Adding Salt to the Womb: The Benefits of Salt Iodization on Infant Health

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Abstract

Iodine deficiency is a major public health issue across the world today affecting an estimated 241 million children. Iodine deficiency is particularly detrimental to fetal and infant health, increasing the risk of infant mortality and inhibiting cognitive development. This paper studies the effects of the nationwide introduction of iodized table salt to the United States in 1924. We exploit this natural experiment and pre-period geographic variation in iodine deficiency driven by differences in naturally occurring iodine using a difference-in-differences design. We find that the introduction of iodized table salt in the United States reduced infant mortality by 0.58–1.1 deaths per 1,000 births (0.8–1.6 percent) for counties at the 75th percentile of the goiter distribution relative to the 25th percentile. We also show that these reductions in infant mortality are concentrated among urban counties, where iodized salt was disproportionately available. Our estimates suggest that salt iodization can explain up to one third of the decline in the county-level urban-rural infant mortality gap between 1919 and 1929. Given the similarities in infant health outcomes between the United States during the 1920s and countries with high infant mortality today, our findings are relevant for contemporary policy targeting iodine deficient regions of the world.

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1 Introduction

Iodine deficiency is a major public health issue across the world today affecting around 1.88 billion people worldwide, including 241 million children (Andersson et al., 2012). Iodine deficiency is particularly detrimental to fetal and infant health, increasing the risk of infant mortality and inhibiting cognitive development (WHO, 2013). Iodine deficiency and its consequences can be prevented in a low-cost, sustainable, and safe way by adding iodine to table salt. As a result of an effort to address widespread thyroid dysfunction, iodized table salt was introduced nationwide in the United States during the 1920s. We use this natural experiment to study the effects of nationwide salt iodization on infant health outcomes in the United States. To our knowledge, this paper provides the first estimates of how salt iodization impacted infant health in the United States.

During the early 20th century, iodine deficiency was widespread throughout the United States. A common manifestation of iodine deficiency is an enlarged thyroid, which is a medical condition know as goiter. During the early 1920s, estimates suggest that up to 40 percent of school age children had a goiter (Schiel and Wepfer, 1976). By the 1950s, the prevalence of goiter fell by around 90 percent based on comparisons of medical examinations of draft age men between WWI and WWII. This reduction is widely attributed to the introduction of iodized table salt in 1924. Previous work has indicated that salt iodization in the United States improved educational attainment and long-run labor market outcomes (Feyrer et al., 2017; Adhvaryu et al., 2020)[1], but these effects may be larger than previously understood if salt iodization allowed infants who might otherwise have died to survive to

[1] Politi (2010) finds similar effects of salt iodization on labor market outcomes in Switzerland during this time period.

adulthood. The medical literature also indicates that the consequences of iodine deficiency start well before human capital can be measured, with negative effects on fetal development. Thus, a natural first order question is how did nationwide salt iodization impact infant health and fertility outcomes?

To analyze the impacts of this nationwide adoption, we use a difference-in-differences design that compares the trajectory of health outcomes before and after salt iodization in places where iodine deficiency was more or less prevalent because of differences in naturally occurring iodine levels. To measure the prevalence of iodine deficiency before the introduction of iodine to table salt in 1924, we use measures of the local incidence of goiter tabulated from 2.5 million medical examinations of draft age men during WWI (Love and Davenport, 1920). We find that the introduction of iodized table salt in the United States reduced infant mortality by 0.58–1.1 deaths per 1,000 births (0.8–1.6 percent) for counties at the 75th percentile of the goiter distribution relative to the 25th percentile. We also document an increase in non-infant mortality in counties with relatively higher levels of goiter after the introduction of iodized salt, which is consistent with findings in the medical literature that adverse health outcomes can occur among iodine deficient elderly individuals if iodine is introduced too rapidly (McClure, 1934; Franklyn et al., 2005; Inoue et al., 2023). This finding suggests there may be a policy relevant trade-off between infant mortality and old-age mortality when considering this type of intervention. We find no evidence that our estimated effects are being driven by changes in health that are unrelated to iodization or changes in the composition of births over this period. Finally, we document that the reductions in infant mortality following iodization are primarily driven by reductions in urban areas, where iodized salt was disproportionately available. Our estimates suggest that salt iodization can

explain up to one third of the decline in the county-level urban-rural infant mortality gap between 1919 and 1929.

Today, 145 counties have some legislation regarding salt iodization and around 88 percent of the world's population uses iodized salt, but iodine deficiency remains prevalent in many developing countries due to inadequate dietary supply (Zimmermann and Andersson, 2021). A number of papers have investigated the health and human capital impacts of treating iodine deficiency in developing countries. In a randomized control trial, DeLong et al. (1997) add iodine to irrigation water in iodine deficient townships in China and find that the infant mortality rate fell by around 40–50 percent. Field et al. (2009) evaluate a large iodine supplement program in Tanzania and find that supplementation during pregnancy increased educational attainment by 0.35–0.56 years 10 to 14 years after treatment. We contribute to this experimental literature by providing intent-to-treat estimates of a *nationwide* iodization intervention in the U.S. during a period of time in which infant health outcomes and levels of iodine deficiency mirrored those of some countries with high infant mortaliy rates today.[2] As global efforts continue to combat iodine deficiency, the lessons learned from this historical intervention can help guide contemporary policy targeting iodine deficient regions of the world.

The remainder of the paper is structured as follows. Section 2 discusses the relationship between iodine deficiency, infant health, and goiter and provides background about the

[2]The average infant mortality rate in the U.S. from 1919–1924 was 66 deaths per 1,000 births in our sample. The mean infant mortality rate today among the World Bank's "low-income" countries is 47 deaths per 1,000. As of 2019, several countries had similar or higher levels of infant mortality than the U.S. during the 1920s: Nigeria (74), Chad (69), Guinea (66.6), Mali (64), Pakistan (56). Total goiter prevalence worldwide ranges from 4.7 percent in Asia to 28.3 percent in Africa (Benoist et al., 2004). Based on careful examinations of men from western U.S. states during WWI, around 6–11 percent had visible goiter (Schiel and Wepfer, 1976).

introduction of iodized table salt in the United States. Section 3 describes the data collected and used in the paper. Section 4 discusses our empirical strategy in further detail. Section 5 presents the main results and Section 6 presents our robustness and placebo exercises. Section 7 presents results on the differential impact of salt iodization in urban and rural areas and discusses potential mechanisms. Finally, Section 8 concludes.

2 Background

2.1 Iodine Deficiency, Infant Health, and Goiter

Iodine deficiency disorders (IDDs) can begin prior to birth and are known to jeopardize the cognitive health and survival of children. During the perinatal, neonatal and early childhood periods, insufficient iodine intake can lead to hyper or hypothyroidism and other health issues. During pregnancy, serious iodine deficiency can lead to prematurity, congenital abnormalities, and stillbirth. These early-life deficiencies can lead to irreversible clinical and sub-clinical mental impairment that can affect later-life performance in school or work. Today, iodine deficiency is typically diagnosed through median urinary iodine concentration. An alternative diagnostic measure that was more common historically is a goiter assessment through palpation. The primary reason for this is that goiter — the medical condition characterized by an enlarged thyroid gland — is primarily caused by iodine deficiency. Since the body needs iodine to produce the thyroid hormone, when iodine levels are insufficient, the thyroid expands in an effort to capture more iodine for hormonal production. Therefore, the presence of goiter is typically associated with iodine deficiency. This relationship has been

well documented since the beginning of the 20th century (Kimball et al., 1919; Mclendon, 1939).

2.2 Salt Iodization in the United States

During the early part of the 20th century, goiter was widespread in the United States. Goiter was particularly endemic in the northern sections of the country. Figure 1A maps the distribution of goiter across the U.S. based on tabulations from medical examinations of draft age men during WWI. This geographic variation in the incidence of goiter is primarily driven by differences in the level of naturally occurring iodine in soil and water (McClendon, 1924; Schiel and Wepfer, 1976). Figure 2 shows the geographic variation in the iodine content of drinking water during the 1920s which closely aligns with the incidence of goiter. The prevalence of goiter was as high as 30 cases per 1,000 men inspected in Michigan and was estimated to affect around 6 percent of men living in Western states (Schiel and Wepfer, 1976).

Iodized table salt was first introduced to the public in 1924 in Michigan. This was the end result of a long campaign by Dr. David Cowie, among others, to address high levels of thyroid dysfunction in his home state. He convinced local salt manufacturers to begin making and distributing iodized salt throughout grocery stores in the state beginning in May of 1924 (Adhvaryu et al., 2020). This public health effort was widely publicized through letters from the State Board of Health. The program was very successful, with rapid distribution of iodized salt to grocery stores McClure (1934). Given the positive reception of iodized table salt in Michigan, national distributors like Morton Salt Company began selling iodized salt

nationwide a few months later. This rapid nationwide distribution of iodized table salt is the natural experiment we exploit for identification in this paper.

Surveys conducted years after the introduction of iodized salt documented decreases in the prevalence of thyroid enlargement. Schiel and Wepfer (1976) note that in a study of Michigan school children in urban areas, the incidence of goiter was 38 percent in 1924, 10 percent in 1928, and declined to 2.4 percent by 1951. Notably, they document large declines in goiter even among children whose families were not using iodized salt which suggests indirect exposure through schools or restaurants. This widespread exposure alleviates potential concerns about selection into iodized salt usage.

3 Data

To measure the local prevalence of goiter, which serves as a proxy for iodine deficiency, we use data from Love and Davenport (1920) who tabulate goiter rates for 156 sections (clusters of counties) in the United States. The goiter rates are calculated based on 2.5 million medical examinations of draft eligible men between the ages of 18 and 30 conducted during WWI. These data provide a snapshot measure of the local prevalence of iodine deficiency during our pre-period.

Our primary outcomes of interest are infant mortality rates (IMR), mortality rates, and birth rates. To measure our outcomes at the county-level we use vitality events data compiled by Bailey et al. (2018). These data come from U.S. Census Bureau publications tabulating vital events for cities and county remainders. Statistics were only tabulated for states in Birth (Death) Registration Areas which began existing in 1915 and were complete by 1933.

For the years in our estimation period (1919–1929) we are able to construct a balanced panel of county-level vital events for 1,273 counties across 24 states. Counties in our estimation sample are displayed in Figure 1B. To construct infant mortality rates, we divide infant deaths by the number of births (in thousands). To measure the mortality and birth rates, we divide non-infant deaths and births by total population and female population aged 15–44 (again in thousands). In order to get annual estimates of these population measures, we do a linear interpolation at the county level using 1910–1940 Census population counts.

To construct these outcomes at the state level, which we use in our robustness analysis, we digitize state-level vitality rates for the period 1915 to 1940 from "Vital Statistics Rates in the United States 1900–1940" published by the United States Public Health Service (USPHS). The USPHS uses its own population interpolations based on deaths, migration, and other factors to compute the vitality rates in intercensal years which is independent from the linear interpolations we use for our county-level data. From these publications, we digitize state-level infant mortality rates (per 1,000 births), all-age mortality rates (per 1,000 population), birth rates (per 1,000 population), and stillbirth rates (per 1,000 births). These vital rates are available for 34 states for the years in our estimation period.

Finally, we use data from the 1920 Census of Population, Agriculture, and Manufacturing schedules from the IPUMS National Historical Geographic Information System (NHGIS). We use the Census data to construct county- and state-level controls. We also use data on county-level number of marriages and divorces from IPUMS NHGIS as placebo outcomes.

Throughout the paper we focus our attention on the infant mortality rate because we believe it is the outcome that is most reliably measured during this period. This is because births and infant deaths are, in theory, reliably recorded through the issuance of birth and

death certifications which facilitates the tabulation of the IMR. In order to calculate mortality and birth rates we need reliable annual measures of county- or state- level population. The only time that population at these levels is systematically recorded is during decennial Censuses. Therefore, we do not have reliable measurements of annual mortality and birth rates. To address this, we use log deaths and log births as population agnostic measures at the county level. At the state level we directly use mortality and birth rates, as opposed to calculating them ourselves from disparate sources. These state-level vitality rates are tabulated using more sophisticated interpolations that take into account births, deaths, and migration over time and are the most reliable data we found covering our time period.

