUMS full count samples when possible, and scale up representative samples when full count samples are unavailable.

[15] Heaping, that is, people rounding their ages to the nearest 0 or 5, should only be a problem for this exercise if it changes by age. To test that possibility, we recreate Figure 3 using data collapsed at the decade of birth rather than year of birth level. Our results (available from authors) are very similar with slightly larger standard errors, but no changes in significance at the 5% levels. Because of problems with data quality, several assumptions had to be made. To distinguish between missing data and true zeroes, the following heuristic was used: if the *Count_in_Ds* is coded as missing and all *Count_in_(D+N)s* were also missing, and *Count_in_D-10* is not missing, then *Count_in_Ds* is coded as zero rather than missing. This recoding only affects cells of surviving to 80s in our sample. In some cases, the *Count_in_D+10* exceeded *Count_in_D*. These cases would result in S_{Dys} greater than 1. The majority of these cases are in state*year cells with small initial counts and are discounted by weighting by cell size in time D. Additionally, taking the logarithm of this variable additionally decreases the effect of these outliers. In regressions with the logarithm of S_{Dys} as the dependent variable, observations that have dropped to zero survivors are replaced with the most negative value for $\ln(S_{Dys})$. This change only affects ages 70-79 and higher, which are not shown in Figure 3.

[16] As before, we cannot perform these exercises on ages 80-89 because of top-coding. In these figures we also do not show results for ages 70-79 because a combination of small cell sizes and cell sizes of 0 (whose results are sensitive to our choice of substitution for ln(0)) greatly increase the standard errors. Appendix Table A2 shows the point estimates corresponding to Figure 3, including results for ages 70-79 with all ln(0) replaced by the most negative number for ln(Y).

The results indicate that having an anti-abortion law decreases survival into the second and third decades of life for both men and women, with an additional small reduction in probability of surviving to the next decade for men in their 40s. That is, having an anti-abortion law in the state in which you were born at the time of birth decreases survivorship to the next decade among those who were ages 0 to 9 (by 3.4 percent) and for those aged 10 to 19 (by 2.5 percent). There is little difference in these estimates between men and women. For men in their early adult years, this decrease in survival probability continues, ranging from 2.1 to 2.6 percent before age 50. Women, on the other hand, exhibit no significant effect on survivorship in these decades.[17]

At older ages, the effect of anti-abortion laws on survival changes sign. The coefficient on having an abortion law at the time of birth in a regression for survival beyond the 50s decade is positive, but small and insignificant. The same coefficient for survival beyond the 60s is positive and significant (an increase of 3 percent). Beyond age 70, our analysis is limited by an increasing amount of missing data. These results, contained in Appendix Table A2, are not statistically significant.

Taken together, these results suggest that anti-abortion laws increase mortality early in life, but, consistent with both survival-of-the-fittest and enhanced immunity hypotheses, those born in states with anti-abortion laws who survive middle age are less likely to die in their 60s and beyond than those born in states without such laws.

A back-of-the-envelope calculation suggests that heightened mortality for larger cohorts fully offsets the boost in cohort size resulting from abortion restrictions before the end of the cohort's life span. For example, assuming a 10 percent increase in cohort size among abortion restricted cohorts (the midpoint of Lahey's 2014 results), abortion-restricted cohorts experience enough heightened mortality to offset this cohort size increase and return to the average size for non-abortion restricted cohorts by age 30-39.

b. Evidence on Cause of Death

We turn to cause of death evidence to help explain why abortion-induced larger cohorts experienced higher death rates at younger ages but lower death rates at older ages. We use fragments of mortality data from the 19th century and Vital Statistics data from the 20th century to answer these questions.[18] Our hypothesis is that heightened cohort size may have increased the spread of infectious

[17] Please see section VII for discussion of robustness checks for two way fixed effects with staggered treatment timing.

[18] Our analysis is constrained by the availability of mortality data at the level of subclassification required to discern abortion law treatment. (Data by state of birth or state of residence, age, and race.)

disease.[19] If so, cohorts born under restrictive abortion laws may have been more likely to die of these diseases and less likely to die of other causes. Later in life, having survived a childhood with more prevalent infectious disease, these cohorts may have proven less susceptible to death from infectious disease. Alternatively, a "survival of the fittest" mechanism would imply that these later cohorts experienced reduced mortality from multiple causes, including chronic disease and not just infectious disease.

We use two separate sources on cause of death by race, gender, year, and state of birth/residence to estimate the effect of abortion restrictions. We require all four of these subclassifications in order to define treatment at the level of state-birthyear, cohort, race, and gender akin to the previous analysis in this paper and in Lahey (2014a, 2014b). These restrictions substantially limit the datasets that are appropriate for our use. The earliest sources usable for this purpose are the available fragments of the U.S. Censuses of Mortality for 1850, 1860, 1870, and 1880, where both state of birth and state of residence are recorded. The next available sources, chronologically, are Vital Statistics of the United States (VSUS) reports from 1939 through 1947, where we can observe state of residence along with the remaining data requirements.[20,21] Putting these sources together, we estimate the effect of abortion restrictions on affected cohorts at ages 0 through 39 (using the Mortality Census) and ages 50 through end of life (using the Vital Statistics data).

In the mortality schedules, enumerators individually report all deaths occurring in the year prior to the Census date --- for example, June 1, 1869 through May 31, 1870 for the 1870 Mortality Census. In doing so, the enumerators capture age at death (and, therefore, assumed year of birth), birthplace, and cause of death, in addition to age, sex, race, and place of death.[22] Thus, each mortality observation can be tied to an abortion law treatment value based on year and state of birth. These data allow us to estimate the effect of abortion law changes on causes through age 39.[23] Our data, courtesy of the Center

[19] Alternative explanations include a decrease in wantedness among abortion-restricted cohorts resulting in a rise in deaths from child abuse or neglect (Bitler and Zavodny 2002). We find no evidence of this, as detailed below.
[20] We do not incorporate data prior to 1939 because 1939 is the first year for which mortality tables are reported by state, age, cause of death, and race; prior to 1939, at least one of those subclassifications is missing from the public VSUS tabulations.
[21] In the Census of Mortality results in Figure 4, we detect differential mortality among children aged 0 to 9 in 1860, 1870, and 1880. These individuals were born between 1851 through 1880. In the survivorship analysis of Figure 3, we find these individuals are more likely to survive their 60s, though results for this age group may not be robust. To the extent that this effect is real, we should expect to see lower death rates from some causes up through 1949 (1880+69).
[22] These data also frequently contain occupation information.
[23] We have restricted the analysis sample throughout to exclude states with laws passed prior to 1840, so there is no law variation for cohorts born before 1840 and the last year of mortality observation is 1880, leaving us without a method for estimating the mortality effects of abortion laws for those older than age 39 using the mortality census data. Vital Statistics data are available beginning in 1939. Only one state added an abortion law after 1883, and we

for Population Economics at the University of Chicago, represent a fragment of the full mortality census in these years. Because these data are only a fragment, we do not estimate effects on death rates *per se*.

The vital statistics data are available for years 1939 and following. For budget reasons, we transcribed data from every other year in this window: 1939, 1941, 1943, 1945, and 1947. The data structure is similar to that for the mortality census: we observe death counts by cause of death, age, state of residence, and race. Because we do not have accurate measures of population to use as denominators, we again do not estimate death rates *per se,* but, rather, cause of death conditional on death.

In the mortality census schedules, we use cause of death strings to assign an International Classification of Disease (ICD) code to causes of death, and we map Vital Statistics cause of death categories to the same taxonomy.[24] We then collapse these ICD codes into one of four categories: Accidental and Acute Causes; Infectious Disease; Chronic Disease; and All Other Causes. We also perform additional analyses to measure any effects on child abuse and neglect, and on infant and maternal mortality. Cause of death ICD coding in the mortality schedules was performed by graduate research assistants, and full details are contained in Appendix B. The underlying sample has numerous misspellings and illegible causes; indeed, there are 97,094 unique causes of death across 1,028,237 observations in these files. We successfully assigned a discernible cause of death to 96.6 percent of observations, and we assigned uncoded data to the "All Other Causes" category.[25] In the Vital Statistics Data, only the most prominent causes of death are separately reported; the "All Other Causes" category includes a large number of unreported causes, likely including some infectious disease categories. Data quality is worse for the 1850 enumeration than for subsequent enumerations, and we drop the 1850 mortality census data from our baseline results. For results including the 1850 mortality census, the effects documented below are attenuated, but remain statistically significant.

To estimate the effects of abortion laws on causes of death, we use a multinomial logit model, estimating the probability of death from each possible cause (in discrete choice parlance, a death cause

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therefore do not have robust identification for mortality effects for individuals in their 40s. (In the 1939 Vital Statistics report, those in their 40s would have born between 1889 and 1899.)

[24] The first ICD guideline, known as the International List of Causes of Death, was in use in Europe by the 1850s. Widespread adoption in the United States did not occur until the 1890s. Nevertheless, the structure of disease classification allows us to organize deaths from 1850 to 1880 according to a set of disease categories internationally recognized at that time.

[25] For these reasons, we are biased against finding an effect of abortion restrictions on death from infectious disease. Most uncoded causes are string orphans or near-orphans (representing one or few individuals in the sample). There are 34,774 uncoded causes after the coding procedure, of which 26,977 are unique strings.

"choice") among all available causes ("choices") for each individual in the mortality census and Vital Statistics samples, as a function of the presence of an abortion-restricting law in the year of birth. For the Vital Statistics regressions, we weight each state-level observation by the number of deaths in that state to mimic the individual-level analysis from the mortality schedules. We refer to this structure as a competing risks model of death because it does not predict death itself but, rather, the probability of dying from a particular cause, conditional on death.[26]

Using the multinomial logit framework, the probability that the cause of death for individual i, D_i, is category j can be expressed as:

$$\Pr(D_i = j) = \frac{e^{\beta_j X_i}}{1 + \sum_{k=1}^{K-1} e^{\beta_k X_i}}$$

where K is the number of cause-of-death categories and X includes a binary indicator for the presence of an abortion-restricting law in the state and year of birth, state by gender fixed effects, and year of birth by mortality census year (or Vital Statistics report year) fixed effects. The estimates of β are derived from maximum likelihood estimation.

How would heightened infectious disease deaths appear in a competing risk model? If more children die of infectious disease in larger cohorts, the coefficient on abortion-restricting laws for infectious disease outcomes will be positive. Because the model is conditional on death, mechanically, coefficients on competing causes will be negative because the relative share of deaths attributable to those causes will fall.

The marginal effects of state abortion laws on the probability of each cause of death category are displayed in Figure 4.[27] Each of the four panels contains the point estimate for a coefficient on the presence of an abortion-restricting law in the state of birth in the year of birth, separately by age group: 0 to 9 year olds at time of death, 10 to 19 year olds, etc. Again, we are unable to estimate the mortality implications between ages 40 and 49 using this method because of data limitations combined with limited abortion law variation for these ages. To mimic sample restrictions in the selection analysis, we restrict the sample to individuals born in 1840 and years following.

At young ages, deceased children in abortion-restricted cohorts were far more likely to have succumbed to infectious disease compared to children in non-abortion-restricted cohorts. The point estimate in Figure 4, Infectious Disease, for ages 0 to 9, 6.68 percentage points, implies that the share

[26] These data pre-date establishment of the Death Registration Area and formal mortality statistics for the entire U.S.
[27] Note that because these are marginal effects, there is no comparison to a single omitted category as would be the case for a standard multinomial logit.

of mortality among children less than 10 attributable to infectious disease rose from a mean of 53 percent to approximately 60 percent with an abortion-restricting law in place. These deaths came at the cost of deaths in two of the three remaining categories. Accident and acute causes and chronic disease causes both declined significantly as a share of all deaths while other causes of death (including those unclassified by our algorithms) were unchanged as a share of the total. Between the ages of 10 and 39, the mortality causes for abortion-restricted cohorts show no substantial shift toward infectious disease; the share of deaths attributable to infectious disease in these age ranges fell by 0.5 and 2.3 percent, respectively. Only the point estimate for 20 to 29 year-olds is statistically significant. If we perform the same analysis using state of residence rather than state of birth to assign treatment, the coefficients for 10 to 19 and 20 to 29 year-olds are both insignificantly different from zero while the coefficient on 0 to 9 year-olds remains positive and highly statistically significant (point estimate: 6.91 percentage points).

At older ages, the changes in cause of death are less clear. There is some evidence of a decline in susceptibility to infectious disease among abortion-restricted cohorts, consistent with theories of long-term immunity from early disease exposure. These results, again contained in Figure 4, have the same structure as the cause of death results for younger ages. The estimates show an enhanced probability of death from chronic diseases for these cohorts, consistent with scarring effects from disease burden at younger ages (McEniry 2013) and a weakly lower probability of death from infectious disease. The results are strongest for deaths in the 60 to 69 age range; pooling cohorts aged 50 to 89 (not shown) produces negative and statistically significant coefficients for accidents and acute causes and for infectious disease and produces positive and statistically significant coefficients for deaths from chronic disease, perhaps due to heightened heart disease risk (McEniry 2013). But the magnitude of these changes is relatively small; point estimates for the 60 to 69 age group (1.05 percentage point decrease in the probability of death from infectious disease) is a 13.3 percent decrease from baseline. Pooling all cohorts aged 50 to 89, the percent reduction in the probability of infectious disease deaths falls to a 1.0 percent decrease from baseline.