4 Empirical Strategy

In order to study the impacts of salt iodization on infant health, we use a treatment intensity (or dose response) difference-in-differences design that compares the trajectory of infant health outcomes before and after 1924 in places where iodine deficiency was more versus less prevalent because of differences in naturally occurring iodine content in diet and water. The main identification assumption is a strong parallel trends assumption that the trajectory of outcomes, had all counties been exposed to a goiter rate g, is equal to the trajectory for counties that actually have goiter rate g. This is stronger than a traditional parallel trends assumption because it imposes restrictions on potential outcomes under different doses (g) rather than just restrictions on untreated potential outcomes ($g = 0$). Under this assumption, the TWFE estimator recovers a weighted mean of average causal response functions. In our setting, the average causal response function tells us how much the average treatment effect

of iodization on infant mortality changes as we vary the prevalence of goiter g. Under the strong parallel trends assumption, the TWFE estimator recovers a weighted average of these treatment effect parameters with positive weights that sum to one.

We estimate the following differences-in-differences model

$$y_{ct} = \gamma + \sum_{k \in [-T,T]} \beta_k f(G_s) \cdot \mathbb{1}\{t = k\} + \alpha_c + \mu_t + \lambda_{r(s),xst} + \Gamma \mathbf{X}_{c,1920} \cdot t + \varepsilon_{st}$$

Here y_{ct} is an outcome of interest in county c in year t, $f(G_s)$ is a function of the pre-period goiter rate (cases per 1,000 men) in section s. Our baseline specification will use the continuous measure G_s, but we also use terciles of G_s in robustness checks. α_c are county fixed effects, μ_t are year fixed effects, and $\lambda_{r(s),t}$ are region (state or census division)-by-year fixed effects. Finally, we control for a set of county-level covariates from the 1920 Census $\mathbf{X}_{c,1920}$ interacted with a linear time trend. In all our analyses, $\mathbf{X}_{c,1920}$ includes the share of the population living in urban areas, share of the population that is Black, share that are under 7 years old, share that are over 44 years old, log average manufacturing wages, log population, percent of the area that is farm land, percent of the population that is illiterate, and the percent of the population that is white and foreign born. Sub-state level variation allows us to potentially control for any confounding state-by-year changes to infant health outcomes (e.g. state laws that might impact infant health). For ease of interpreting the estimated coefficients, we standardize the treatment variable so that β_k is interpreted as the estimated effect of a one standard deviation increase in the goiter rate. Additionally, due to potential noise and transcription errors in these historical data, we winsorize both the goiter rates and our outcome variables at the 99th percentile. Finally, we cluster our standard

errors at the section level.

Our main estimation sample consists of 1,237 counties in 24 states across the United States. Our primary period of interest begins in 1919, five years prior to the mass introduction of iodized salt, and ends in 1929, prior to the Great Depression. Although, we have measures of section-level goiter rates for all sections in the U.S. going back to 1919, we only observe vital events starting in 1919 for these 1,237 counties. These counties account for 66 percent of the observed variance in our section-level goiter rates. Table 1 presents summary statistics for counties in our estimation sample. The mean county-level goiter rate in our sample is 6.21 cases per 1,000 men inspected. At the time of these examinations, there was substantial heterogeneity across locations in the prevalence of goiter with the 10th percentile of the county-level distribution around 1.08 cases per 1,000 men and the 90th percentile around 15.7 per 1,000. Iodine deficiency, as proxied by the prevalence of goiter, was the the highest in Midwestern and North Western states. Infant mortality in the year prior to iodization was around 66 infant deaths per 1,000 births and also varied substantial across counties ranging from as low as 42.6 at the 10th percentile to 90.9 at the 90th percentile of the county-level distribution. To put this into perspective, the mean infant mortality rate among low-income countries today is around 47 deaths per 1,000 births.

5 Effects of Salt Iodization

We begin by discussing the impact of salt iodization on infant mortality at the county level. Figure 5 displays the effect of the 1924 mass introduction of iodine to table salt on the infant mortality rate. Prior to 1924, we see no differences in the trajectory of infant mortality

rates across places with higher or lower levels of goiter prevalence with the 1919–1923 point estimates centered around zero. In 1925 and 1926 we see muted effects on infant mortality rates in higher goiter counties. Interestingly, this pattern is consistent with Adhvaryu et al. (2020) who find the long-run labor market gains of salt iodization are concentrated among individuals born between 1928 to 1931 and find muted effects for those born between 1924 and 1927. This may partly reflect the fact that children born in these years would have only been partially treated during gestation by iodized table salt which began to be distributed during the latter half of 1924. Starting in 1927, the estimated reductions in infant mortality rates began to grow linearly until the end of our sample period in 1929. The coefficient in 1929 reflects a reduction of around 2 infant deaths per 1,000 births in counties with a one standard deviation higher pre-period goiter rate, which is a 2.8 percent reduction relative to the pre-period mean.

Over the entire post period, we estimate that salt iodization reduced the infant mortality rate in counties with a one standard deviation higher goiter relative to the mean county by 1.06 deaths per 1,000 (see Table 2, column 1). In columns (2) – (4) of Table 2 we show that our estimates are not sensitive to additional controls or potential confounds. If there are state or region specific shocks in areas with higher levels of goiter that impact infant health and coincide with the timing of salt iodization then these changes could drive our results. In our baseline county-level specification, we control for region-by-year fixed effects which should account for region specific shocks. U.S. regions are large so there may be sub-region time-varying shocks that impact infant health. To address this, in Table 2 column (2) we add division-by-year fixed effects. Additionally, in Table 2 column (3) we add state-by-year fixed effects to address any state-specific policies enacted during this period. Including these addi-

tional controls does not meaningfully change the magnitude of our estimated effects. Finally, in our baseline specification we only allow our 1920 controls to impact our outcomes through a linear trend. In Table 2 column (4) we relax this linearity by binning each of our 1920 controls into quintiles and including covariate quintile-by-year fixed effects. Importantly, our results are also robust to this alternate specification.

To assist with the interpretation of the magnitudes of these estimated effects, we compare them to other interventions that affected infant health during this time period. As one point of comparison, Barreca et al. (2014) find that reductions in household use of bituminous coal for heating between 1945 and 1960 reduced winter infant mortality rates by about 2 to 3 percent depending on the specification. At the mean level of goiter prevalence, we find that salt iodization reduced infant mortality rates between 1919 and 1929 by 0.9–1.8 percent. Similarly, Clay et al. (2014) find that a one standard deviation increase in the pH level of pipes among cities that used lead pipes reduced the infant mortality rate by around 11 percent in 1900. Our estimates imply that counties with one standard deviation higher goiter rates experienced reductions in infant mortality of around 1–2 percent or around 9–18 percent of the effect size found in Clay et al. (2014).

6 Robustness

6.1 State-Level Estimates

Reliable county-level birth and infant death statistics allows us to evaluate the effects of salt iodization on infant mortality rates, but there are other measures of population health that

merit further attention. We do not have a reliable measure of intercensal population for the denominator at the county-level (as discussed in Section 3), so we conduct further analysis at the state-level where population estimates are measured with greater accuracy. First, given concerns that rapid increases in iodine consumption may have adverse health effects on the elderly population, we estimate effects of salt iodization on the total morality rate. Second, if iodized salt affected fertility outcomes, the reductions in infant mortality we find could be driven by the composition of infants born, so we estimate effects of salt iodization on fertility rates. Finally, stillbirth data are not available on the county-level but are available on the state level, so we we estimate effects using state-level variation for this additional measure of infant health.

To our knowledge, the state-level vital rates published in the USPHS publication are the best measure of mortality and birth rates, which are based on their population interpolations. In robustness exercises in Section 6.3, we replicate this analysis at the county level using log deaths and births as population-agnostic alternate measures.

Infant Mortality. We begin the state-level analysis by replicating our county-level infant mortality results. We do this because the sample of states where we observe state-level vitality rates differs from the sample where we observe the county-level data since they come from different sources. Figure 6 displays the estimate effects of iodization on infant mortality rates at the state level. Similar to our county-level results, the estimated coefficients prior to 1924 are centered around zero and the estimated effect increases starting in 1927. The overall estimated effect is a statistically insignificant reduction of 1.14 deaths per 1,000 births (see Table 3, column 1). However, we do estimate a significant effect by 1929 in our event study specification. Reassuringly, the state-level results are very similar

to our county-level results.

Overall Mortality. Next, we look at the impact on overall mortality. Figure 7 shows the estimated effect of salt iodization on total mortality rates. Again we find little evidence of differences prior to the treatment year. If anything the impact of salt iodization on mortality is positive. Initially, the estimated effect rises after 1924 peaking three years post at an additional 0.2 deaths per 1,000 (see Table 3, column 2) for counties with a one standard deviation larger goiter rate compared to the mean. The estimated coefficient returns to zero by 1928. Overall, we estimate a statistically significant positive effect of salt iodization of an additional 0.21 deaths per 1,000 (1.8 percent increase relative to the pre-period mean) for counties with a one standard deviation higher goiter rate relative to the mean county (see Table 3, column 2). These findings are consistent with a medical literature that documents thyroid related deaths among older individuals following rapid changes in iodine levels (McClure, 1934; Franklyn et al., 2005; Inoue et al., 2023). This increase in mortality introduces an interesting and relatively undocumented trade-off between improvements in infant mortality and deterioration in old-age mortality for policymakers considering iodization interventions.

Births and Stillbirths. We also look at the impact of salt iodization on fertility to understand whether these changes in infant health are driven by changes in the composition of births across places with different levels of goiter prevalence. Figure 8 presents the state-level event study estimates for birth rates. Overall, we estimate a small and statistically insignificant effect of iodization of an additional 0.13 births per 1,000 (see Table 3, column 3). This increase in fertility is plausibly consistent with improvements in fetal health that move the marginal stillborn child into a live birth. We show evidence consistent with this in

Figure 9, which displays the estimated effect of iodization on the stillbirth rate (stillbirths per 1,000 births). The estimated effect on stillbirths is an insignificant reduction of 0.07 stillbirths per 1,000 births.

Overall, the results in this section are consistent with our county-level estimates and provide evidence that the changes in infant health we document are not being driven by changes to the composition of births.

6.2 Placebo Exercises

Another concern is that our results could be driven by differential trends in infant health or determinants of infant health unrelated to iodine deficiency in places with overall higher prevalence of disease. To provide evidence against this alternate explanation, we re-estimate our baseline specification but use the prevalence of scoliosis as opposed to goiter as our "treatment" variable. We use scoliosis since it is an alternate measure that is correlated with overall health but is unlikely to be driven by factors that contribute to the prevalence of goiter and is not treated by iodine. Figure A6 displays the estimated effects using this placebo treatment. When using the prevalence of scoliosis as our treatment variable we find no impact on infant mortality rates. This suggests that our findings are not driven by changes in health across counties that are unrelated to the introduction of iodized salt.

To further rule out the concern that that differential trends in unobserved determinants of infant health in counties with higher or lower goiter prevalence are driving our results, we estimate the impact of salt iodization on marriage and divorce rates at the county-level. If changes in unobserved determinants are driving the impacts on infant health we may expect

to see changes in marriage and divorce rates. Figure A7 displays the estimated effects on marriage and divorce. Reassuringly, we see no evidence of pre-trends in these outcomes and no impact of iodization.

6.3 Alternate Specifications

County Log Outcomes. One potential concern with our county-level analysis is that we do not have good measures for the denominator of mortality and birth rates. This is why we analyze these outcomes at the state-level instead. However, to further address this issue, we also estimate our county-level specification on the logarithm of infant deaths, non-infant deaths, and births. Table A6 displays the county-level estimates on log infant deaths. Similar to our infant mortality rate analysis, we estimate that salt iodization reduced infant deaths by 3–6 percent in counties with one standard deviation higher goiter rates relative to the mean county. Table A7 and Table A8 do the same for log non-infant deaths and log births. For non-infant deaths, we estimate a marginally significant increase in log non-infant deaths of around 1–2 percent. We find no impact on log births.

Discrete Treatment. To address issues associated with using continuous treatment variables in difference-in-difference specifications we discretize our treatment variable into region specific terciles and re-estimate our county-level specifications. Table A3 presents the estimates using our discrete treatment. The coefficients here represent the estimated effect of salt iodization for counties in the top tercile of the regional goiter distribution relative to the bottom tercile. Using this specification, we estimate that infant health declined by 1.9–3 infant deaths per 1,000 births for counties in the top tercile of the pre-period goiter

distribution relative to the bottom tercile. The estimated effects are similar in magnitude to our county-level estimates that use our continuous treatment.

7 Urban Rural Differences and Food Technology

Although iodized table salt quickly diffused to grocery stores across the United States, Americans in rural and urban places differed in the types of salt they purchased and the quantities of salt purchased at a time. As the Executive Vice President of Morton Salt Company, J.A. Clements, described in his 1947 testimony to Congress, "The farmer buys from the country dealer or village store 100-pound bags of pure granulated salt or in some cases, the coarser or grainer salt. He uses this in a variety of ways: for his livestock, in his own kitchen and on the table." An image of such salt is depicted in Figure 12. Up through the 1940s, coarse salt and salt stored in large bags could not be successfully iodized due to the chemical instability of iodine. J.A. Clements further explains:

> "Up to the present time the salt industry has confined the iodization of salt to the type known as fine or granulated. The layman sees this type on his table. There are, however, other grades, varying in crystal size and structure, which are produced both for farm use and for industrial use in food processes. Broadly speaking, these types are referred to in the trade as "grainer" or "coarse flake" salt. Thus far no process has been developed to the best of our knowledge by which these particular grades of salt could be satisfactorily iodized...Many years of experience in producing and marketing iodized salt has established the fact that the best method of packaging this type of salt is in fiber cans, treated against moisture penetration. Originally all iodized salt was so packed." (J.A. Clements, 1947).

If the iodized salt in small fiber cans never made it to the tables of individuals living in rural areas, we would expect that rural counties, which were thus not fully treated by the iodization of salt, would not experience the same declines in infant mortality that we see in

aggregate. These differences in the types of salt purchased in urban and rural areas motivate our evaluation of heterogeneous treatment effects by the share of the population in a county living in an urban area.

Figure 10 displays trends in infant mortality rates for urban and rural counties separately. The infant mortality rates in urban counties are about 6 percent higher (4.5 additional infant deaths per 1,000 live births) on average in 1919 but converge to the rural county average by 1929. The urban-rural convergence is especially stark following the introduction of iodine to salt in 1924. We investigate the relationship between the urban-rural convergence in infant mortality rates and salt iodization more rigorously in the regressions presented in Table 4. Column 1 displays estimates from our baseline specification but adds an additional interaction for counties that are in sections where the 1920 percent urban is above the national median. We find that almost all of the reduction in infant mortality rates is driven by counties in sections with more urban populations.

An alternate explanation for these differences is that there may be pre-period baseline differences in goiter prevalence among urban and rural counties. If this is the case, then the urban counties located in sections with high goiter rates would drive the results. To assess whether baseline goiter rates are driving our urban-rural differences, we collect data from surveys conducted during the 1920s on the prevalence of goiter among school children across 130 localities in four states. We regress the measured prevalence in each locality against the population and find no statistically significant relationship between goiter prevalence and locality size (see Figure 11). We view this as evidence against the explanation that these urban rural differences are driven by differences in baseline goiter rates.

This urban-rural heterogeneity in the effects of salt iodiziation may also indirectly con-

tribute to other infant health disparities for groups that tend to live in urban areas. We run similar regressions to explore the relationship between iodization and other covariates of interest that are known to be correlated with urbanicity (occupation score, share Black, and share foreign born, see Figure A11 for the raw relationships). In columns (2)–(4) we estimate similar regressions allowing the effect of salt iodization to differ by whether a county is in a section with above or below median occupation scores, share Black, and share foreign born. We find that the infant mortality reductions of salt iodization are also concentrated among counties in sections with above median occupation scores, below median shares of the population that is Black, and above median shares of the white population that is foreign born. These patterns are all consistent with the impacts of salt iodization on infant health being concentrated among urban counties, suggesting that salt iodization may have affected other infant health disparities through geographical segregation of low-skill workers, Black individuals, and the native-born population.

Overall, we interpret this as evidence that the differences in urban-rural infant mortality reductions were likely driven by differences in access to iodized salt. These findings indicate that technological limitations may be responsible for the differential effects we find in urban versus rural counties. Our estimates suggest that salt iodization can explain up to one third of the decline in the urban-rural infant mortality gap between 1919 and 1929 in our sample.[3] More broadly, these disparate impacts highlight how the reach of public health policy can be limited by technology, limiting progress for the health of individuals living in rural areas.

[3]The average infant mortality rate gap between above median and below median urban counties decreased by 4.8 deaths per 1,000 births in our sample between the pre (1919–1924) and post (1925–1929) periods. To arrive at this back-of-the-envelope approximation we use the the equivalent unstandardized coefficient from the regression in Table 4 (column 1) is -0.259. This implies a differential decline of $-0.259 \times 6.2 = 1.61$ deaths per 1,000 live births for urban areas (evaluated at the mean goiter rate of 6.2 cases per 1,000 examined men). This explains $1.61/4.8 \approx 34\%$ of the observed decline.

8 Conclusion

In this paper, we provide the first estimates on the impact of nationwide salt iodization on infant health in the United States. To do so, we exploit the rapid adoption of iodized table salt in 1924 and the fact that some areas were more iodine deficient due to geological variation in the natural levels of iodine. To proxy for iodine deficiency we use the incidence of goiter among draft age men examined during WWI. We document significant reductions in infant mortality rates following iodization. We estimate that counties at the 75th percentile of the goiter distribution experienced decreases of 0.58–1.1 deaths per 1,000 births compared to those at the 25th percentile. We also find no evidence that our estimated effects are driven by health trends unrelated to iodization or changes in fertility. The magnitude of these effects is similar to the effects of reductions in the use of coal heating (Barreca et al., 2014) and around 10 percent of the effect size of decreases in lead exposure (Clay et al., 2014). We also document increases in non-infant mortality rates in areas with higher goiter prevalence suggesting a trade-off between long-run reductions in infant mortality and short-term increases in elderly mortality. Finally, we show that these improvements in infant health are concentrated in urban counties where iodized salt was more widely distributed.

Through our evaluation of salt iodization in the United States, we build on the historical economic analyses of large scale public health interventions. Given the similarities in infant health outcomes between the United States during the 1920s and several countries today, our findings are relevant for contemporary policy-making in iodine deficient regions of the world. We also shed light on the mechanisms behind the long-run benefits of iodization found in prior literature (Politi, 2010; Feyrer et al., 2017; Adhvaryu et al., 2020) by docu-

menting direct effects on infant health. Our results suggest that the estimated effects of salt iodization on human capital may be larger than previously thought when taking into account changes in infant mortality. Lastly, we document how limitations in food science technology can limit the effectiveness of public health interventions, with implications for urban-rural health disparities. Given our findings on the differential impact of iodization in urban areas, investigating the consequences of this disparate impact on differences in long-run human capital outcomes between urban and rural areas may be fruitful.

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9 Tables

Table 1: Summary Statistics

	Mean (Std. Dev.)	$p10$	$p25$	Median	$p75$	$p90$
Treatment						
Goiter Rate	6.21	1.08	1.22	4.30	6.46	15.71
	(6.56)					
Outcome Variables (in 1924)						
Infant Mortality Rate	66.33	42.60	52.94	64.79	77.67	90.91
	(21.86)					
Mortality Rate	9.63	6.06	7.75	9.39	11.02	12.82
	(4.29)					
Birth Rate	110.39	80.86	90.58	103.37	125.15	147.98
	(37.32)					
Control Variables (in 1920)						
Population (thousands)	48.59	7.14	12.44	21.08	36.27	75.57
	(187.92)					
Females 15-44 (thousands)	11.42	1.47	2.65	4.49	7.88	16.78
	(49.14)					
Pct. urban	0.24	0.00	0.00	0.18	0.41	0.64
	(0.27)					
Pct. Black	0.08	0.00	0.00	0.01	0.06	0.35
	(0.16)					
Pct. population under 7	0.16	0.12	0.14	0.16	0.18	0.21
	(0.03)					
Pct. population over 44	0.22	0.15	0.18	0.22	0.26	0.29
	(0.05)					
Pct. farmland	0.69	0.21	0.53	0.78	0.91	0.94
	(0.27)					
Log mean value of farmland	4.16	3.30	3.69	4.14	4.62	5.04
	(0.70)					
Pct. literate	0.94	0.86	0.92	0.97	0.99	0.99
	(0.06)					
Pct. foreign born white	0.10	0.00	0.01	0.07	0.18	0.26
	(0.11)					
Log value-added in manufacturing	14.17	11.37	12.67	14.16	15.59	16.90
	(2.15)					
Obs.	1,273	1,273	1,273	1,273	1,273	1,273

Notes: Sample is all counties where we observe mortality outcomes starting in 1919. Goiter rates are from Love and Davenport (1920) and are measured prior to 1920. Statistics for outcome variables are computed in the treatment year 1924. Controls are from the 1920 Census.

Table 2: Impact on Infant Mortality Rate

	Infant Mortality Rate			
	(1)	(2)	(3)	(4)
Goiter $\times \mathbb{1}\{t > 1924\}$	-1.06***	-0.72**	-1.37**	-1.14***
	(0.32)	(0.32)	(0.67)	(0.30)
Mean Outcome	69.93	69.93	69.92	69.93
Division-Year FE		✓		
State-Year FE			✓	
Control Quintile × Year FE				✓
R-sq	0.72	0.72	0.73	0.73
Obs.	13,596	13,596	13,585	13,596

Notes: The outcome variable is the infant mortality rate measured as the number of infants deaths per 1,000 population. The goiter rate is standardized so that the coefficients represent the estimated effect of a 1 standard deviation increase. All columns include year and county fixed effects. Columns (1) – (3) include 1920 county level covariates interacted with a linear time trend. Column (1) is our baseline specification and includes region-by-year fixed effects, column (2) includes division-by-year fixed effects, and column (3) uses state-by-year fixed effects. Column (4) is our baseline specification but controls for county characteristics more flexibly by including covariate quintile-by-year fixed effects for each covariate. The estimation period is 1919-1929. All regressions are weighted by pre-period (1919-1924) average births. Standard errors are clustered at the section level.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$

Table 3: State Level Estimates

	Infant Mortality Rate	Mortality Rate	Birth Rate	Stillbirth Rate
	(1)	(2)	(3)	(4)
Goiter $\times \mathbb{1}\{t > 1924\}$	-1.14	0.21**	0.13	-0.07
	(0.67)	(0.09)	(0.12)	(0.57)
Mean Outcome	72.80	12.13	21.84	37.73
R-sq	0.95	0.93	0.98	0.97
Obs.	239	363	239	221

Notes: The outcome variables are infant deaths per 1,000 births, all age deaths per 1,000 population, births per 1,000 population, and stillbirths per 1,000 births. The goiter rate is standardized so that the coefficients represent the estimated effect of a 1 standard deviation increase. All columns include year, state, and region-by-year fixed effects and 1920 state level covariates interacted with a linear time trend. The estimation period is 1919-1929. All regressions are weighted by the 1920 populaiton. Standard errors are clustered at the state level.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$

Table 4: County Infant Mortality Rate – Heterogeneity

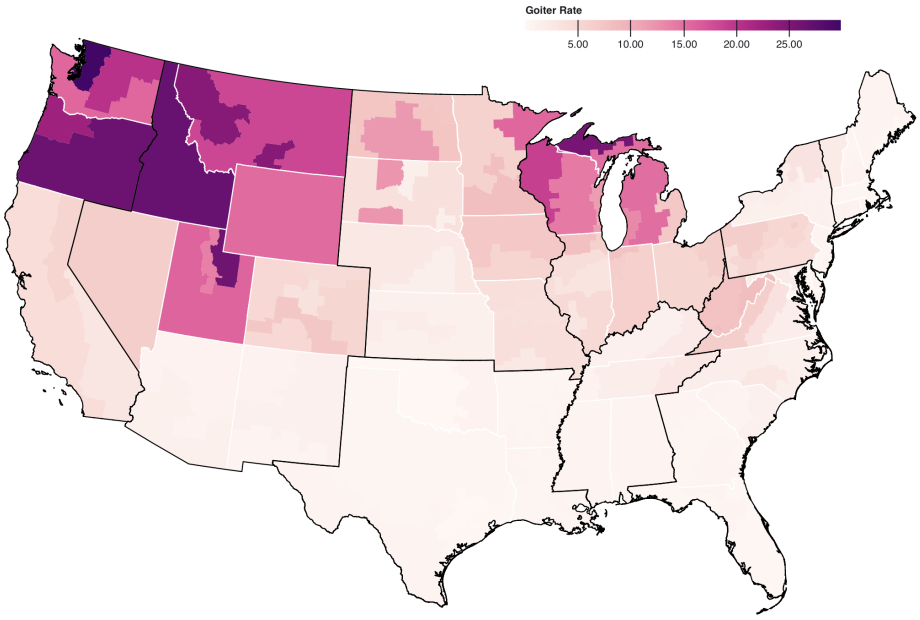
	Pct. Urban	Occupation Score	Pct. Black	Pct. Foreign Born
	(1)	(2)	(3)	(4)
Goiter $\times \mathbb{1}\{t > 1924\}$	-0.26	-0.05	-1.37***	0.26
	(0.45)	(0.40)	(0.34)	(0.50)
Goiter $\times \mathbb{1}\{t > 1924\} \times$ Above Med.	-1.68**	-1.86***	-1.00	-1.90***
	(0.69)	(0.60)	(1.87)	(0.71)
Mean Outcome	69.93	69.93	69.93	69.93
R-sq	0.72	0.72	0.72	0.72
Obs.	13,596	13,596	13,596	13,596

Notes: The outcome variable is infant deaths per 1,000 births. "Above Median" is an indicator that equals one if the section is above the median of the national section-level distribution for the relevant variable (i.e. occupation score). The goiter rate is standardized so that the coefficients represent the estimated effect of a 1 standard deviation increase. All columns include county fixed effects as well as year and region-by-year fixed effects interacted with the "above median" indicator, and 1920 county level covariates interacted with a linear time trend. The estimation period is 1919-1929. All regressions are weighted by the pre-period number of births. Standard errors are clustered at the section level.