We also examined the potential rise in death from chronic diseases among older age cohorts by looking at cardiac diseases directly. One hypothesis is that heightened exposure to infectious disease in childhood resulted in higher levels of cardiac disease at older ages as a result of permanent cardiac scarring (McEniry 2013). Splitting out heart disease (not including high blood pressure, rheumatism, or diseases of the blood vessels) from the remaining chronic disease categories, we find a positive and statistically significant increase in deaths from heart disease

among both the 50-59 (significant at 10% level) and 60-69 age groups (significant at 1% level) that were exposed to abortion restrictions. Those increases were substantial --- up to 10 percent of the baseline for the 60-69 age group. Visual results are contained in Figure A4 and coding details in Appendix B.

c. Isolating Deaths from Specific Causes

The infectious disease results at young ages in Figure 4 are driven by deaths from diseases passed person-to-person, consistent with enhanced cohort size as a mechanism. To document this, we divided infectious disease deaths into two categories: infectious diseases with person-to-person transmission (e.g., measles) and infectious diseases that spread in other ways, such as waterborne diseases (e.g., cholera). Heightened infectious disease deaths from waterborne diseases might indicate either reduced attention to hygiene among families with additional children or negative selection among born children (for example, if lower income families are more susceptible to cholera). On the other hand, if the heightened deaths are due to communicable diseases passed person-to-person, cohort size itself is more likely to blame than selection effects. Figure 5 shows that for the youngest age group, all of the increased infectious disease death probability arises from diseases that transfer between individuals; at older ages, the effects are not as clearly driven by diseases with person-to-person transmission.

We also assess the impact of abortion-restricting laws on deaths from child abuse and neglect. Perhaps abortion-restricting laws increased unwantedness and raised the probability of child death from abuse or neglect. To test this hypothesis, we generate a new cause of death category for abuse and neglect causes, re-categorizing deaths from malnutrition, exposure, burns, falls, drowning, suffocation, poisoning, suicide, and any cause of death string containing "abuse" or "neglect" as "abuse and neglect" causes.[28] We then re-estimate the competing cause model for children aged 0 to 9. The results from this exercise, contained in Figure 6, indicate no significant difference in the probability of death from these causes in abortion-restricted cohorts relative to other cohorts, and the point estimate

[28] This is a relatively narrow definition of abuse and neglect. A broader definition includes all causes included in the narrow abuse and neglect categorization plus cerebral hemorrhage, rape, any cause of death containing "fracture" or "injury", deaths from fighting, deaths from firearms, and all other violent causes of death not already included (ICD-1 codes 175-191). In all cases, coefficients on the presence of an anti-abortion law in the year of birth are negative and only marginally significant, perhaps because of the competing risks structure and enhanced mortality from infectious disease.

is negative. A similar exercise for infant mortality among children aged 0 and 1 (not shown) also shows no significant effect.[29]

As a final view of the mortality consequences of abortion restrictions, we test whether abortion law restrictions are associated with heightened maternal mortality among mothers affected by the law when they died. We estimate a competing risk model of mortality among women aged 16 to 39 akin to that depicted in Figure 4 and using the presence of an abortion law in the year of mortality observation as the treatment variable.[30] In this exercise, maternal mortality is a distinct categorization of cause of death, and we do not differentiate by age within the fertile age band.[31] Again, all results are conditional on death and must be interpreted as such. These results, contained in Figure 7, suggest maternal mortality was not a more (or less) likely cause of death among women of childbearing age in states with an abortion-restricting law in place between 1850 and 1880. On the other hand, we estimate a more than 7 percent increase in the probability of death from infectious disease among women in this age range, consistent with a richer disease environment in years when abortion-restricting laws were in place.[32]

VII. Robustness Checks and Placebo Tests

In this section, we provide robustness checks for what we consider to be our main results, the effect of being born in a state with an abortion law on the change in probability of surviving to the next census (Figure 3), particularly for the age 0-9 cohort.

One concern is that our treatment effect could be picking up differences between states with and without anti-abortion laws instead of the effects of the laws themselves. Lahey (2014a) provides extensive robustness checks for the first stage, the effect of these laws on cohort size, and shows that that law passage is not related to the medical movement, immigration patterns, urbanization, or literacy. That paper also demonstrates that changes in cohort size are not predicted by other "morality" laws such as obscene singing, sodomy, indecency, or exhibition laws. In addition, the effect of anti-abortion laws on cohort size in this time period is robust to the inclusion of birth control legislation (e.g. Myers

[29] We begin with the structure in Figure 4, and then re-code infant mortality as a separate cause of death that includes infant mortality, premature birth, congenital hydrocephalus, other congenital defects, injury at birth, aelectasis, want of breast milk, teething, thrush, stillborn, and malformation.
[30] The fertile age range does not end at age 39, but our ability to measure mortality effects for women between 40 and 49 is limited by data availability.
[31] The maternal mortality category includes deaths from puerperal septicaemia, puerperal septic intoxication, puerperal pyaemia, puerperal fever, abortion (a term that includes spontaneous miscarriages), miscarriage, puerperal mania, puerperal convulsions, placenta praevia, and other accidents of pregnancy and childbirth.
[32] The point estimate corresponding to Figure 7 infectious disease is 4.3 percentage points when the average share of deaths from infectious disease among women in this age range is 57.7 percent (4.3/57.7=7.4 percent increase).

2017). Finally, there is no evidence of reverse causality as previous child/woman ratios, the measure of cohort size in that paper, do not predict law passage.

Even so, it may be the case that while changes in social mores do not affect cohort size, they could affect cohort-level mortality. Thus, we repeat our falsification checks with different morality-based legislation. We rerun the cohort-level mortality analyses using legislation prohibiting exhibition, indecency, obscene singing, and sodomy in place of anti-abortion legislation, shown in Figures 8a-d. These coefficient estimates are mostly insignificant, including coefficients for 0-9, and when they are significant, such as the positive result on 0-9 for anti-sodomy laws, they do not follow the same patterns and trends by age as that the anti-abortion laws shown in Figure 3. Similarly, the effects of anti-abortion laws on mortality are unchanged when concurrently controlling for birth control laws, as in Figure 8e, and birth control laws themselves do not seem to affect mortality, at least when anti-abortion laws are controlled for.

There may also be concerns that our results are driven by major events like the 1918 flu pandemic; however, removing the 1920 census entirely from our sample does not affect the estimates for the 0-9 cohort and does not change the pattern of our results by age, although the results for survival to the next census for ages 10-19 and 20-29 become significant only at the 10% level, as might be expected with the loss in number of observations. Similarly, the Civil War is unlikely to drive any of our results; our results are largely unchanged when states with Civil War deaths exceeding 2% of their 1860 population are removed.

Unfortunately, data limitations prohibit us from showing a full event study with pre-period information. Individual census data are not available and tabulated census data (as in Carter et al. 2006) do not have the age categorizations that we would need to create mortality tables. Instead, following Gruber and Hungerman (2008) and Lahey (2014a), we create a placebo law by pretending that each law was passed 10 years prior to its actual passage and include both the actual law and this "fake law" in our regression. While this test is imperfect, it is reassuring that the pattern we found for the actual anti-abortion laws still holds, especially at younger ages, while the fake law tends to be small, closer to zero, and with no discernible pattern, shown in Figure 8f. It remains possible that the positive results on survival on ages 60-69 that we were find in Figures 3a-c are spurious.[33]

[33] Finally, we made a number of choices about data year cutoffs. As noted throughout the paper, our results are robust to alternate choices; our results are little changed if we keep states that have laws adopted before 1840 as an additional control group, if we include states that were incorporated prior to 1890 instead of prior to 1880, and if we drop people who were born before 1840.

Goodman-Bacon (2021) gives guidance on identification in a two-way fixed effects framework with staggered treatment timing, as is the case here. We use the bacondecomp.ado file in Stata to explore the sources of variation in these two-way fixed effects models. Some changes must be made to fit the programming constraints of the BaconDecomp model. We round all fractional law variables to the nearest 0 or 1, use average state weights rather than state*year weights, and remove Nevada as a state of birth since bacondecomp needs a strongly balanced panel. For the ages 0-9 regression, we drop two states (Alabama and Maine) that are always treated for this younger age group. (The bacondecomp point results are very similar with them included.) For all other age groups, late versus early adopters are 100 percent of the total estimate in all regressions. Appendix Figure A5 provides graphical output for the focus of our main results, the probability of surviving to the next decade for age group 0-9, separately by sex. Here the average decrease in survivorship is slightly larger than 4% compared to 3.4% with the main results.[34] Appendix Figure A6 plots the overall difference in difference estimates for survival outcomes for each age group separately by sex. For men, there is a decline in survivorship for the 20-29 age group not found in the main results in Figure 3, but also not ruled out given its standard errors. For women, there is a smaller decline in survivorship at the 40-49 age group. Both sexes show a decline in survivorship at age 60-69, further bolstering the idea that the positive coefficient estimates for the age 60-69 cohort in the main results are spurious.

We provide an additional check for identification in a two-way fixed effects model with staggered treatment timing based on de Chaisemartin and D'Haultfoeuille (2020) and the associated did_multiplegt.ado Stata command. Here we are able to weight by population in the previous period, as we do for the main regressions in Figure 3. Figure A7 provides placebo tests of the parallel trends assumption (periods -2 through -5 are placebos), which suggest a relatively flat pre-period and possibly a slight negative trend for the female sample, followed by a comparatively steep decline upon treatment. Figure A8 again shows the average effects by age group and separately by sex. The pattern for men shows a similar increase and flattening to that of Figure 3 but the point estimates change from negative to positive around age 20-29. The point estimates for women show less of a striking pattern and also turn positive earlier. For both sexes, the estimated negative effect on probability of survival for age group 0-9 is larger than in Figure 3, around 6%, and still significantly different from zero.

VIII. Conclusion

[34] Plots for later age groups are available on request.

Our results provide important nuance to existing evidence on abortion selection at birth and new evidence on the lifecycle mortality effects of large birth cohorts. In contrast to 20th century abortion-restricted US cohorts, marginal children induced by 19th century abortion restrictions were born into circumstances comparable, or perhaps somewhat more favorable, than cohorts born in state-years without these laws in place. That is, we find no evidence of negative selection into birth in the 19th century.

Minimal selection at birth allows us to investigate the impact of larger cohorts on population health. We find larger cohorts were associated with increased mortality at younger ages, and these results are robust to a number of specification checks. Digging deeper into the increased mortality results, infectious diseases appear to have been heightened among these cohorts, resulting in a natural check on population growth.

Our focus on 19th century cohorts allows us to measure the longer-term implications of large cohorts for mortality. At older ages, having survived infectious disease waves in their youth, abortion-restricted cohorts appear to benefit from disease immunity or positive selection, as evidenced by decreased deaths from infectious diseases. But these same cohorts also demonstrate evidence of long-term scarring from early exposure, marked by increased deaths from chronic diseases. Our results suggest a nuanced effect of abortion restriction on the welfare of affected cohorts.

References

Almond, Douglas. 2006. "Is the 1918 Influenza Pandemic Over? Long-term Effects of *In Utero* Influenza Exposure in the Post-1940 U.S. Population." *Journal of Political Economy* 114(4): 672-712.

Ananat, Elizabeth Oltmans, Jonathan Gruber, Philip B. Levine, and Doug Staiger. 2009. "Abortion and selection." *Review of Economics and Statistics* 91: 124-36.

Ananat, Elizabeth Oltmans, and Daniel M. Hungerman. 2012. "The Power of the Pill for the Next Generation: Oral Contraception's Effects on Fertility, Abortion, and Maternal and Child Characteristics." *Review of Economics and Statistics* 94(1): 37-51.

Antón, José-Ignacio, Zuleika Ferre, and Patricia Triunfo. 2018. "The Impact of the Legalisation of Abortion on Birth Outcomes in Uruguay." *Health Economics* 27: 1103-19.

Bailey, Martha J. 2010. "Momma's Got the Pill": How Anthony Comstock and Griswold v. Connecticut Shaped US Childbearing." *American Economic Review* 100: 98-129.

———. 2012. "Reexamining the Impact of Family Planning Programs on US fertility: Evidence from the War on Poverty and the Early Years of Title X." *American Economic Journal: Applied Economics* 4: 62-97.

Barker, David J. P., ed. 1990. *Fetal and Infant Origins of Adult Disease: Papers*. London, UK: British Medical Journal.

Behrman, Jere Richard, Zdenek Hrubec, Paul Taubman, and Terence J. Wales. 1980. *Socioeconomic Success: A Study of the Effects of Genetic Endowments, Family Environment, and Schooling*. New York, NY: North-Holland Pub. Co.

Bitler, Marianne and Madeline Zavodny. 2002. "Did Abortion Legalization Reduce the Number of Unwanted Children? Evidence from Adoptions." *Perspectives on Sexual and Reproductive Health* 34: 25-33.

Callaway, Brantly and Pedro H.C. Sant'Anna. 2021. Difference-in-differences with multiple time periods. *Journal of Econometrics* 225(2): 200–30.

Carter, Susan B, Scott S. Gartner, Michael R. Haines, Alan L. Olmstead, Richard Sutch and Gavin Wright. 2006. *The Historical Statistics of the United States*.

Charles, Kerwin Kofi, and Melvin Stephens Jr. 2006. "Abortion Legalization and Adolescent Substance Use." *The Journal of Law & Economics* 49: 481-505.

Costa, Dora L. 2012. "Scarring and Mortality Selection Among Civil War POWs: A Long-term Mortality, Morbidity, and Socioeconomic Follow-Up." *Demography* 49: 1185-1206.

Costa, Dora L and Joanna N. Lahey. 2005. "Becoming Oldest-Old: Evidence from Historical US Data." *Genus*, 61: 125-6.