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

10 Figures

(A) Entire U.S.



(B) Estimation Sample

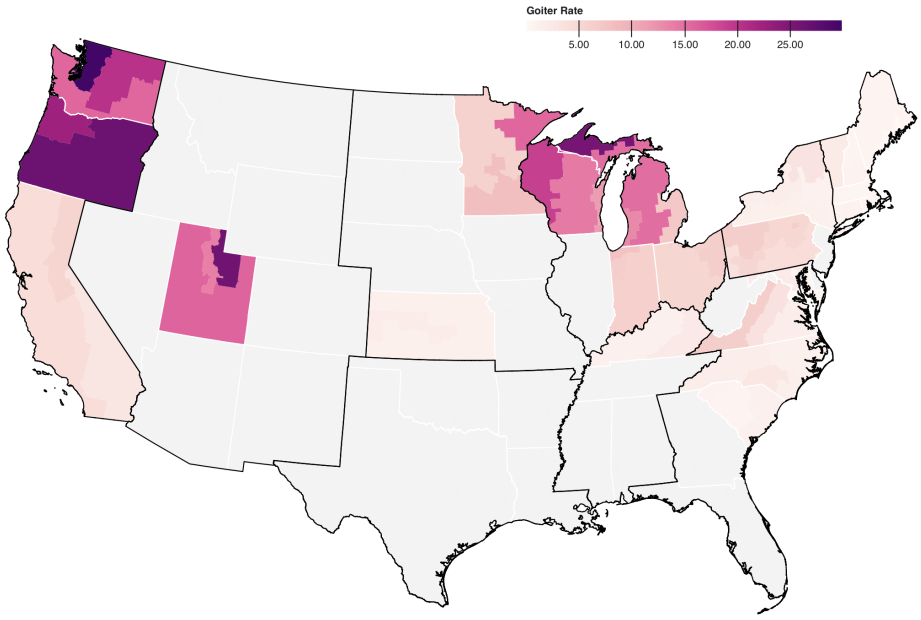


Figure 1: Goiter Rates. This figure displays rates of simple goiter per 1,000 inspected soldiers at the section level from Love and Davenport (1920). State boundaries are denoted by the white lines and Census divisions are denoted by the black lines. Panel A displays goiter rates for all sections in the United States and Panel B displays goiter rates for sections in our estimation sample.

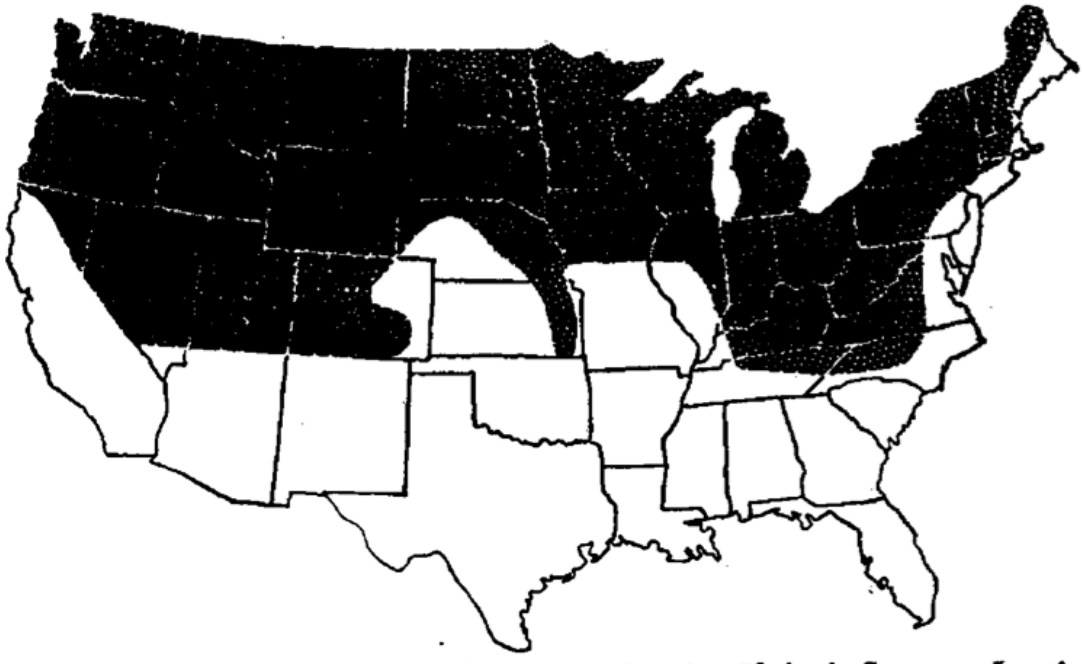


Figure 2: Iodine Content of Drinking Water. This graph displays areas of the United States with high and low levels of iodine in drinking water. Areas marked in black have low levels of iodine (1 to 22 parts iodine per hundred billion parts water). Source: McClendon (1924).

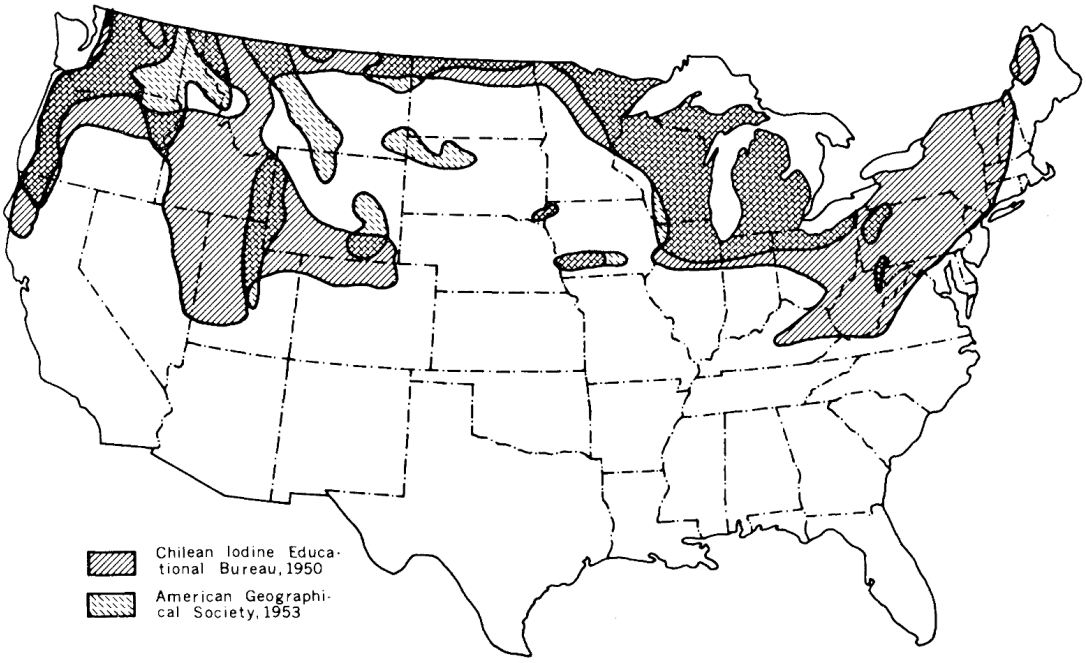


Figure 3: Prevalence of Goiter During 1950s. This map displays areas of endemic goiter from two independent surveys during the 1950s. Source: Schiel and Wepfer (1976).

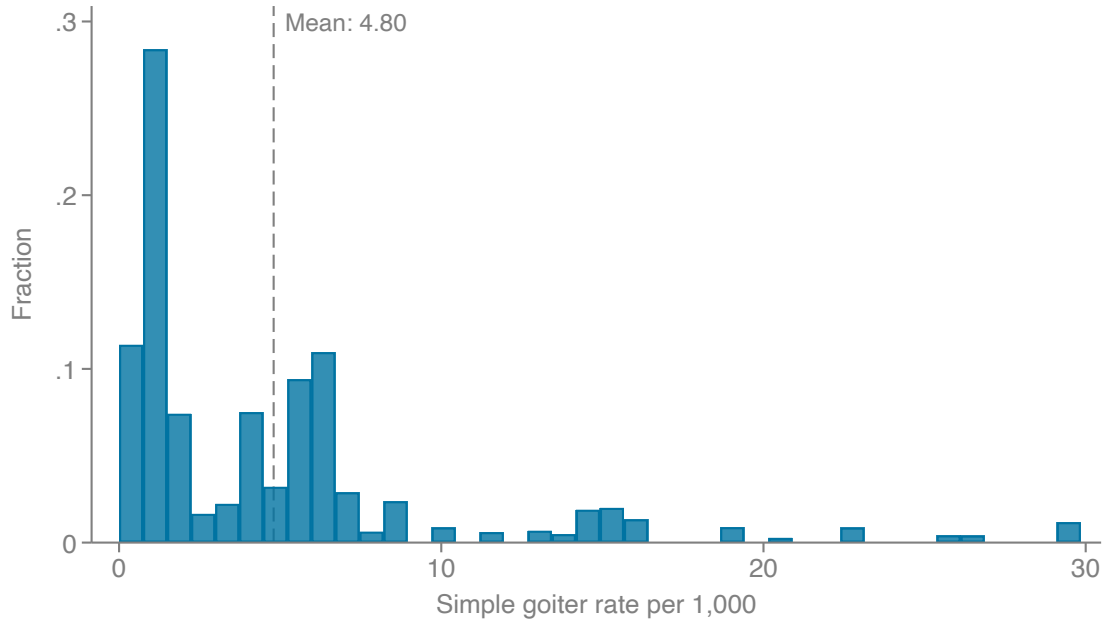


Figure 4: Distribution of Goiter Rates. This graph displays the distribution of simple goiter rates across sections in our estimation sample. Each section is weighted by its 1920 population.

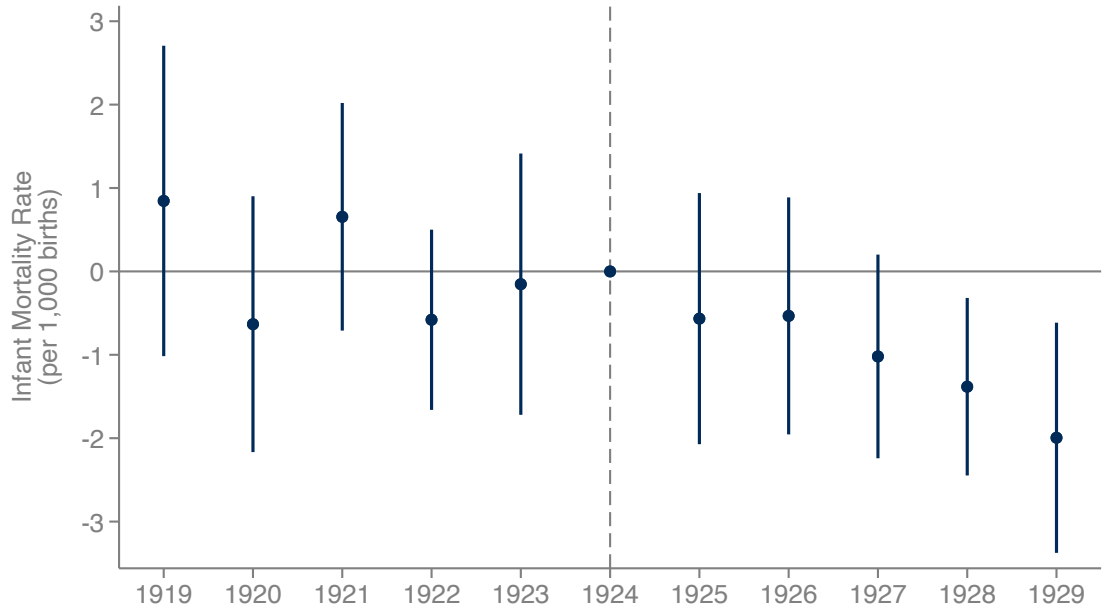


Figure 5: Infant Mortality Rate. This figure displays coefficients from an event study of infant mortality rates on goiter rates. The event study uses the same specification as Column (1) in Table 2, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend.

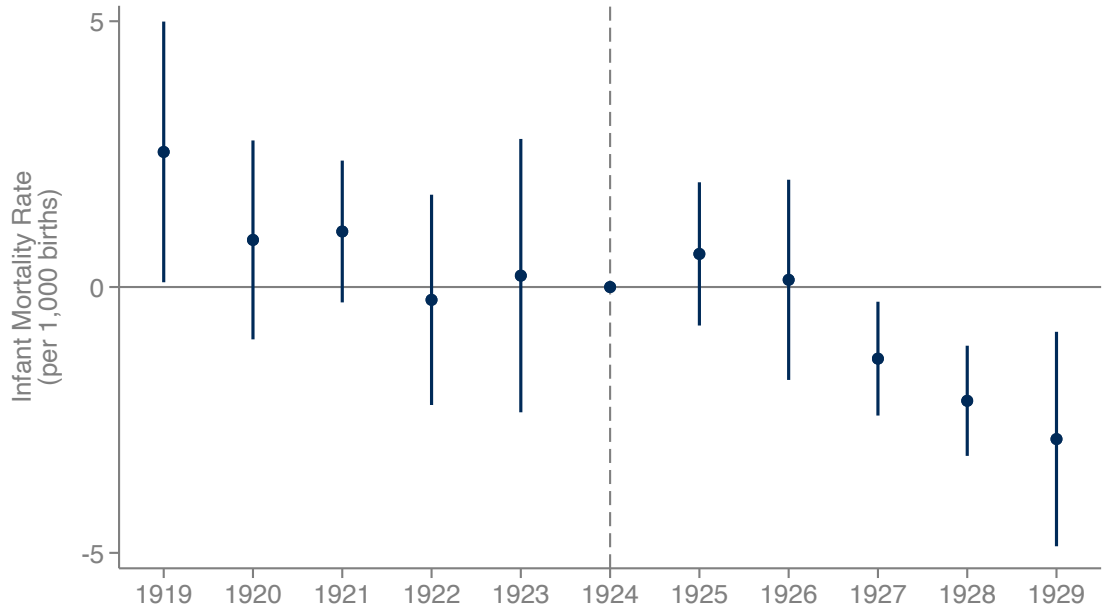


Figure 6: State Level Infant Mortality Rate. This figure displays coefficients from an event study of infant mortality rates on goiter rates. The event study uses the same specification as Column (1) in Table 3, which includes state fixed effects, year fixed effects, region-by-year fixed effects, and 1920 state covariates interacted with a linear time trend.

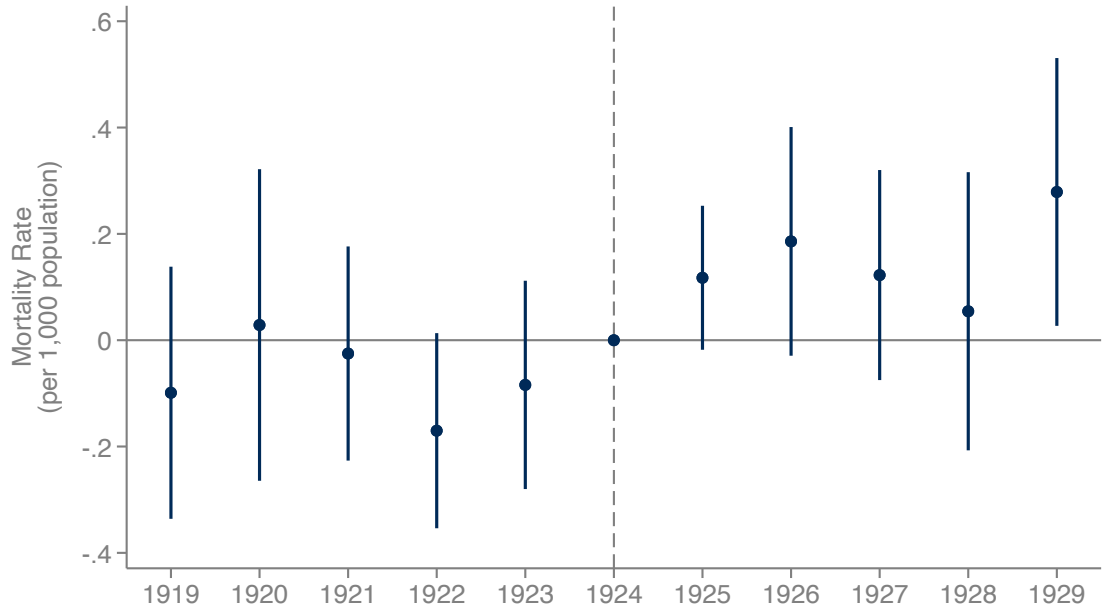


Figure 7: State Level Mortality Rate. This figure displays coefficients from an event study of mortality rates on goiter rates. The event study uses the same specification as Column (2) in Table 3, which includes state fixed effects, year fixed effects, region-by-year fixed effects, and 1920 state covariates interacted with a linear time trend.

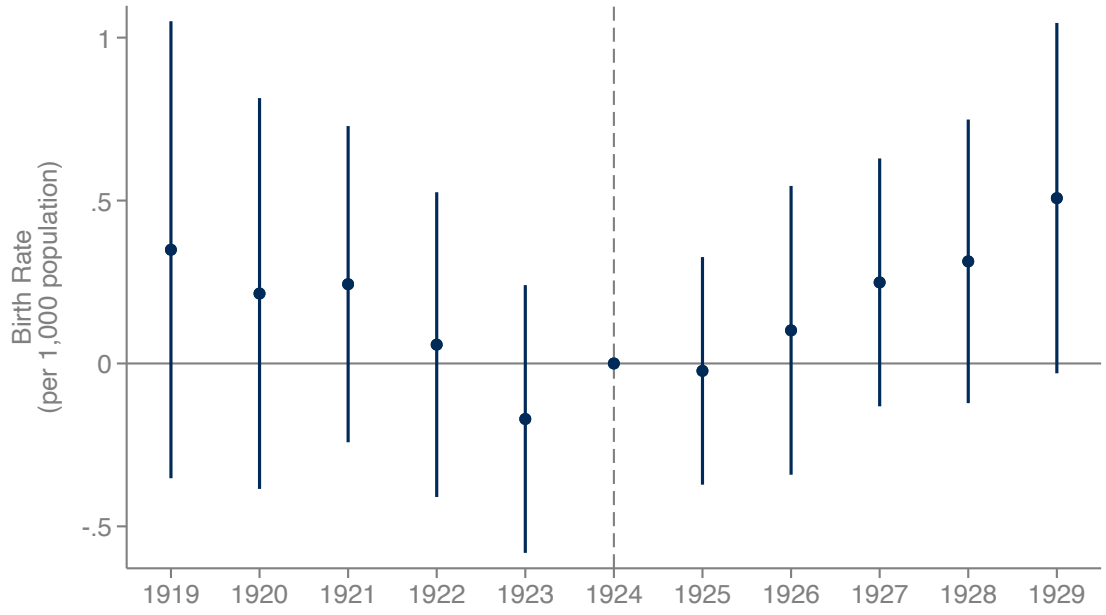


Figure 8: State Level Birth Rate. This figure displays coefficients from an event study of birth rates on goiter rates. The event study uses the same specification as Column (3) in Table 3, which includes state fixed effects, year fixed effects, region-by-year fixed effects, and 1920 state covariates interacted with a linear time trend.

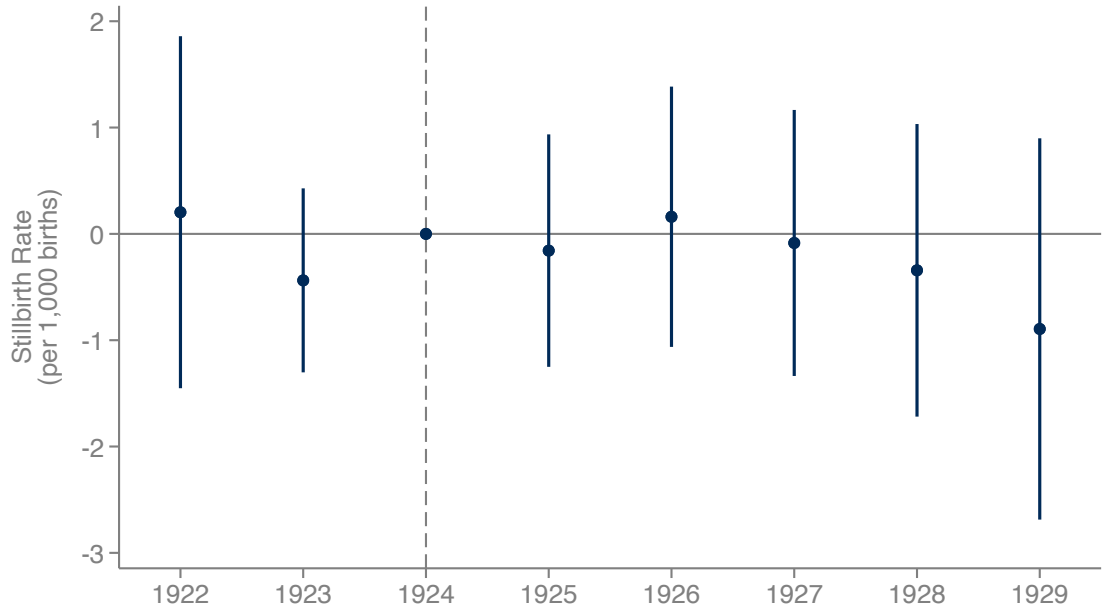


Figure 9: State Level Stillbirth Rate. This figure displays coefficients from an event study of stillbirth rates on goiter rates. The event study uses the same specification as Column (4) in Table 3, which includes state fixed effects, year fixed effects, region-by-year fixed effects, and 1920 state covariates interacted with a linear time trend.

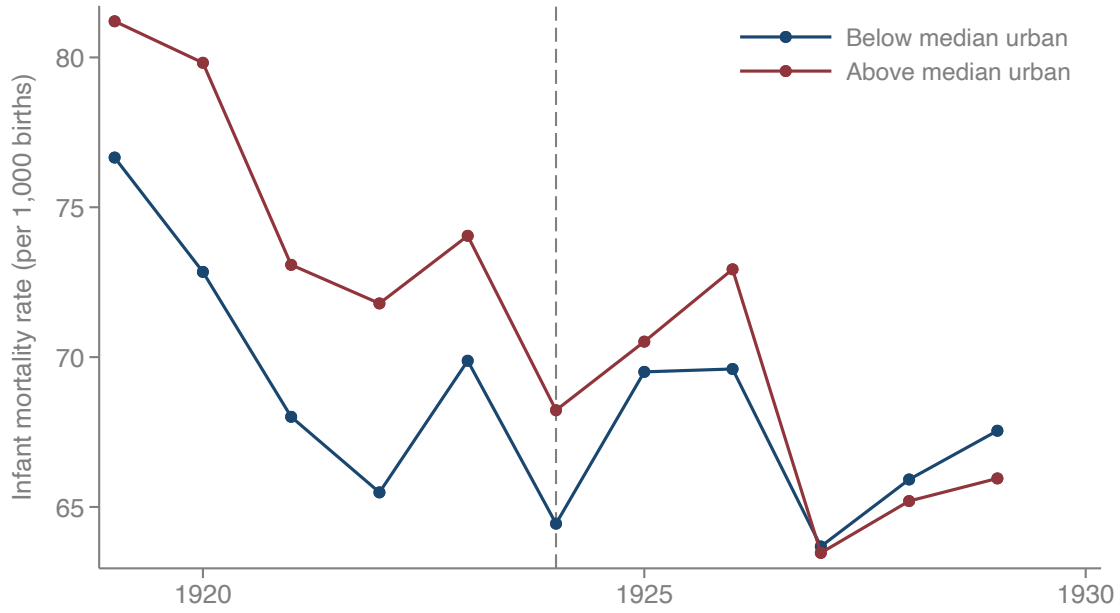


Figure 10: Trends in Urban-Rural Infant Mortality. This figure displays trends in infant mortality between urban and rural counties. Urban and rural status are based on whether or not the percent of the population living in an urban area is above or below the median level of the national county distribution.