Cummings, Derek A. T., Sopon Iamsirithaworn, Justin T. Lessler, Aidan McDermott, Rungnapa Prasanthong, Ananda Nisalak, Richard G. Jarman, Donald S. Burke and Robert V. Gibbons. 2009. "The Impact of the Demographic Transition on Dengue in Thailand: Insights from a Statistical Analysis and Mathematical Modeling." *PLOS Medicine* 6(9): e1000139.

David, Paul A. and Warren C. Sanderson. 1986. "Rudimentary Contraceptive Methods and the American Transition to Marital Fertility Control, 1855-1915." In *Long-Term Factors in American Economic Growth*, edited by Stanley L. Engerman and Robert E. Gallman, 307-90 Chicago and London: University of Chicago Press.

de Chaisemartin, Clément and Xavier d'Haultfoeuille. 2021. "Two-Way Fixed Effects and Differences-in-Differences with Heterogeneous Treatment Effects: A Survey." *SSRN Working Paper* http://dx.doi.org/10.2139/ssrn.3980758.

-------. 2020. "Difference-in-Differences Estimators of Intertemporal Treatment Effects." *NBER Working Paper* 29873

Degler, Carl N. 1980. *At Odds: Women and the Family in America from the Revolution to the Present*. New York, NY: Oxford University Press.

Dellapenna, Joseph W. 2006. *Dispelling the Myths of Abortion History*. Durham, N.C.: Carolina Academic Press.

Dennett, Mary Ware. 1926. *Birth control laws, shall we keep them, change them or abolish them?* New York, NY: F. H. Hitchcock.

Donohue, John J., and Steven D. Levitt. 2001. "The Impact of Legalized Abortion on Crime." *Quarterly Journal of Economics* 116: 379-420.

Easterlin, Richard A. 1980. *Birth and Fortune: The Impact of Numbers on Personal Welfare*. New York, NY: Basic Books.

——— 1987. "Easterlin Hypothesis." In *The New Palgrave: A Dictionary of Economics*, edited by John Eatwell, Murray Milgate and Peter Newman. London, UK: Macmillan Press Ltd.

Ernst, Cécile, and Jules Angst. 1983. *Birth Order: Its Influence on Personality.* New York, NY: Springer-Verla.

Ernst, Edzard, 2002. "Herbal Medicinal Products During Pregnancy: Are they safe?" *BJOG: An International Journal of Obstetrics & Gynecology* 109: 227-35.

Finkenstädt, Bärbel F. and Bryan T. Grenfell. 2000. "Time Series Modelling of Childhood Diseases: A Dynamical Systems Approach." *Journal of the Royal Statistical Society: Series C (Applied Statistics)* 49: 187-205.

Gao, Linda and Herbert Hethcote. 2006. "Simulations of Rubella Vaccination Strategies in China." *Mathematical Biosciences* 202 371-85.

Geard, Nicholas, Kathryn Glass, James M. McCaw, Emma S. McBryde, Kevin B. Korb, Matt J. Keeling, and Jodie McVernon. 2015. "The Effects of Demographic Change on Disease Transmission and Vaccine Impact in a Household Structured Population." *Epidemics* 13: 56-64.

Goodman-Bacon, A. 2021. Difference-in-differences with variation in treatment timing. *Journal of Econometrics* 225(2): 254–77.

Gordon, Linda, 1976. *Woman's Body, Woman's Right: A Social History of Birth Control in America*. New York, NY: Penguin Books.

------ . 2002. *The Moral Property of Women: A History of Birth Control Politics in America*. Chicago, IL: University of Illinois Press.

Gruber, Jonathan, and Daniel M. Hungerman. 2008. "The church versus the mall: What happens when religion faces increased secular competition?" *The Quarterly Journal of Economics* 123: 831–62.

Gruber, Jonathan, Phillip B. Levine, and Douglas Staiger. 1999. "Abortion Legalization and Child Living Circumstances: Who Is the 'Marginal Child?" *Quarterly Journal of Economic*s 114: 263-92.

Kannisto, Väinö, Kaare Christensen, and James W. Vaupel. 1997. "No increased mortality in later life for cohorts bom during famine." *American Journal of Epidemiology* 145: 987-94.

King, C. R. 1992, "Abortion in Nineteenth Century America: A Conflict between Women and Their Physicians." *Womens Health Issues* 2: 32-9.

Klemp, Marc, and Jacob Weisdorf. 2012. "The lasting damage to mortality of early-life adversity: evidence from the English famine of the late 1720s." *European Review of Economic History* 16: 233-46.

Lader, Lawrence. 1966. *Abortion*. New York, NY: Bobbs-Merril Company.

Lahey, Joanna N. 2014a. "Birthing a Nation: The Effect of Fertility Control Access on the Nineteenth-Century Demographic Transition." *The Journal of Economic History* 74: 482-508.

———2014b. "The Effect of Anti-Abortion Legislation on Nineteenth Century Fertility." *Demography* 51: 939-48.

Levine, Phillip, Douglas Staiger, Thomas Kane, and David Zimmerman. 1999. "Roe v. Wade and American Fertility." *American Journal of Public Health* 89: 199-203.

Liu, Fengchen, Wayne T. A. Enanoria, Kathryn J. Ray, Megan P. Coffee, Aubree Gordon, Tomás J. Aragón, Guowei Yu, Benjamin J. Cowling and Travis C. Porco. 2014. "Effect of the One-Child Policy on Influenza Transmission in China: A Stochastic Transmission Model." *PLOS ONE* 9: e84961.

Macunovich, Diane J. 2000. "Relative Cohort Size: Source of a Unifying Theory of Global Fertility Transition?" *Population and Development Review* 26: 235-61.

——— , 2002. *Birth Quake: The Baby Boom and Its Aftershocks*. Chicago, IL: University of Chicago Press.
Madari, Hamta and Robert S. Jacobs. 2004. "An Analysis of Cytotoxic Botanical Formulations Used in the Traditional Medicine of Ancient Persia as Abortifacients." *Journal of Natural Products* 67: 1204-10.
Manfredi, Piero and John R. Williams. 2004. "Realistic Population Dynamics in Epidemiological Models: The Impact of Population Decline on the Dynamics of Childhood Infectious Diseases: Measles in Italy as an Example." *Mathematical Biosciences* 192: 153-75.
Marziano, Valentina, Piero Poletti, Giorgio Guzzetta, Marco Ajelli, Piero Manfredi and Sstefano Merler. 2015. "The Impact of Demographic Changes on the Epidemiology of Herpes Zoster: Spain as a Case Study." *Proceedings of the Royal Society B: Biological Sciences* 282: 20142509-201425.
McDonald, Scott A., Alies van Lier, Dietrich Plass and Mirjam E. E. Kretzschmar. 2012. "The Impact of Demographic Change on the Estimated Future Burden of Infectious Diseases: Examples from Hepatitis B and Seasonal Influenza in the Netherlands." *BMC Public Health* 12: 1046.
McEniry, M. 2013. "Early-life conditions and older adult health in low- and middle-income countries: a review." *Journal of Developmental Origins of Health and Disease* 4: 10-29.
McFarlane, Deborah R. and Kenneth J. Meier. 2001. *The Politics of Fertility Control*. New York, NY: Chatham House Publishers.
McLaren, Angus, 1990. *A History of Contraception*. Cambridge, MA: Basic Blackwell.
Merler, Stefano and Marco Ajelli. 2014. "Deciphering the Relative Weights of Demographic Transition and Vaccination in the Decrease of Measles Incidence in Italy." *Proceedings of the Royal Society B: Biological Sciences* 281: 20132676-2013267.
Mohr, James C. 1978. *Abortion in America: The origins and evolution of national policy, 1800-1900*. New York, NY: Oxford University Press.
Myers, Caitlin Knowles. 2017. "The Power of Abortion Policy: Reexamining the Effects of Young Women's Access to Reproductive Control." *Journal of Political Economy* 125: 2178-224.
Pabayo, R., A. Ehntholt, D. M. Cook, M. Reynolds, P. Muennig, and S. Y. Liu. 2020. "Laws restricting access to abortion services and infant mortality risk in the United States." *International Journal of Environmental Research and Public Health* 17(11).
Pepper, Gillian V. & Nettle, Daniel. 2017. "The behavioural constellation of deprivation: Causes and consequences." *Behavioral and Brain Sciences* 40: 1-66.
Polsky, S. 1970. "Legal Aspects of Abortion." *Seminars in Psychiatry* 2: 246–57.
Pop-Eleches, Cristian. 2006. "The Impact of an Abortion Ban on Socioeconomic Outcomes of Children: Evidence from Romania." *Journal of Political Economy* 114: 744-73.
Quay, Eugene. 1961. "Justifiable abortion – Medical and legal foundations." *The Georgetown Law Journal* 49: 395-538.
Reagan, Leslie J. 1991. "About to Meet Her Maker: Women, Doctors, Dying Declarations, and the State's Investigation of Abortion." *The Journal of American History* 78: 1240-64.
Ruggles, S., Alexander, J. T., Genadek, K., Goeken, R., Schroeder, M. B. and Sobek, M. 2015. "Integrated public use microdata series: Version 6.0 [Machine-readable database]." ed. U. o. Minnesota. Minneapolis, MN.
Sanderson, Warren C. 1979. "Quantitative Aspects of Marriage Fertility and Family Limitation in Nineteenth Century America: Another Application of the Coale Specifications." *Demography* 16: 339-58.
Smith-Rosenberg, Carroll. 1985. *Disorderly Conduct: Visions of Gender in Victorian America.* New York, NY: Oxford University Press.
Storer, Horatio R. 1860. *Criminal abortion in America*. Philadelphia, PA: J. P. Lippincott & Co.
Storer, Horatio R., and Franklin F. Heard. 1868. *Criminal abortion: Its nature, its evidence and its law*. Boston, MA: Little, Brown and Company.
Sun, Liyang and Sarah Abraham. 2021. "Estimating dynamic treatment effects in event studies with heterogeneous treatment effects." *Journal of Econometrics* 225(2): 175–99.
Tribe, Laurence H. 1990. *Abortion: The Clash of Absolutes*. New York, NY: W. W. Norton & Company.

Wanamaker, Marianne H. 2014. “Fertility and the Price of Children: Evidence from Slavery and Slave Emancipation.” *The Journal of Economic History* 74: 1045-71.

Williams, John R and Piero Manfredi. 2004. "Ageing Populations and Childhood Infections: The Potential Impact on Epidemic Patterns and Morbidity." *International Journal of Epidemiology* 33: 566-72.

FIGURE 1: ABORTION RULE

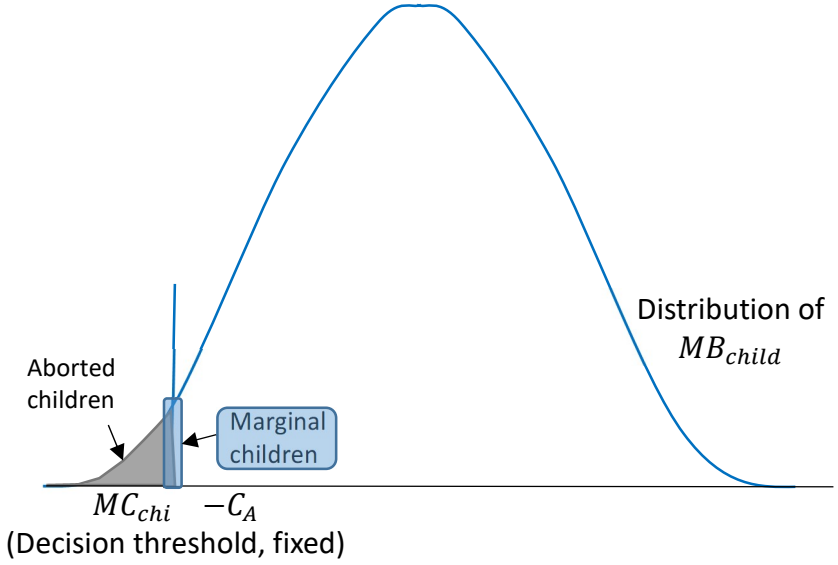
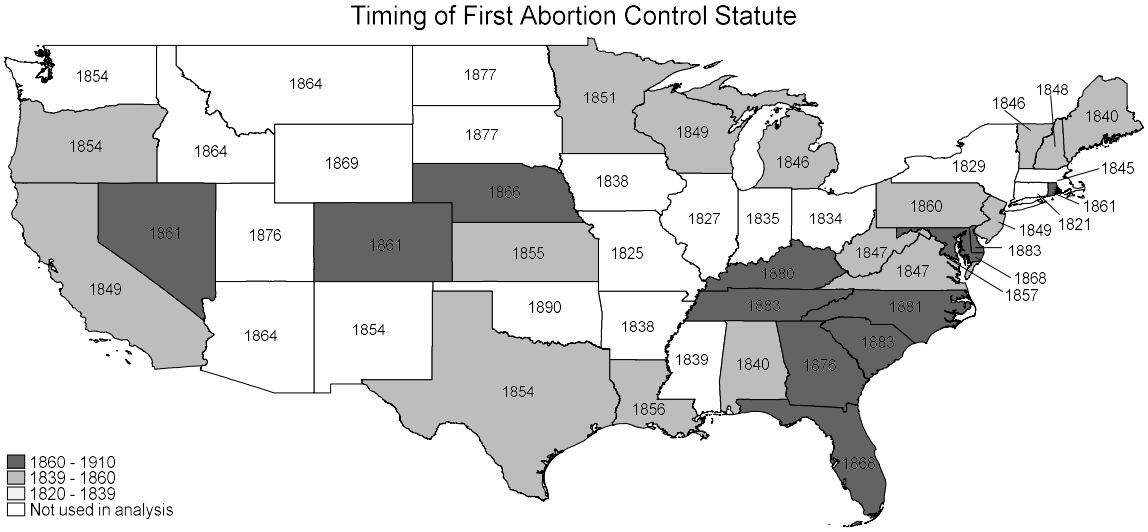


FIGURE 2: TIMING OF FIRST ABORTION CONTROL STATUTE



Note: Figure plots the first passage of an abortion law in each state. States without shading were excluded in the analysis because they had not been admitted to the Union in 1880 or because they had laws prior to 1840. Law dates are preceded by rulings in Kentucky (1879); Massachusetts (1812); and Pennsylvania (1846).