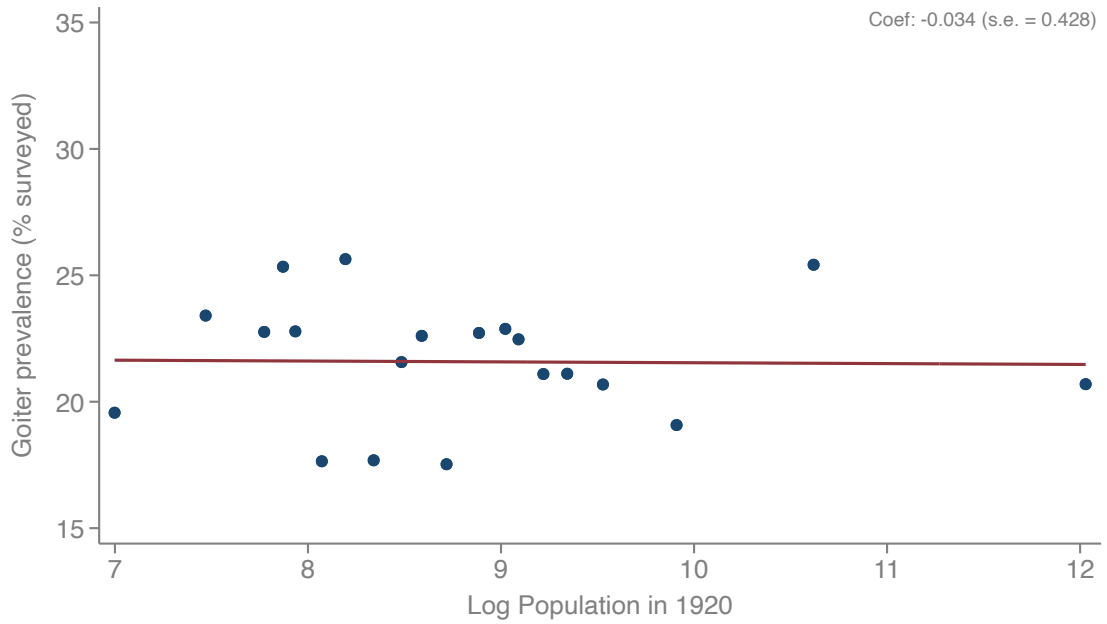


Figure 11: Relationship between Goiter Prevalence and Population. This figure displays a binscatter of the prevalence of goiter on the log population controlling for state fixed effects. The data are based on surveys of goiter prevalence across 130 localities in four states (Massachusetts, Connecticut, Oregon, and Minnesota). The estimated coefficient from the corresponding regression implies that a 10 percent increase in population is associated with 0.0034 percentage point reduction in goiter prevalence. The relationship is not statistically significant.

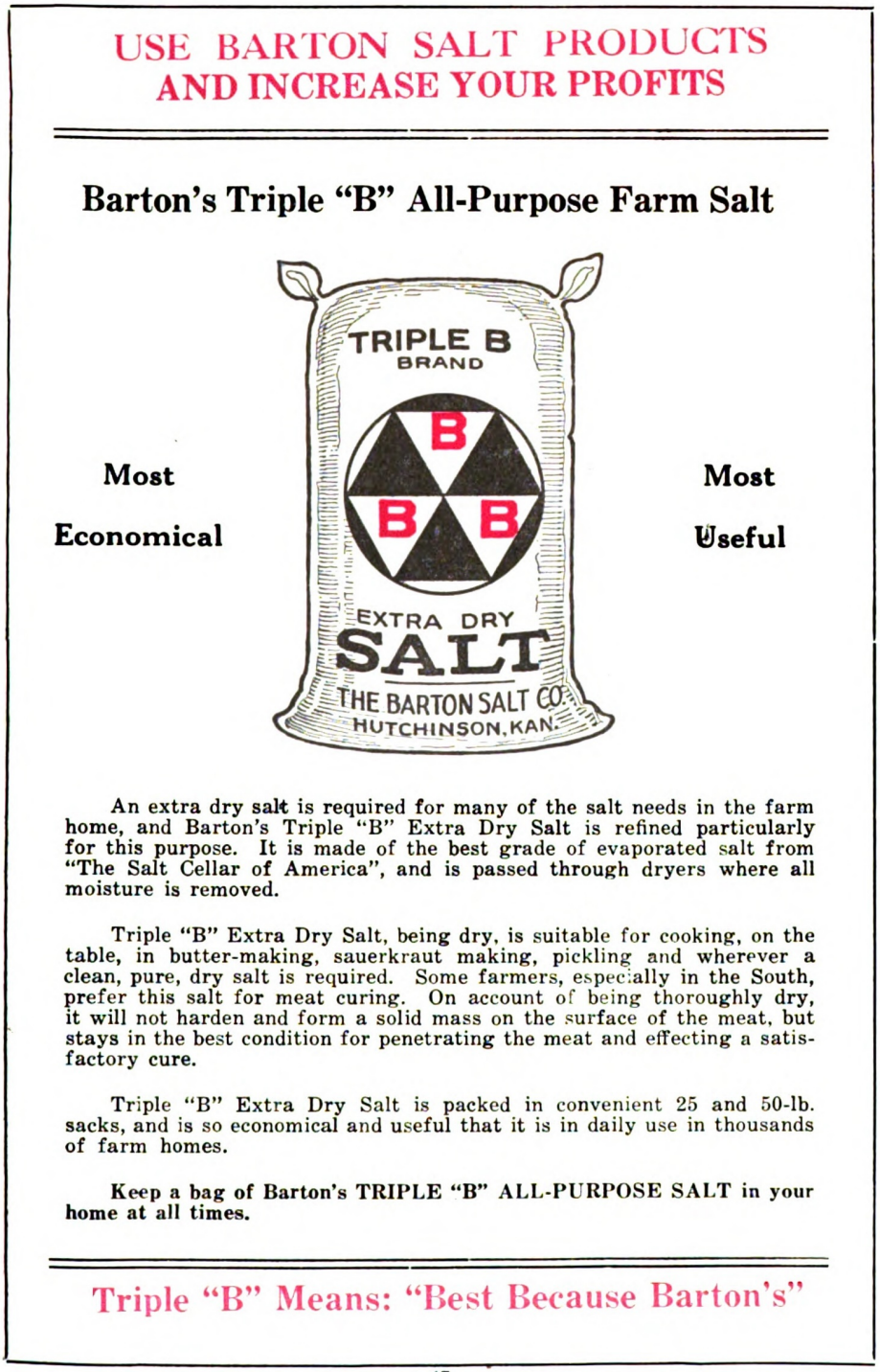


Figure 12: Barton Salt Advertisement. This advertisement displays a 25–50 lb. bag of salt that was typically purchased by farmers for home use. Salt sold in this bulk quantity was typically packaged in cloth bags which were not as effective in keeping out moisture as the smaller tin cans that were used for smaller scale distribution.

A Figures and Tables

A.1 Appendix Tables

Table A1: Long Run Impacts

	In Labor Force	Employed	Worked 40 Weeks	Years Schooling	High School
	(1)	(2)	(3)	(4)	(5)
Goiter $\times \mathbb{1}\{t \in [1920, 1922]\}$	-0.0021	-0.0016	0.0118	-0.0080	-0.0030
	(0.0023)	(0.0024)	(0.0077)	(0.0586)	(0.0070)
Goiter $\times \mathbb{1}\{t \in [1925, 1926]\}$	0.0011	0.0030	-0.0006	-0.0293	-0.0070
	(0.0033)	(0.0031)	(0.0071)	(0.0736)	(0.0085)
Goiter $\times \mathbb{1}\{t \in [1927, 1929]\}$	0.0065***	0.0072***	0.0098	-0.0316	-0.0000
	(0.0022)	(0.0025)	(0.0083)	(0.0570)	(0.0083)
Mean Outcome	0.88	0.84	0.73	10.00	0.48
R-sq	0.03	0.05	0.06	0.05	0.06
Obs.	249,571	247,104	44,030	48,095	49,215

Notes: Standard errors are clustered at the section level.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$

Table A2: Long Run Impacts – Urban

	In Labor Force	Employed	Worked 40 Weeks	Years Schooling	High School
	(1)	(2)	(3)	(4)	(5)
Goiter $\times \mathbb{1}\{t \in [1920, 1922]\}$	-0.0049	-0.0056	0.0103	0.1054	0.0174
	(0.0039)	(0.0045)	(0.0158)	(0.1563)	(0.0221)
Goiter $\times \mathbb{1}\{t \in [1925, 1926]\}$	-0.0024	-0.0062	0.0040	0.0370	-0.0064
	(0.0049)	(0.0039)	(0.0183)	(0.1499)	(0.0234)
Goiter $\times \mathbb{1}\{t \in [1927, 1929]\}$	-0.0044	-0.0051	-0.0204	0.0227	0.0216
	(0.0034)	(0.0052)	(0.0123)	(0.1383)	(0.0174)
Goiter $\times$ Urban $\times \mathbb{1}\{t \in [1920, 1922]\}$	0.0037	0.0050	0.0035	-0.1397	-0.0250
	(0.0036)	(0.0044)	(0.0202)	(0.1637)	(0.0253)
Goiter $\times$ Urban $\times \mathbb{1}\{t \in [1925, 1926]\}$	0.0041	0.0113***	-0.0021	-0.0838	-0.0028
	(0.0050)	(0.0040)	(0.0194)	(0.1811)	(0.0266)
Goiter $\times$ Urban $\times \mathbb{1}\{t \in [1927, 1929]\}$	0.0132***	0.0149**	0.0409**	-0.0695	-0.0266
	(0.0046)	(0.0060)	(0.0162)	(0.1512)	(0.0180)
Mean Outcome	0.88	0.84	0.73	10.00	0.48
R-sq	0.03	0.05	0.06	0.05	0.06
Obs.	249,571	247,104	44,030	48,095	49,215

Notes: Standard errors are clustered at the section level.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$

Table A3: Impact on Infant Mortality Rate — Discrete Treatment

	Infant Mortality Rate			
	(1)	(2)	(3)	(4)
Goiter Tercile (=3)				
$\times \mathbb{1}\{t > 1924\}$	-2.14**	-2.11**	-3.03***	-1.93**
	(0.84)	(0.83)	(0.79)	(0.75)
Mean Outcome	69.93	69.93	69.92	69.93
Division-Year FE		✓		
State-Year FE			✓	
Control Quintile ×				
Year FE				✓
R-sq	0.72	0.72	0.73	0.73
Obs.	13,596	13,596	13,585	13,596

Notes: The outcome variable is the infant mortality rate measured as the number of infant deaths per 1,000 births. The treatment variable is a now an indicator for being in the top tercile of the goiter distribution within a Census division (the omitted group is the first tercile). All columns include year and county fixed effects. Columns (1) – (3) include 1920 county level covariates interacted with a linear time trend. Column (1) is our baseline specification and includes region-by-year fixed effects, column (2) includes division-by-year fixed effects, and column (3) uses state-by-year fixed effects. Column (4) is our baseline specification but controls for county characteristics more flexibly by including covariate quintile-by-year fixed effects for each covariate. The estimation period is 1919-1929. All regressions are weighted by pre-period (1919-1924) average births. Standard errors are clustered at the section level.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$

Table A4: Impact on Mortality Rate

	Mortality Rate			
	(1)	(2)	(3)	(4)
Goiter $\times \mathbb{1}\{t > 1924\}$	0.26***	0.25***	0.04	0.25***
	(0.06)	(0.07)	(0.10)	(0.06)
Mean Outcome	9.91	9.91	9.91	9.91
Division-Year FE		✓		
State-Year FE			✓	
Control Quintile × Year FE				✓
R-sq	0.97	0.97	0.97	0.97
Obs.	13,596	13,596	13,585	13,596

Notes: The outcome variable is the mortality rate measured as the number of non-infant deaths per 1,000 population. The population measure is the linearly interpolated population between Census years. The goiter rate is standardized so that the coefficients represent the estimated effect of a 1 standard deviation increase. All columns include year and county fixed effects. Columns (1) – (3) include 1920 county level covariates interacted with a linear time trend. Column (1) is our baseline specification and includes region-by-year fixed effects, column (2) includes division-by-year fixed effects, and column (3) uses state-by-year fixed effects. Column (4) is our baseline specification but controls for county characteristics more flexibly by including covariate quintile-by-year fixed effects for each covariate. The estimation period is 1919-1929. All regressions are weighted by preperiod (1919-1924) population. Standard errors are clustered at the section level.