FIGURE 3: THE EFFECT OF HAVING AN ANTI-ABORTION LAW ON $ln(S_{DYS})$

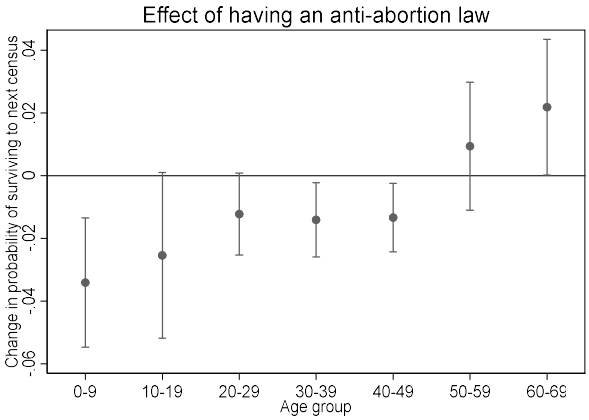


Figure 3a

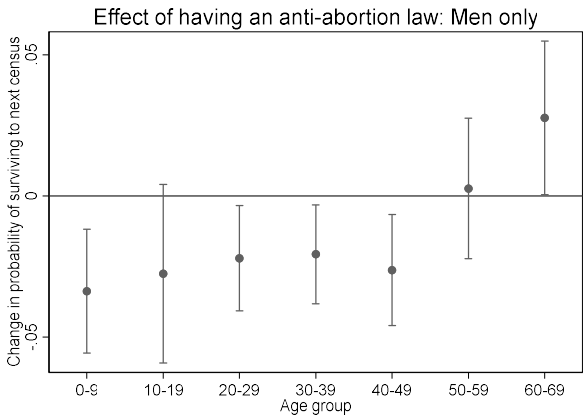


Figure 3b

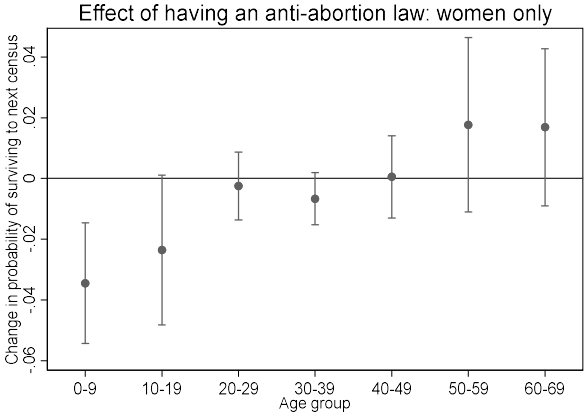
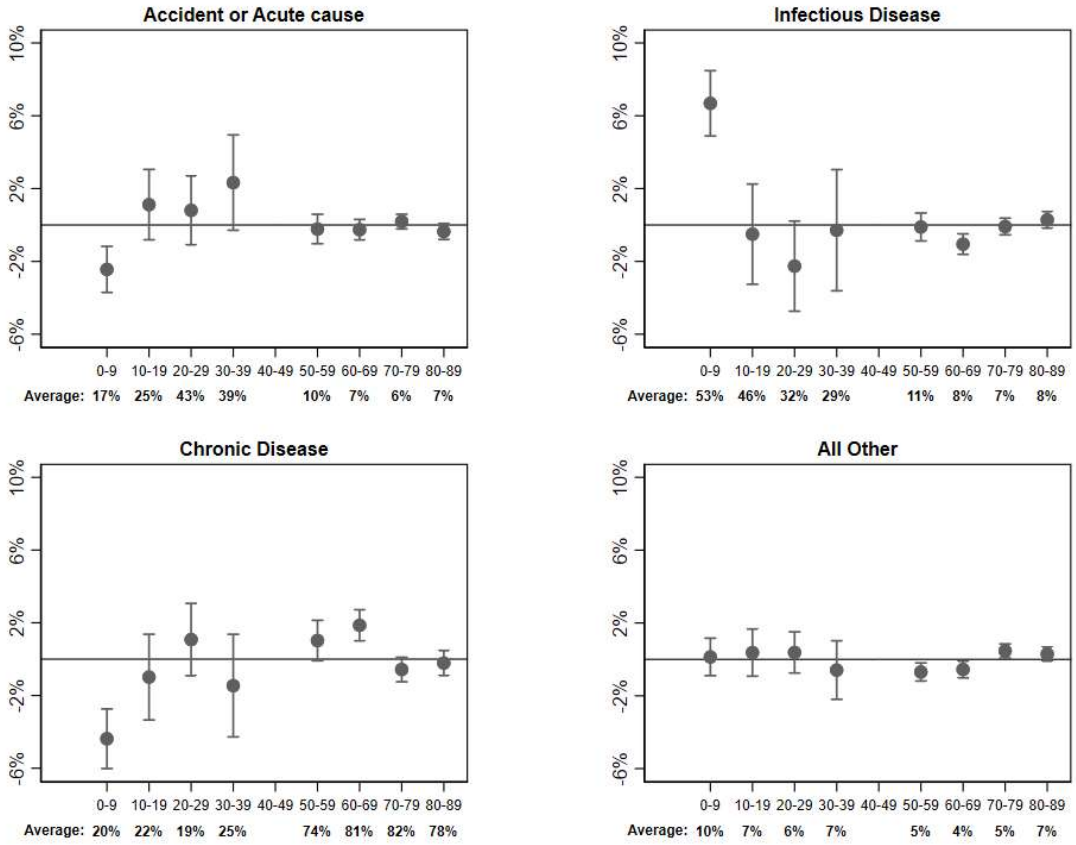


Figure 3c

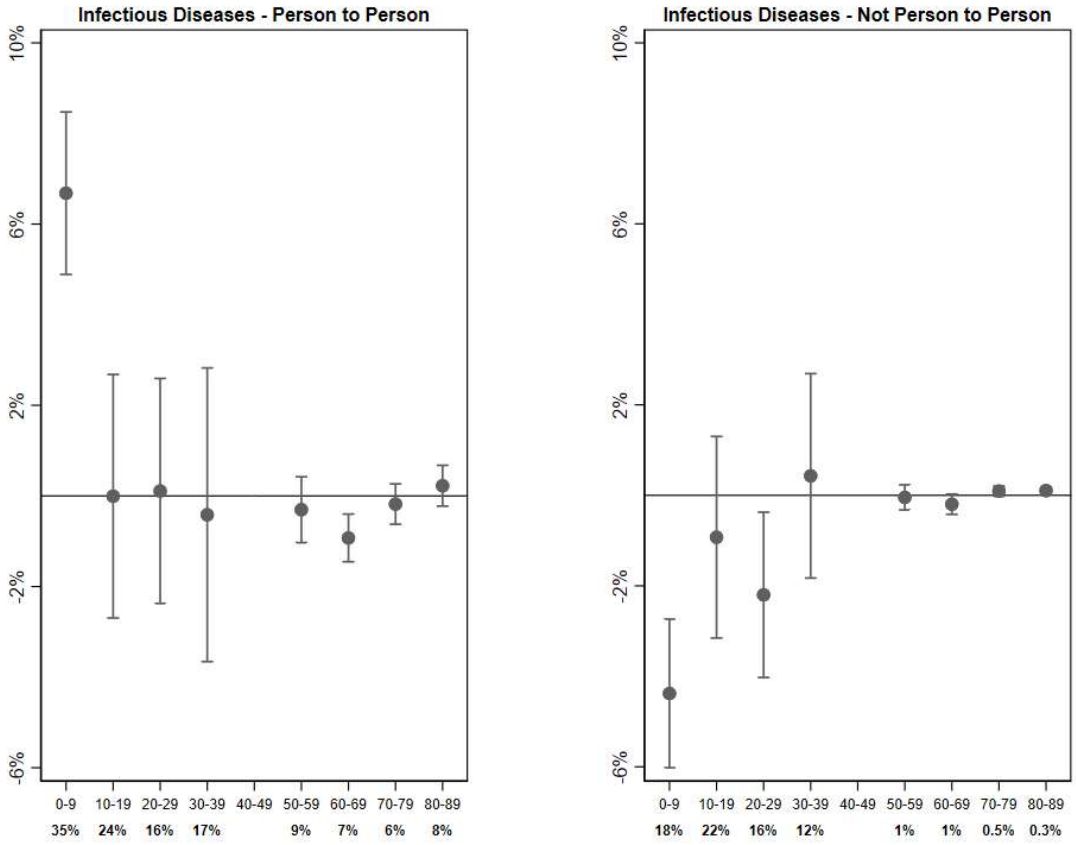
Note: Each dot represents the coefficient of a separate regression with ln(% survival from previous decade) as the Y variable. Lines represent 95% confidence intervals clustered by state of birth. Data come from the 1850-1950 censuses of population from IPUMS. Universe includes cohorts born prior to 1900. Sample sizes for all observations are 2,196 (0-9); 2,763 (10-19); 3,330 (20-29); 3,875 (30-39); 3,962 (40-49); 4,001 (50-59); 4,017 (60-69). Sample sizes for men only are 1,100 (0-9); 1,383 (10-19); 1,670 (20-29);1,942 (30-39);1,987 (40-49); 2,005 (50-59); 2,008 (60-69). Sample sizes for women only are 1,096 (0-9); 1,380 (10-19);1,660 (20-29);1,933 (30-39);1,975 (40-49);1,996 (50-59); 2,009 (60-69).

FIGURE 4: CAUSE OF DEATH RESULTS



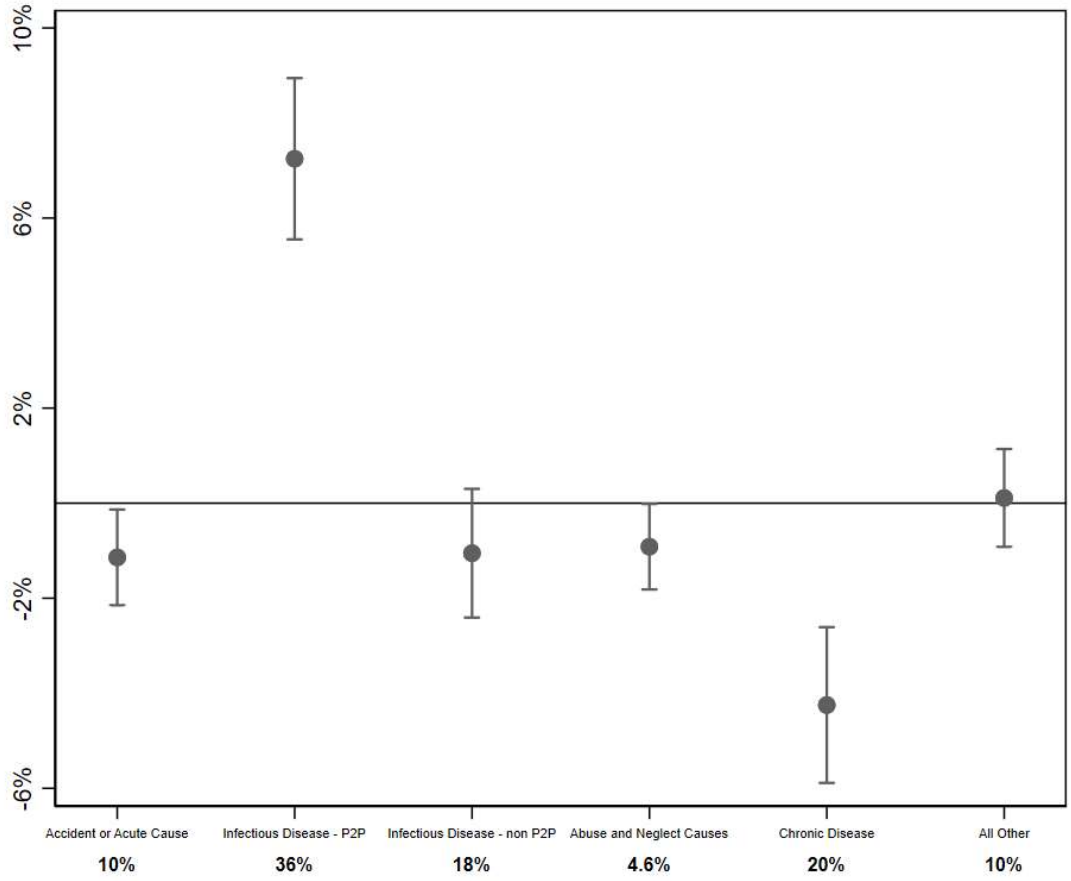
Note: Based on separate multinomial logit regressions (one for each age decade) using an 1860, 1870, and 1880 mortality census fragment courtesy of the University of Chicago CPE and Vital Statistics data from 1939, 1941, 1943, 1945, 1947. See text for details. Sample sizes are 168,812 (0-9); 26,425 (10-19); 25,441 (20-29); 12,824 (30-39); 640,179 (50-59); 936,052 (60-69); 1,046,905 (70-79); 616,558 (80-89) based on raw mortality entries for the 19th century (ages 0-9 through 30-39) and the number of total deaths represented by mortality statistics in remaining years (weighted N for ages 50-59 through 80-89). Ages 40-49 are missing because of lack of data coverage for those ages. Coefficients shown are the marginal effects for each cause of death category along with a 95% confidence interval. Average values represent the share of all deaths from each underlying cause within an age bracket.