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

Table A5: Impact on Birth Rate

	Birth Rate (per 1,000 Females 15-44)			
	(1)	(2)	(3)	(4)
Goiter $\times \mathbb{1}\{t > 1924\}$	1.03**	0.73	-0.49	-0.19
	(0.47)	(0.45)	(1.10)	(0.52)
Mean Outcome	104.82	104.82	104.85	104.82
Division-Year FE		✓		
State-Year FE			✓	
Control Quintile × Year FE				✓
R-sq	0.97	0.97	0.98	0.98
Obs.	13,596	13,596	13,585	13,596

Notes: The outcome variable is the birth rate measured as the number of births per 1,000 female 15-44 population. The population measure is the linearly interpolated population between Census years. The goiter rate is standardized so that the coefficients represent the estimated effect of a 1 standard deviation increase. All columns include year and county fixed effects. Columns (1) – (3) include 1920 county level covariates interacted with a linear time trend. Column (1) is our baseline specification and includes region-by-year fixed effects, column (2) includes division-by-year fixed effects, and column (3) uses state-by-year fixed effects. Column (4) is our baseline specification but controls for county characteristics more flexibly by including covariate quintile-by-year fixed effects for each covariate. The estimation period is 1919-1929. All regressions are weighted by pre-period (1919-1924) average female 15-44 population. Standard errors are clustered at the section level.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$

Table A6: Impact on Log Infant Deaths

	Log Infant Deaths			
	(1)	(2)	(3)	(4)
Goiter $\times \mathbb{1}\{t > 1924\}$	-0.03*	-0.04**	-0.06***	-0.04**
	(0.02)	(0.02)	(0.02)	(0.02)
Mean Outcome	3.56	3.56	3.56	3.56
Division-Year FE		✓		
State-Year FE			✓	
Control Quintile × Year FE				✓
R-sq	0.985	0.985	0.985	0.985
Obs.	13,562	13,562	13,551	13,562

Notes: The outcome variable in columns (1) – (4) is the number of infant deaths. The outcome variable in columns (5) – (8) is the log of the number of infant deaths. The goiter rate is standardized so that the coefficients represent the estimated effect of a 1 standard deviation increase. All columns include year and county fixed effects. Columns (1) – (3) and (5) – (7) include 1920 county level covariates interacted with a linear time trend. Columns (1) and (5) are our baseline specification and includes region-by-year fixed effects, columns (2) and (6) include division-by-year fixed effects, and columns (3) and (7) use state-by-year fixed effects. Columns (4) and (8) are our baseline specification but controls for county characteristics more flexibly by including covariate quintile-by-year fixed effects for each covariate. The estimation period is 1919-1929. All regressions are weighted by pre-period (1919-1924) births. Standard errors are clustered at the section level.

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

Table A7: Impact on Log Deaths

	Log Deaths			
	(1)	(2)	(3)	(4)
Goiter $\times \mathbb{1}\{t > 1924\}$	0.01**	0.01	0.02*	0.02***
	(0.00)	(0.01)	(0.01)	(0.01)
Mean Outcome	5.38	5.43	5.43	5.43
Division-Year FE		✓		
State-Year FE			✓	
Control Quintile × Year FE				✓
R-sq	0.996	0.997	0.997	0.997
Obs.	13,976	13,595	13,584	13,595

Notes: The outcome variable in columns (1) – (4) is the number of non-infant deaths. The outcome variable in columns (5) – (8) is the log of the number of non-infant deaths. The goiter rate is standardized so that the coefficients represent the estimated effect of a 1 standard deviation increase. All columns include year and county fixed effects. Columns (1) – (3) and (5) – (7) include 1920 county level covariates interacted with a linear time trend. Columns (1) and (5) are our baseline specification and includes region-by-year fixed effects, columns (2) and (6) include division-by-year fixed effects, and columns (3) and (7) use state-by-year fixed effects. Columns (4) and (8) are our baseline specification but controls for county characteristics more flexibly by including covariate quintile-by-year fixed effects for each covariate. The estimation period is 1919-1929. All regressions are weighted by pre-period (1919-1924) population. Standard errors are clustered at the section level.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$

Table A8: Impact on Log Births

	Log Births			
	(1)	(2)	(3)	(4)
Goiter $\times \mathbb{1}\{t > 1924\}$	-0.02	-0.02	-0.03*	-0.01
	(0.01)	(0.01)	(0.02)	(0.01)
Mean Outcome	6.21	6.26	6.26	6.26
Division-Year FE		✓		
State-Year FE			✓	
Control Quintile × Year FE				✓
R-sq	0.996	0.996	0.996	0.996
Obs.	13,983	13,596	13,585	13,596

Notes: The outcome variable in columns (1) – (4) is the number of births. The outcome variable in columns (5) – (8) is the log of the number of births. The goiter rate is standardized so that the coefficients represent the estimated effect of a 1 standard deviation increase. All columns include year and county fixed effects. Columns (1) – (3) and (5) – (7) include 1920 county level covariates interacted with a linear time trend. Columns (1) and (5) are our baseline specification and includes region-by-year fixed effects, columns (2) and (6) include division-by-year fixed effects, and columns (3) and (7) use state-by-year fixed effects. Columns (4) and (8) are our baseline specification but controls for county characteristics more flexibly by including covariate quintile-by-year fixed effects for each covariate. The estimation period is 1919-1929. All regressions are weighted by pre-period (1919-1924) female 15-44 population. Standard errors are clustered at the section level.

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

A.2 Appendix Figures

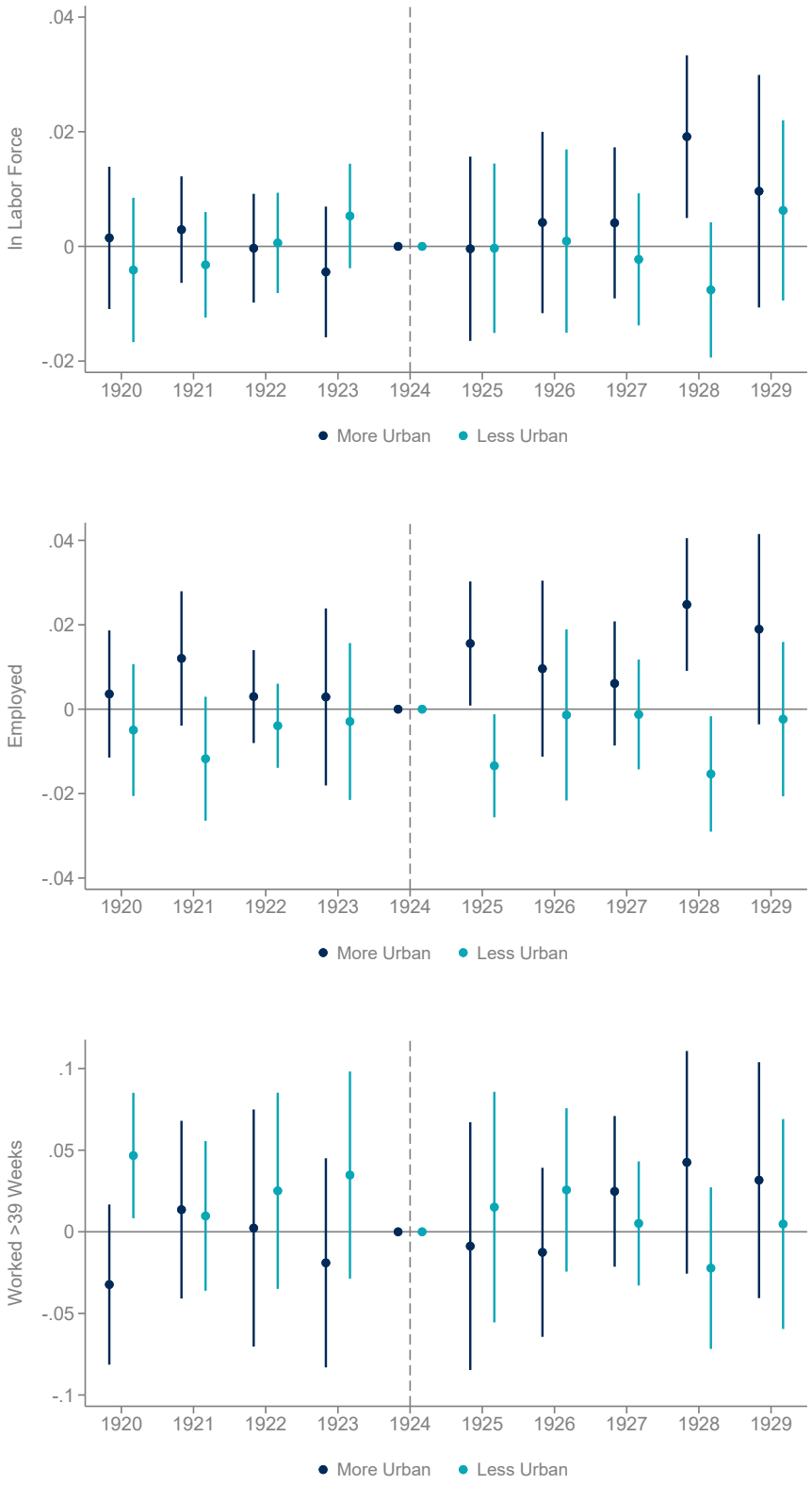


Figure A1: TEMP.

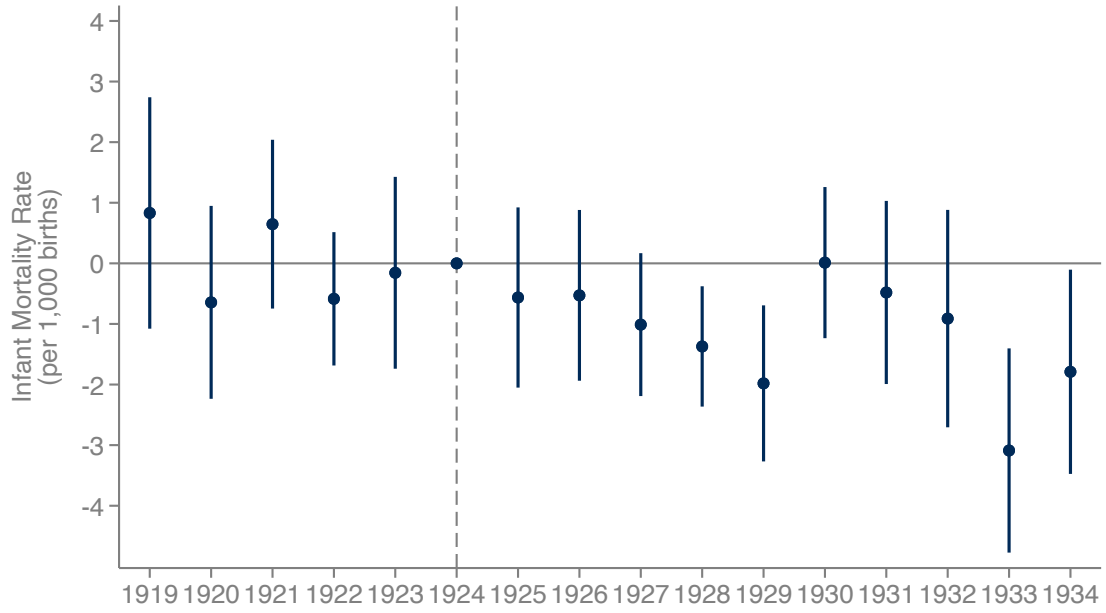


Figure A2: Infant Mortality Rate All Years. This figure displays coefficients from an event study of infant mortality rates on goiter rates. The event study uses the same specification as Column (1) in Table A5, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend. The estimation period is 1919 – 1934.