FIGURE 5: CAUSE OF DEATH RESULTS, SEPARATING INFECTIOUS DISEASE INTO PERSON-TO-PERSON AND NON-PERSON-TO-PERSON TRANSMISSION



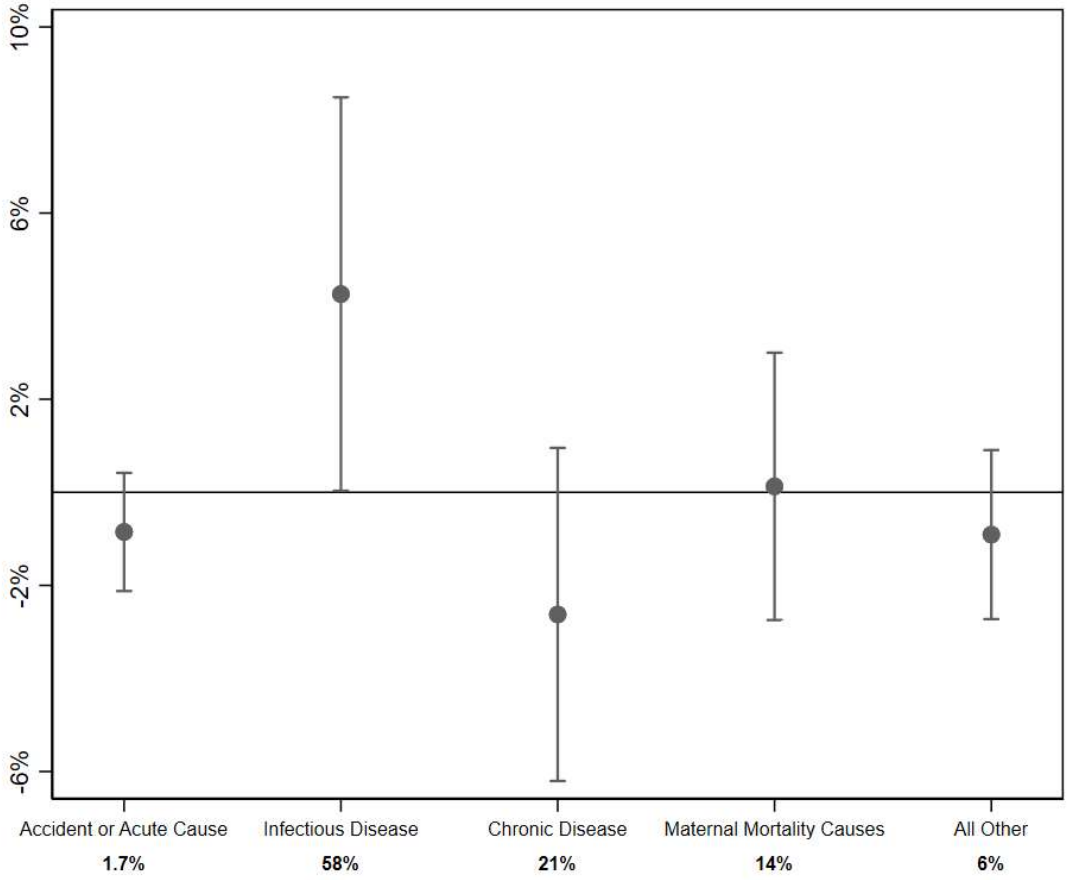
Note: See notes to Figure 4 for sample sizes and other details. Non person-to-person infectious diseases include cow-pox, typhus, enteric fever, cholera, dysentery, tetanus, malaria, rabies, tonsillitis, worms, phegmon, carbuncle, tabes mesenterica, phelgmasia alba dolens, syphilis, gonorrhea, erysipelas. Ages 40-49 are missing because of lack of data coverage for those ages. Coefficients shown are the marginal effects for each cause of death category along with a 95% confidence interval. Average values represent the share of all deaths from each underlying cause within an age bracket.

FIGURE 6: CHILD ABUSE CAUSE OF DEATH RESULTS



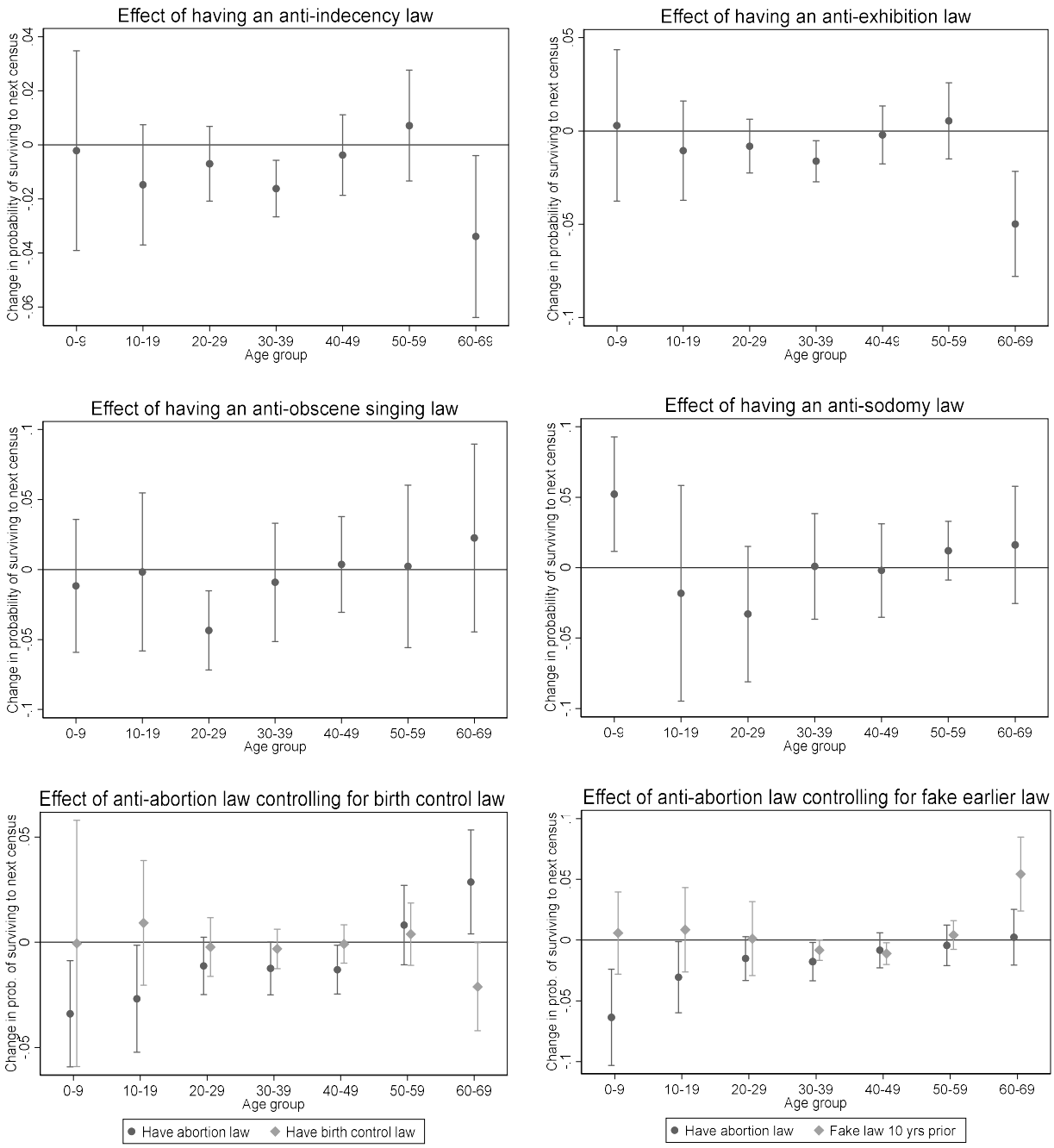
Note: See notes to Figure 4 and details in text. The universe is limited to children aged 0 to 9.

FIGURE 7: MATERNAL MORTALITY CAUSE OF DEATH RESULTS



Note: See notes to Figure 4 and details in text. The universe is limited to women age 16 to 39.

FIGURE 8: Falsification Exercises and Robustness Checks



Note: Each dot represents the coefficient of a separate regression with ln(% survival from previous decade) as the Y variable. Lines represent 95% confidence intervals clustered by state of birth. See figure 3 for additional notes. For Figures 8e and f, each paired grey dot and diamond represent coefficients from a single regression. Sample sizes are 2,196 (0-9); 2,763 (10-19); 3,330 (20-29); 3,875 (30-39); 3,962 (40-49); 4,001 (50-59); 4,017 (60-69) for Figures 8a-e. Sample sizes are 1,674 (0-9); 2,183 (10-19); 2,750 (20-29); 3,295 (30-39); 3,384 (40-49); 3,423 (50-59); 3,442 (60-69) for Figure 8f.

TABLE 1: SUMMARY STATISTICS

	Mean	Years Observed
EARLY CHILDHOOD OUTCOMES (Ages 0-9)		
Maternal Age	28.433	*1850-1880, 1900*
Paternal Age	33.549	*1850-1880, 1900*
Lives with both parents	0.835	*1850-1880, 1900*
Lives with single mother	0.058	*1850-1880, 1900*
Lives in group quarters	0.007	*1850-1880, 1900*
Paternal LFP	0.958	*1850-1880, 1900*
Parental Property >0	0.757	*1850-1870*
Urban Dummy	0.208	*1850-1880, 1900*
Immigrant Mother	0.183	*1850-1880, 1900*
Immigrant Father	0.213	*1850-1880, 1900*
Literate Mother	0.860	*1850-1880, 1900*
Literate Father	0.889	*1850-1880, 1900*

Note: Raw mean presented from the 1850-1900 censuses for analysis sample: cohorts born prior to 1899.

TABLE 2: IMPACT OF ABORTION RESTRICTION LAWS ON EARLY CHILDHOOD ENVIRONMENTS

	Maternal age *Mean is 28.433*	**Paternal age** *Mean is 33.549*	**Lives with both parents** *Mean is 0.835*	**Lives with single mother** *Mean is 0.058*	**Lives in group quarters** *Mean is 0.007*	**Paternal LFP** *Mean is 0.958*
Full Sample	0.1073 *(0.1068)*	0.2856*** *(0.1017)*	0.0093* *(0.0049)*	-0.0095*** *(0.0026)*	-0.0015*** *(0.0003)*	-0.0056* *(0.0030)*
Census Years After 1860 Only	0.0422 *(0.0969)*	0.4319** *(0.1651)*	0.0148* *(0.0075)*	-0.0135** *(0.0057)*	-0.0010** *(0.0004)*	-0.0146 *(0.0097)*
	Reported Parental Property > $0 *Mean is 0.757*	**Urban Dummy** *Mean is 0.208*	**Immigrant Mother** *Mean is 0.213*	**Immigrant Father** *Mean is 0.213*	**Literate Father** *Mean is 0.86*	**Literate Mother** *Mean is 0.889*
Full Sample	-0.0199 *(0.0192)*	-0.0277 *(0.0174)*	0.0068 *(0.0163)*	0.0027 *(0.0155)*	0.006 *(0.0039)*	0.0099 *(0.0064)*
Census Years After 1860 Only	-0.0025 *(0.0061)*	-0.0548*** *(0.0191)*	-0.0012 *(0.0203)*	-0.006 *(0.0180)*	0.0092 *(0.0070)*	0.0258*** *(0.0079)*

*Note: Each cell represents a separate regression. Census years include 1850-1900 and birth cohorts born between 1841 and 1899; sample restricted to individuals age 0 to 9 at time of observation. Robust standard errors in parentheses, clustered by state; *** p<0.01, ** p<0.05, * p<0.1. Sample sizes vary slightly by outcome because of occasional missing data. For the full sample regressions, sample size ranges from 2,721 to 2,728 (1,677 for the parental property outcome). For census years after 1860, sample size (state by year by sex observations) ranges from 1,308 to 1,312 (1,038 for the parental property outcome). For the 20 year rule, sample size ranges from 977 to 980 (799 for the parental property outcome).*

ONLINE APPENDIX TO
Effects of Restrictive Abortion Legislation on Cohort Mortality
Evidence from 19th Century Law Variation

Joanna N. Lahey --- Texas A&M University, NBER, and J-PAL ---jlahey@nber.org.
Marianne H. Wanamaker --- University of Tennessee, University of Stellenbosch, NBER, and IZA

May 2022

APPENDIX A: Model of Selection Under Abortion

The figures below contain a general characterization of the socioeconomic selection framework described in Section III of the manuscript. Figures A1a and A1b plot the expected value of both the marginal cost of an additional child, $E[MC(child)]$, and the child's marginal benefit, $E[MB(child)]$, against a household's socioeconomic status. These expectations reflect the mean value of a distribution of marginal costs and benefits, and therefore reflect a population average. Figure A1a shows the case where the marginal benefit of children increases with household income, reflecting the case of more developed countries such as the U.S. in the mid to late 20th century. In contrast, Figure A1b highlights a case where marginal benefits of children are falling with socioeconomic status, as might be the case for a society with high levels of child labor (including the 19th century U.S.). The marginal cost curve is assumed to be increasing at the same constant rate in both cases, although certainly differences likely exist between developing and developed settings. For each case, the bottom panel contains a net benefit curve, reflecting the difference between expected benefits and expected costs.

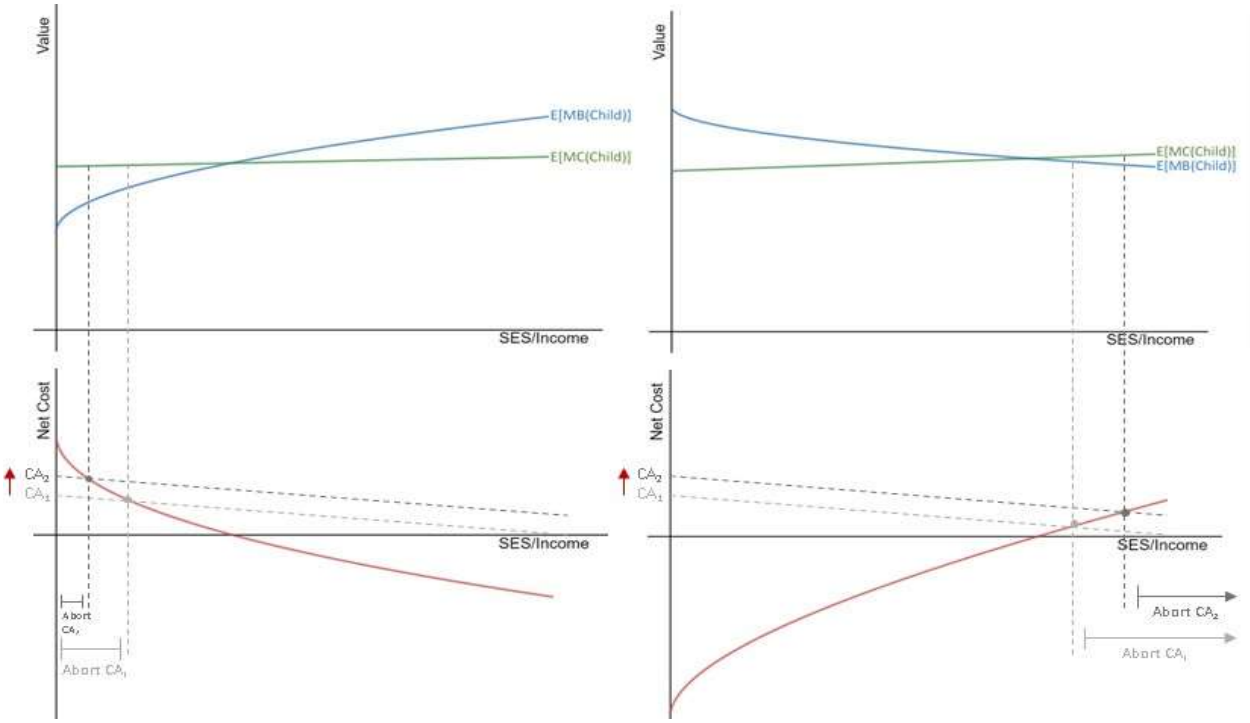
Figure A2 shows a hypothetical population distribution of $MB(child)$ against a fixed value of $MC(child)$- $C_A(income)$ where $C_A'(.) < 0$. In this case, all children to the left of the fixed green line representing $MC(child)$- $C_A(income)$ would be aborted according to the decision rule:

$$Abort\ if\ Net\ Cost(child) > C_A$$

Again, we assume households know with certainty the $MB(child)$ prior to birth.

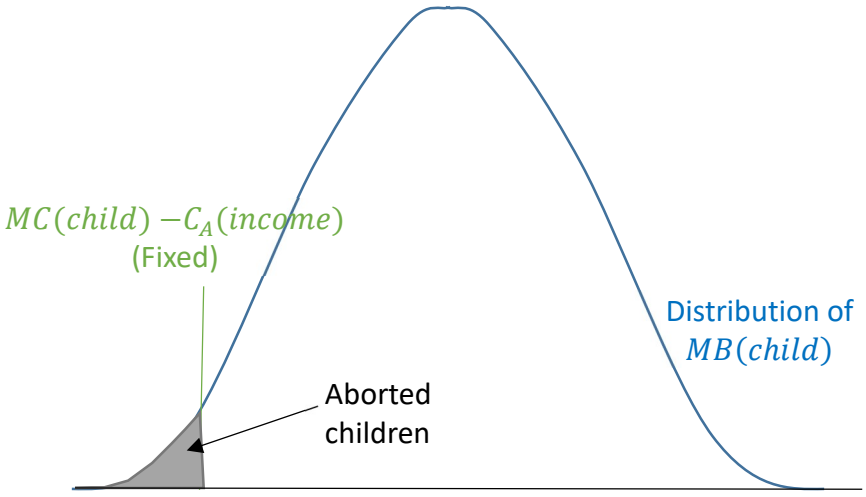
FIGURE A1: CONCEPTUAL FRAMEWORK

a: High Levels of Economic Development b: Low Levels of Economic Development



Note: When the cost of abortion is increased, in the high development scenario, higher SES families on the margin will opt out of abortion. In low development scenario, on the other hand, lower SES families on the margin will opt out of abortion.

FIGURE A2: ABORTION RULE



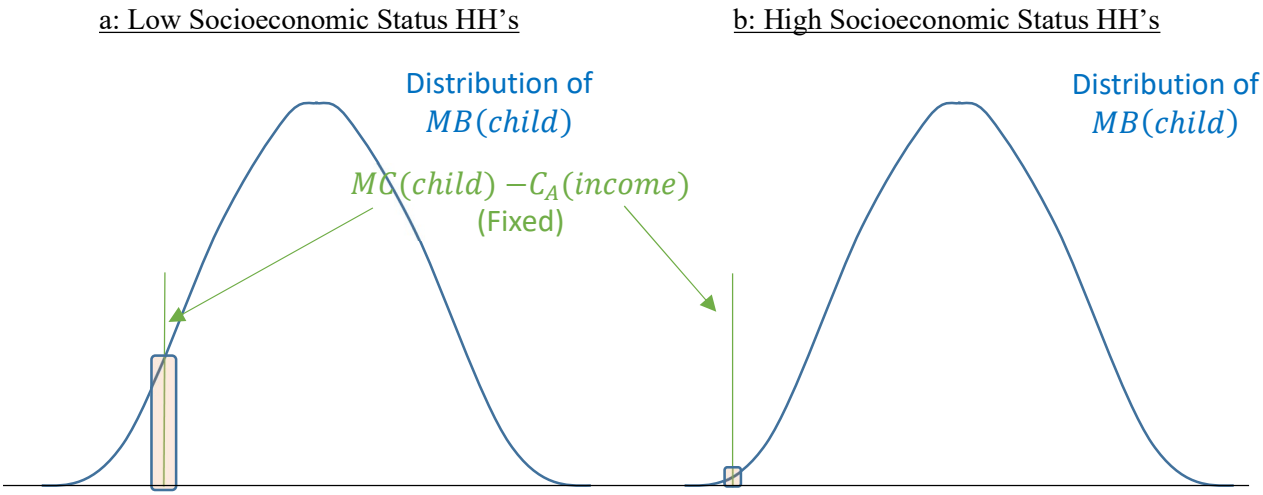
We apply the decision rule in Figure A2 to the marginal cost and marginal benefit curves in Figure A1 to derive predictions about socioeconomic selection in developed and developing countries. To simplify the analysis, we assume that $MC(child) - C_A(income)$ is determined fully by socioeconomic status and, therefore, E$[MC(child) - C_A(income)]$ is fixed for all individuals at a particular level of socioeconomic status, although $C_A(income)$ may be enhanced for lower SES households for the reasons listed above. We further assume that the marginal benefit of children is unimodal and symmetric.

Under these assumptions, Figures A1a and A1b give different predictions about the socioeconomic status of marginal children. Figure A3 represents the characteristics of marginal children in developed countries, corresponding to Figure A1a above. For low SES households, shown in A3a, the distribution of E$[MB(child)]$ is centered to the right of children's (constant) marginal cost less the cost of an abortion, but a substantial part of the left tail is aborted. For high SES households, shown in A3b, the distribution of E$[MB(child)]$ is even further to the right of the cost line, and fewer children are aborted. The shaded region in each panel of Figure A3 represents marginal children: those at risk of being aborted with small changes in the cost of an abortion. Enhancements to the costs of abortion, $C_A(income)$, coming from budget constraints, differences in discount rates, etc., will result in a leftward shift in the vertical green line in Figure A3. *If the enhancements to the costs of abortion,* $C_A(income)$*, for low SES households are large enough*, the shaded region in Figure A3a can approach the size of the region in Figure A3b.

In the case of abortion legalization, as occurred in the 20th century U.S., the associated decreases in the costs of abortion would shift both cost lines to the right, inducing additional abortions among both low and high socioeconomic status households, but moreso (as shown by the vertical rectangles) among low socioeconomic status households. In other words, if enhancements to the costs of abortion among low SES households are low enough, Figure A1a implies more low socioeconomic status households were on

the margin of terminating pregnancies than high socioeconomic status households, and abortion legalization results in positive socioeconomic selection among born children.

FIGURE A3: MARGINAL CHILDREN IN DEVELOPED COUNTRIES, BY SOCIOECONOMIC STATUS



For developing countries, those depicted in Figure A1b, however, Figures A3a and A3b are reversed. Pregnancies among low socioeconomic status household have a higher expected marginal benefit and an enhanced value of $C_A(income)$. As a result, fewer pregnancies are at risk of being aborted among low socioeconomic status households than among high socioeconomic status households and marginal children are disproportionately of higher socioeconomic status. Consequently, abortion restrictions that raise the cost of abortions will induce positive socioeconomic selection among born children.

APPENDIX B: Data Appendix: Cause of Death

i. International Classification of Disease Cause of Death Assignment Rules

For the cause of death work in this manuscript, causes of death in mortality census data and in tabulated volumes of VSUS were assigned International Classification of Disease Version 1 (ICD-1) categories before proceeding with analysis. [35]

Coding for the mortality census string causes of death proceeded as follows:

1. Cleaned all strings, removing non-letter characters and converting to lower case. At this step, there are 1,028,237 observations with 97,094 unique causes of death from 1850 to 1880 mortality census data.
2. Assigned ICD codes based on ICD descriptions and simple variants thereof (e.g. typhoid, typhoid lunch fever, entericfever all received ICD code 18).
3. Used STATA function *regexm* to identify likely deaths from common causes like cancer, accident, maternal mortality, consumption, typhoid, etc, using hand examination of tabulated results to confirm ICD assignments.
4. Tabulated remaining, uncoded causes and coded those with >100 observations by hand.
5. Crosswalked remaining, uncoded causes to Soundex version using R. Among most common soundex codes, tabulated all strings and assigned ICD code (e.g., *mazsles*, *measlees*, *miaslis* all share a Soundex code with *measles*).
6. In all cases, assign a second cause of death if warranted, (e.g., *hepatis consumption* is primary cause of death hepatitis and secondary cause consumption)

In total, 34,774 (3.4%) observations representing 26,977 unique causes of death remained uncoded after these steps.

For VSUS data, the crosswalk to ICD-1 codes is below:

CAUSE OF DEATH - VSUS	ICD-1 CODE
Accidental deaths	187
Acute rheumatic fever	36
Alcoholism (ethylism)	58
All other causes	999
Appendicitis	131
Arteriosclerosis and high blood pressure	113
Biliary calculi, etc.	137
Bronchopneumonia	38
Cancer (other forms)	70
Cancer (other sites)	70
Cancer and other malignant tumors	70

[35] ICD-1 codes found here: http://www.wolfbane.com/icd/icd1h.htm

Cancer of breast	70
Cancer of digestive organs, peritoneum	70
Cancer of female genital organs	70
Cerebrospinal (meningococcus) meningitis	83
Chronic rheumatic diseases of heart	64
Chronic rheumatic diseases of the heart	64
Cirrhosis of the liver	136
Congenital malformations	78
Diabetes mellitus	75
Diarrhea, enteritis, etc.	20
Diphtheria	15
Diseases of coronary arteries, angina pectoris	101
Diseases of ear, nose, and throat	97
Diseases of heart	99
Diseases of heart (other forms)	99
Diseases of pregnancy	158
Diseases of pregnancy, etc	158
Diseases of the heart	99
Diseases of the prostate	146
Diseases peculiar to first year	76
Dysentery	23
Exophthalmic goiter	141
Hemorrhage, trauma and shock	187
Hernia, intestinal obstruction	132
Homicide	187
Influenza	12
Intracranial lesions of vascular origin	95
Lobar pneumonia	36
Malaria	25
Measles	6
Motor vehicle accidents	187
Nephritis	144
Other accidents	187
Other diseases of nervous system, etc.	95
Other diseases peculiar to first year	76
Other puerperal causes	202
Pellagra (except alcoholic)	165
Pneumonia (all forms)	39
Pneumonia (all forms) and Influenza	39
Pneumonia (unspecified)	39
Poliomyelitis, polioencephalitis (acute)	204

Premature birth	76
Puerperal septicemia	202
Puerperal toxemia	202
Puerperal toxemias	202
Scarlet fever	8
Senility, ill-defined and unknown causes	167
Suicide	190
Syphilis	29
Tuberculosis (all forms)	48
Tuberculosis (other forms)	48
Tuberculosis of respiratory system, etc.	48
Typhoid and paratyphoid fever	18
Typhoid, paratyphoid fever	18
Ulcer of stomach or duodenum	162
Whooping cough	13

ii. Categorization

ICD-1 codes in both the mortality census data and the VSUS tabulations were then grouped into broader cause of death categories according to the following rules:

BROAD CATEGORY	**NARROW CATEGORY**	**ICD-1 CODES**
Infectious Disease	Infectious Disease	1-31, 35-49, 51-55, 114-117, 123, 198-199
Accident or Acute Cause	Maternal Mortality	32-34, 154-158, 202
Chronic Disease	Heart Disease	63-65, 72-74, 99-110, 112-113, 168, 197
Chronic Disease	Cancer	68-70, 148, 150, 169
Chronic Disease	Diabetes	75
Accident or Acute Cause	Infant Mortality	76-82, 192-194, 203
Chronic Disease	Other Chronic	50, 57-59, 66-67, 71, 124-125, 127-128, 136-137, 139-147, 149, 151-153, 159-161, 163-164, 166-167, 196, 200, 204
Accident or Acute Cause	Other Acute	56, 60-62, 90, 126, 129-135, 138, 162, 165, 170-172, 205
Chronic Disease	Nervous System	83-89, 91-98, 111

Chronic Disease	Respiratory Disease	118-122, 201
Accident or Acute Cause	Accident	175-191
	Child Abuse (narrow definition)	Age < 18 **AND** (ICD code 56, 57, 166, 181-185, or 190, or any string containing "neglect" or "abuse")
	Child Abuse (broad definition)	Above definition plus Age<18 **AND (**ICD code 175-191, or 106, or any string containing "fract", "injury", "killed in a fight", "gun" or "shot")

In addition, maternal mortality group causes, and ICD codes 29 (syphilis), 30 (gonorrhea) and 166 (atrophy, debility) are all coded as infant mortality if occurring in children younger than age 5. All uncoded observations and those corresponding to ICD-1 codes 173 and 174 were assigned to the broad category "All Other".

iii. Heart Disease Categorization

For the purposes of Figure A4, ICD-1 codes in both the mortality census data and the VSUS tabulations chronic heart-related disease categories plausibly exacerbated by childhood illness were separately identified. From the heart disease codes in the table above, those NOT plausibly exacerbated by childhood illness are codes 65 (chronic rheumatism), 72 (purpura), 73 (haemophilia), 74 (anaemia; leuococythaemia), 106 (cerebral haemorrhage; cerebral embolism), 107 (apoplexy; hemiplegia), 108 (aneurysm), 112 (varicose veins), 168 (dropsy; ascites; anasarca), and 197 (hemorrhoids). These disease categorizations are present in the mortality census data.

In the VSUS data, there are four heart disease sub-categories: chronic rheumatic diseases of the heart; diseases of coronary arteries, angina pectoris; arteriosclerosis and high blood pressure; and other diseases of the heart. Only the last category is defined as NOT plausibly exacerbated by childhood illness.

iv. Infectious Disease Categorization

Infectious disease ICD codes were subdivided into those transmitted person-to-person (and therefore likely to be more apparent with larger cohorts) and those not transmitted person-to-person. Those categorized as not transmitted person to person for the purpose of this exercise are ICD codes 4 (cow-pox), 9 (typhus), 11 (relapsing fever), 18 (enteric fever), 19 (Asiatic cholera), 20 (diarrhea due to food), 23 (dysentery), 24 (tetanus), 25 (malaria), 26 (rabies, hydrophobia), 29 (syphilis), 30 (gonorrhea), 31 (phlegmasia alba dolens), 40 (erysipelas), 41 (septicaemia), 42 (pyaemia), 43 (phegmon, carbuncle), 50 (tabes mesenterica), 123 (tonsillitis/quinsy), 198 (worms).

v. Estimating Sample Restrictions

In addition to the restrictions on statehood (dropping all states not established by 1880) and early law adoption (dropping all states with laws established prior to 1840), the paper's main results are restricted to a sample that is white only, excludes 1850 mortality census data, drops all individuals with missing birth years, and drops all foreign-born individuals.

The estimating sample also includes a common support requirement: all cohorts included in the sample represent birth years when at least one state had passed an anti-abortion law. Cohorts born prior to the passage of any abortion-restricting laws are excluded from the estimating sample.

APPENDIX C: Supplemental Tables and Figures

TABLE A1: ADDITIONAL CHILD HOME ENVIRONMENT OUTCOMES

	Adopted *Mean is 0.001*	**Number of Children <5 in Home** *Mean is 1.472*	**Father is Farmer** *Mean is 0.104*	**Father is Farm Laborer** *Mean is 0.093*	**Father is White Collar Worker** *Mean is 0.137*
Full Sample	-0.0004***	0.0245**	0.0104	-0.008	0.0051
	(0.0001)	*(0.0110)*	*(0.0209)*	*(0.0053)*	*(0.0042)*
Census Years After 1860 Only	-0.0006***	-0.0008	0.0499***	-0.0189*	0.0035
	(0.0001)	*(0.0189)*	*(0.0146)*	*(0.0095)*	*(0.0046)*

	Father is Skilled Blue Collar Worker *Mean is 0.104*	**Father is Semi-Skilled Blue Collar Worker** *Mean is 0.037*	**Father is Unskilled Blue Collar Worker** *Mean is 0.504*	**Mother's Share of Parental Property** *Mean is 0.009*	**Lives with Both Parents (teen)** *Mean is 0.765*
Full Sample	0.0058	0.0013	0.0009	0.0007	0.0182**
	(0.0055)	*(0.0083)*	*(0.0065)*	*(0.0008)*	*(0.0083)*
Census Years After 1860 Only	-0.0032	0.0032	-0.006	-0.0008	0.0233*
	(0.0054)	*(0.0069)*	*(0.0060)*	*(0.0006)*	*(0.0127)*

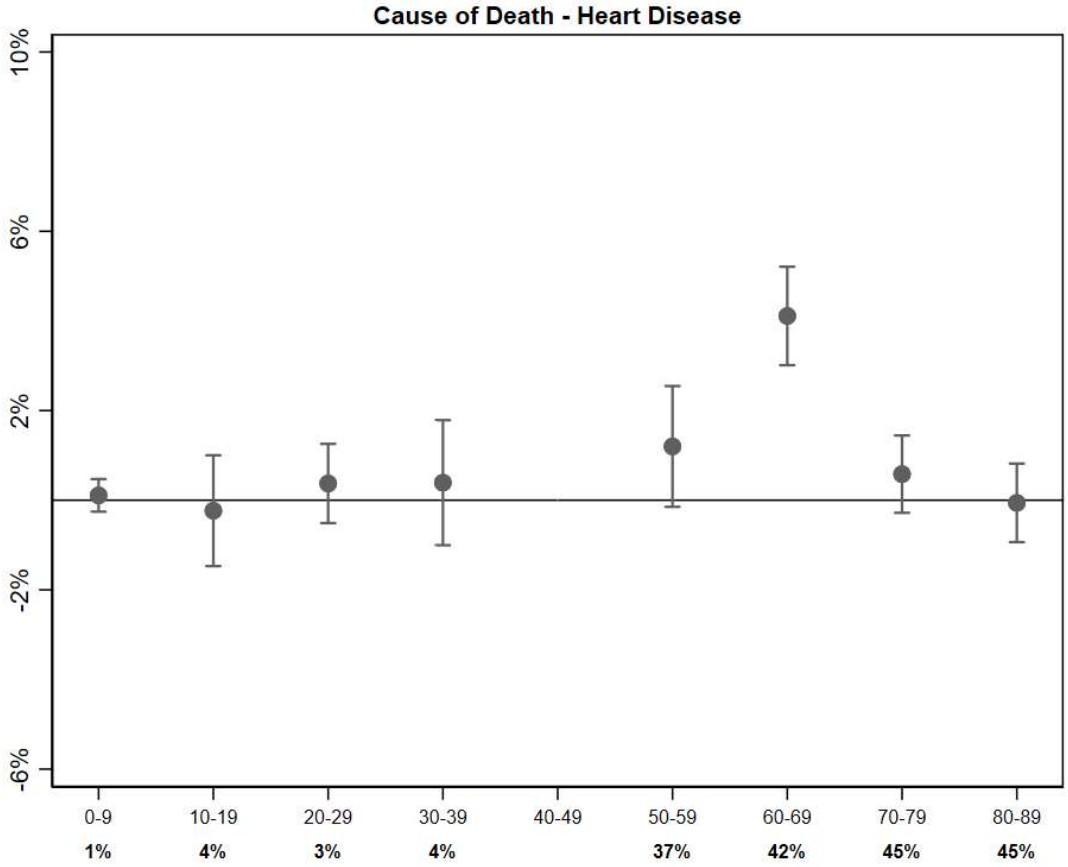
*Note: Each cell represents a separate regression. Census years include 1850-1900 and birth cohorts born between 1841 and 1899; sample restricted to individuals age 0 to 9 at time of observation. Robust standard errors in parentheses, clustered by state; *** p<0.01, ** p<0.05, * p<0.1. Sample sizes vary slightly by outcome because of occasional missing data. For the full sample regressions, sample size ranges from 2,718 to 2,728 for early child outcomes (1,657 for the mother's share of property outcome). For the teen outcome, living with both parents, sample size for the full sample is 3,126.*

TABLE A2: FIGURE 3 RESULTS, IN TABLE FORM

ln(Change in Probability of Surviving to Next Census)	Age 0-9	Age 10-19	Age 20-29	Age 30-39	Age 40-49	Age 50-59	Age 60-69	Age 70-79
Full Sample	-0.034***	-0.025*	-0.012*	-0.014**	-0.013**	0.009	0.022**	-0.011
	(0.010)	(0.013)	(0.006)	(0.006)	(0.005)	(0.010)	(0.011)	(0.019)
Women Only	-0.034***	-0.024*	-0.002	-0.007	0.001	0.018	0.017	0.000
	(0.010)	(0.012)	(0.005)	(0.004)	(0.007)	(0.014)	(0.013)	(0.022)
Men Only	-0.034***	-0.028*	-0.022**	-0.021**	-0.026**	0.003	0.028**	-0.023
	(0.011)	(0.015)	(0.009)	(0.009)	(0.010)	(0.012)	(0.013)	(0.023)
Obs (Full Sample)	2,196	2,763	3,330	3,875	3,962	4,001	4,017	4,500

Notes: Each cell is an individual regression. All regressions include birth year and birth place fixed effects. The Full Sample regressions also include birthplace by sex fixed effects. Each regression is weighted by the count in the previous decade's cell. See Figure 3 for additional notes.

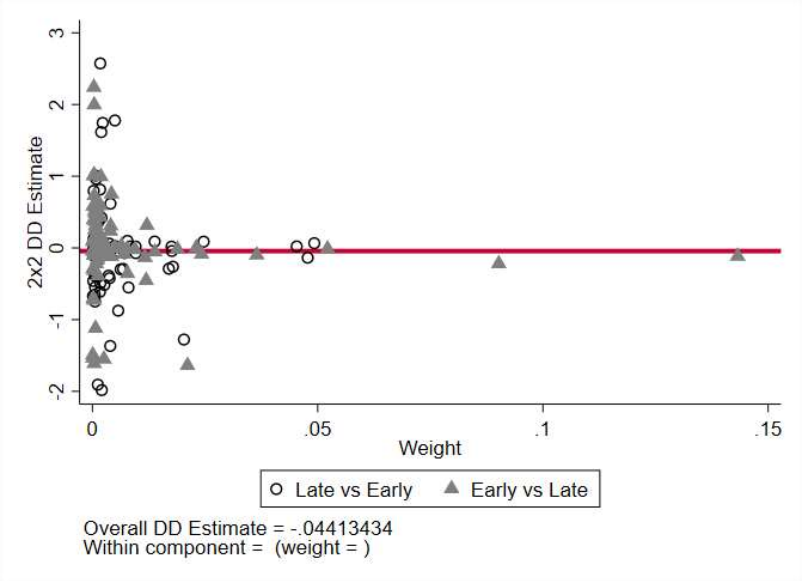
FIGURE A4: CAUSE OF DEATH RESULTS, CHRONIC HEART DISEASE PLAUSIBLY EXACERBATED BY CHILDHOOD ILLNESS AS A SEPARATE CAUSE OF DEATH CATEGORY



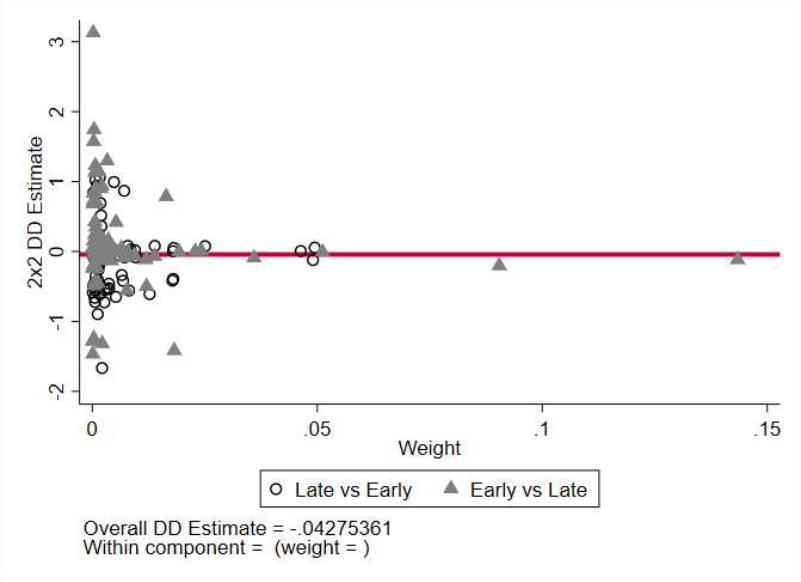
Note: See notes to Figure 4 in the main text for sample sizes and other details and see Appendix Section B.iii. for details on how causes of death are categorized for this exercise. Coefficients shown are the marginal effects for only chronic heart disease deaths plausibly exacerbated by childhood illness as defined in Appendix Section B.iii., leaving other chronic disease deaths as a separate cause of death category, along with a 95% confidence interval. Average values represent the share of all deaths from heart disease within an age bracket. Mortality census data has a lower share of deaths attributable to these chronic heart disease causes because of finer coding in those data than in VSUS.

FIGURE A5: BACONDECOMP RESULTS FOR AGE 0-9

MEN

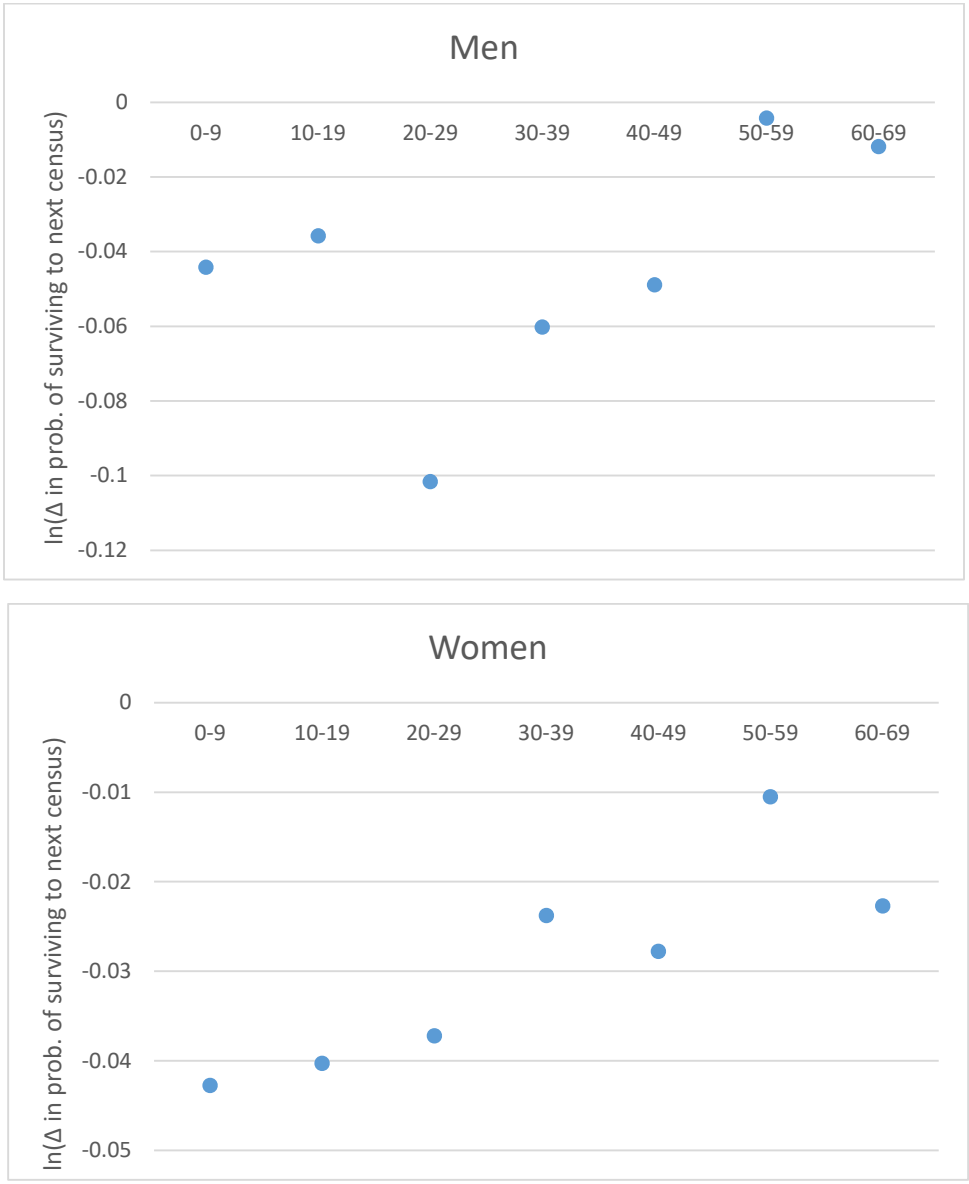


WOMEN



Note: Output from bacondecomp.ado file in Stata corresponding to Figures 3b and c, ages 0-9 (bacondecomp.ado does not currently allow a gender control, so we cannot replicate 3a). To fit the programming constraints of the BaconDecomp model, we round all fractional law variables to the nearest 0 or 1, use average state weights (rather than weights from the previous cell), and remove Nevada as a state of birth since bacondecomp needs a strongly balanced panel.

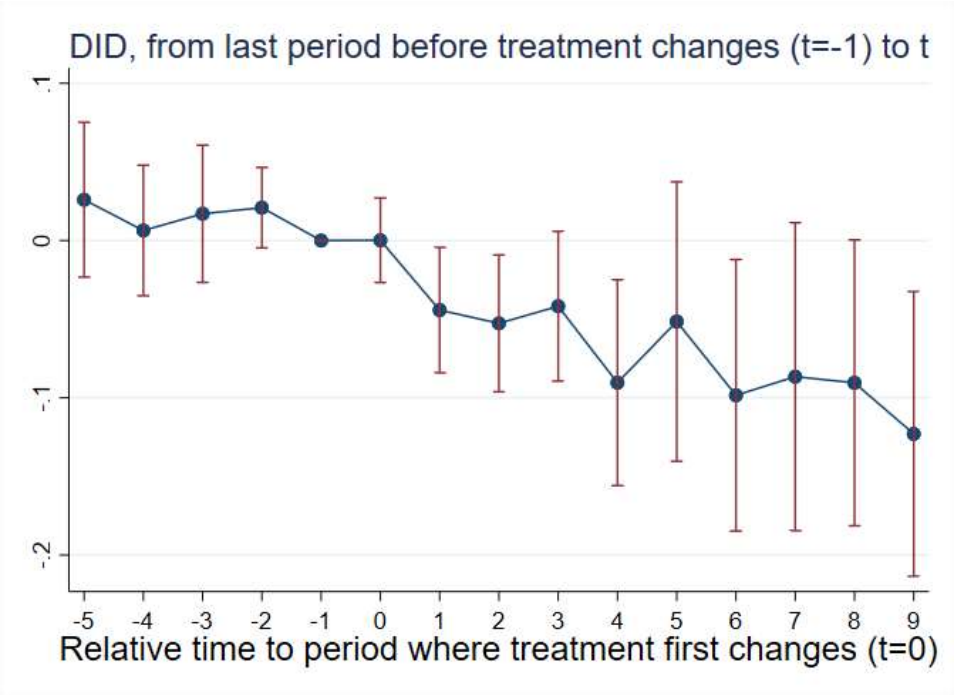
FIGURE A6: BACONDECOMP RESULTS BY AGE GROUP



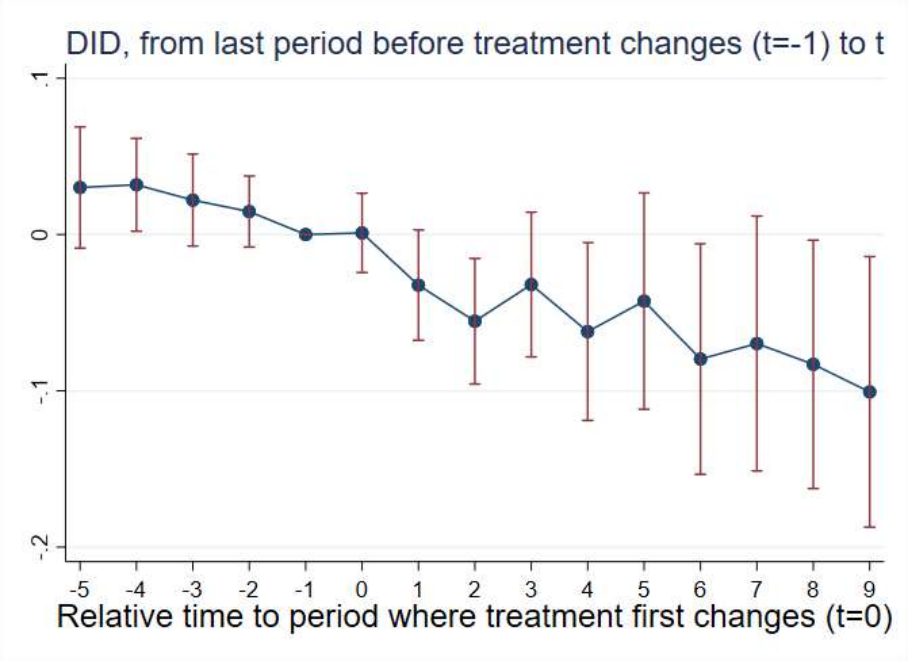
Note: Each dot represents results from a separate bacondecomp regression. Results are the "Overall DD Estimate" for each age group. To fit the programming constraints of the BaconDecomp model, we round all fractional law variables to the nearest 0 or 1, use average state weights (rather than weights from the previous cell), and remove Nevada as a state of birth since bacondecomp needs a strongly balanced panel.

FIGURE A7: DID_ MULTIPLEGT RESULTS FOR AGE 0-9

MALE

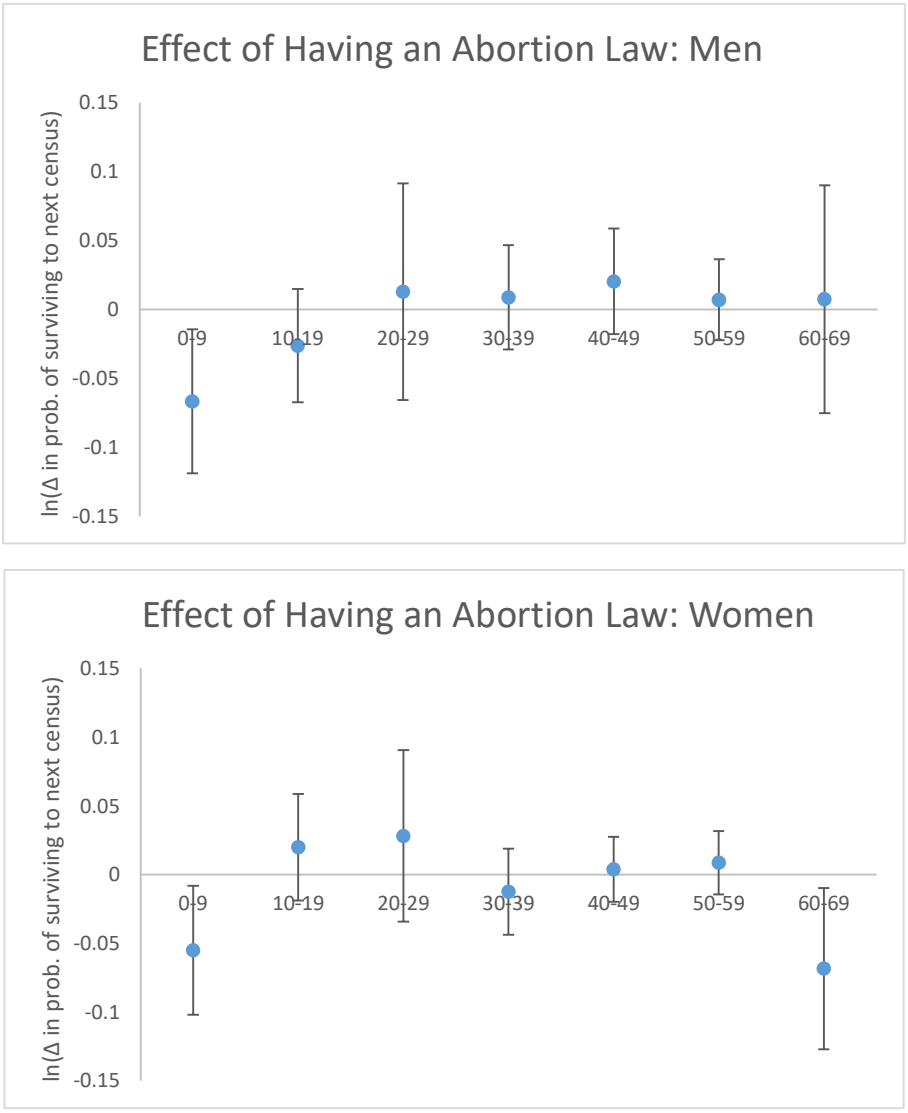


FEMALE



Note: Results from did_multiplegt on ln(percent survive) with year of birth and state of birth fixed effects, weighted by the count in the previous decade cell, using robust_dynamic and average_effect options with 999 bootstrap repetitions. Placebo results are calculated for years -2 to -5 using the placebo option.

FIGURE A8: DID_ MULTIPLEGT RESULTS BY AGE GROUP



Note: Each dot represents the coefficient of a separate regression. Figure shows the results for plotting the results for "Average" using the DID_multiplegt Stata command on the regressions shown in Figure 3 in the main text. Results are weighted by the count in the previous decade cell, using robust_dynamic with dynamic(9) and average_effect options with 999 bootstrap repetitions. (Results with different dynamic choices are very similar.)