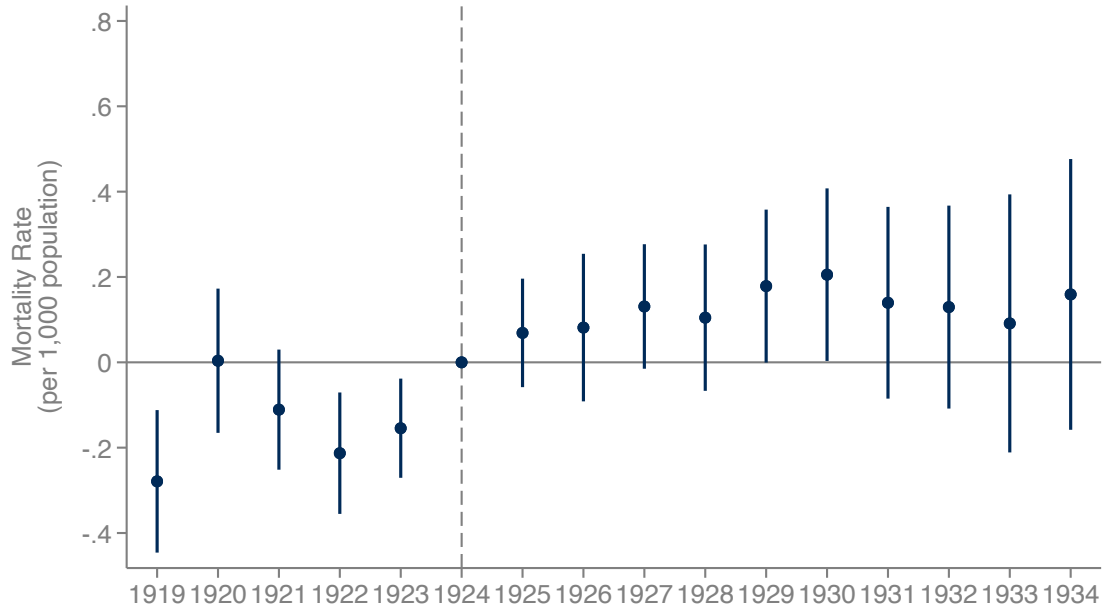


Figure A3: Mortality Rate All Years. This figure displays coefficients from an event study of mortality rates on goiter rates. The event study uses the same specification as Column (1) in Table A4, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend. The estimation period is 1919 – 1934.

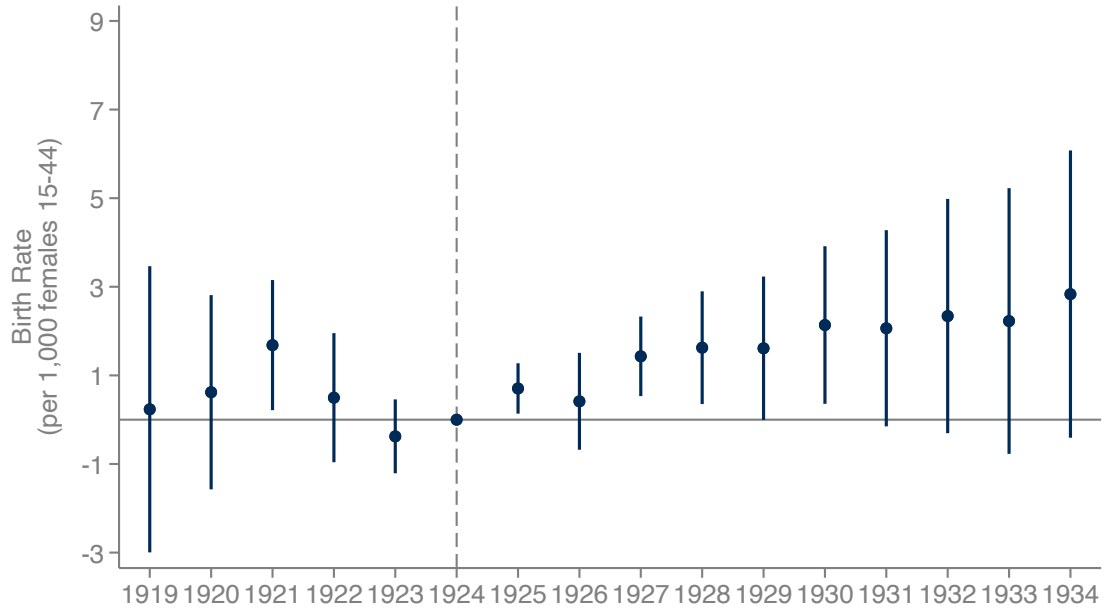


Figure A4: Birth Rate All Years. This figure displays coefficients from an event study of infant mortality rates on goiter rates. The event study uses the same specification as Column (1) in Table A5, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend. The estimation period is 1919 – 1934.

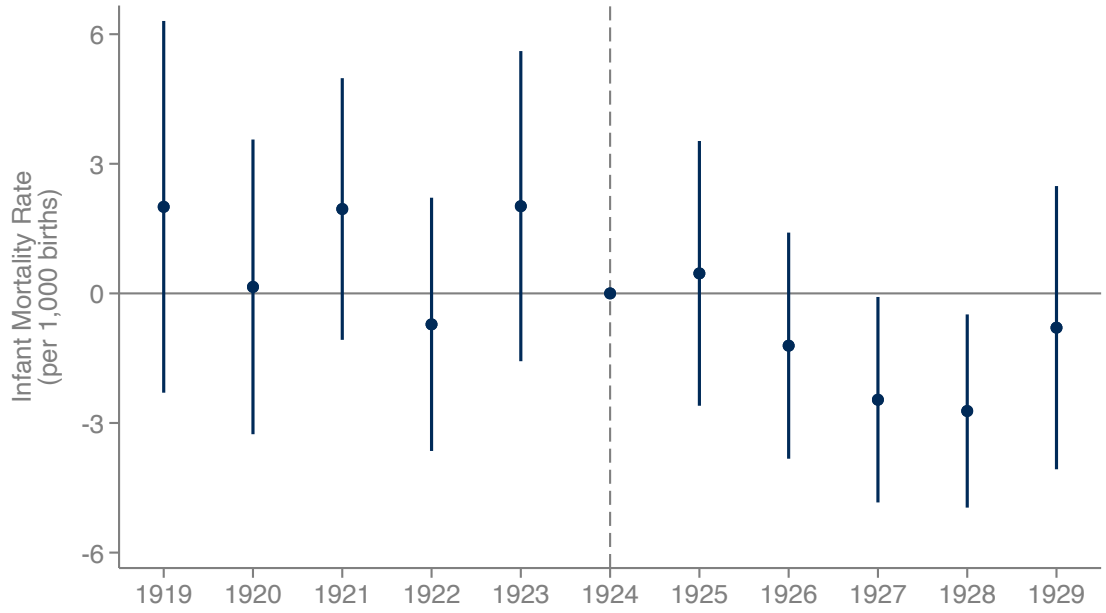


Figure A5: Infant Mortality Rate Discrete Treatment. This figure displays coefficients from an event study of infant mortality rates on an indicator for being in the top tercile of the within-Census division goiter distribution. The event study uses the same specification as Column (1) in Table A3, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend.

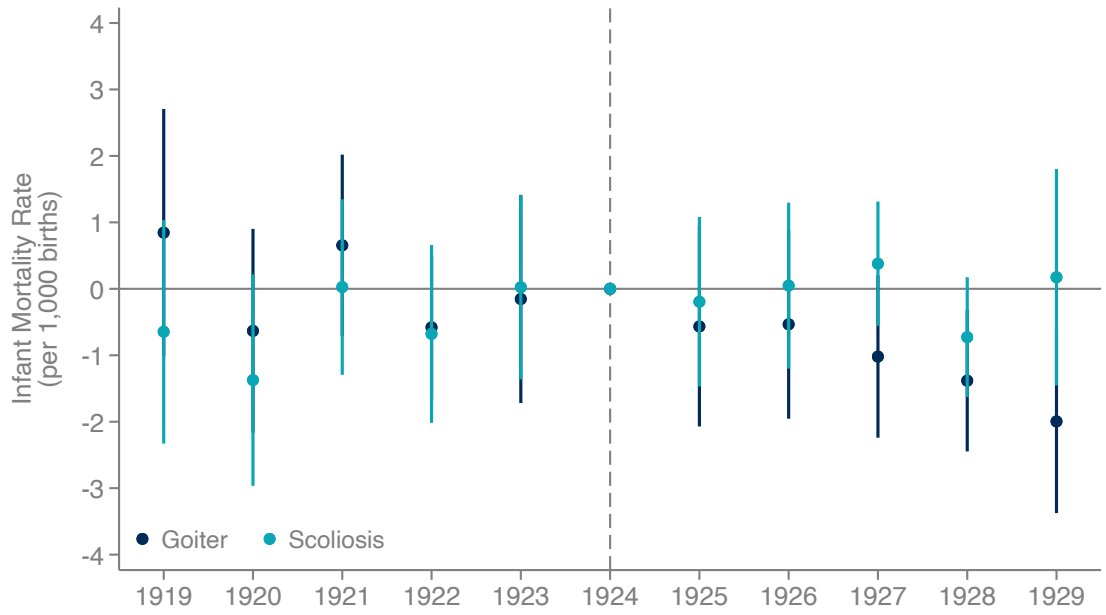


Figure A6: Infant Mortality Rate Scoliosis Placebo. This figure displays coefficients from an event study of infant mortality rates on scoliosis rates. The event study uses the same specification as Column (1) in Table 2, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend.

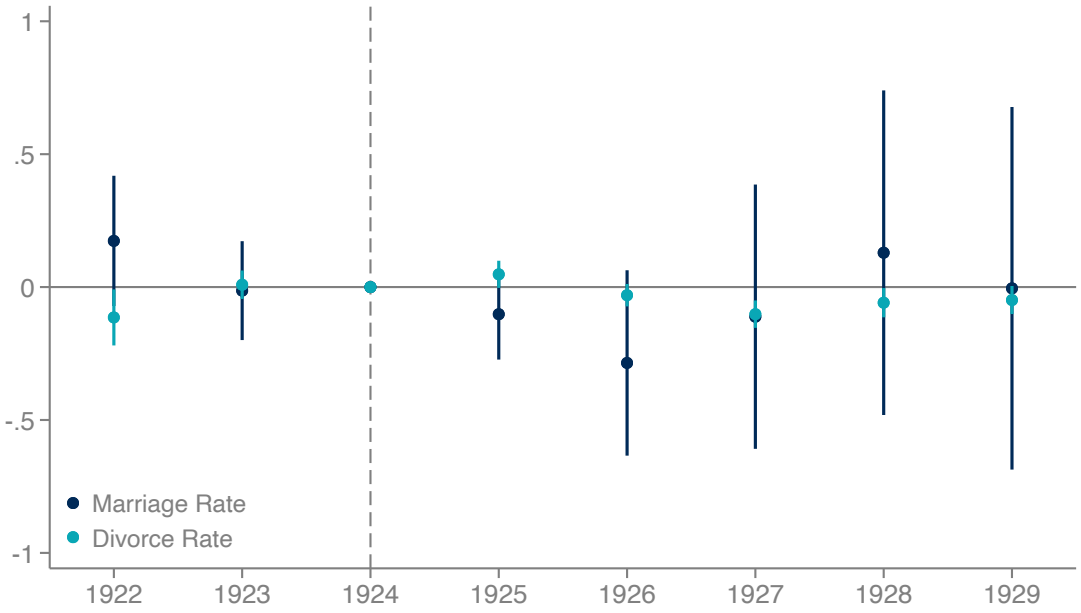


Figure A7: Marriage and Divorce Placebo. This figure displays coefficients from an event study of marriage and divorce rates on goiter rates. The event study uses the same specification as Column (1) in Table 2, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend.

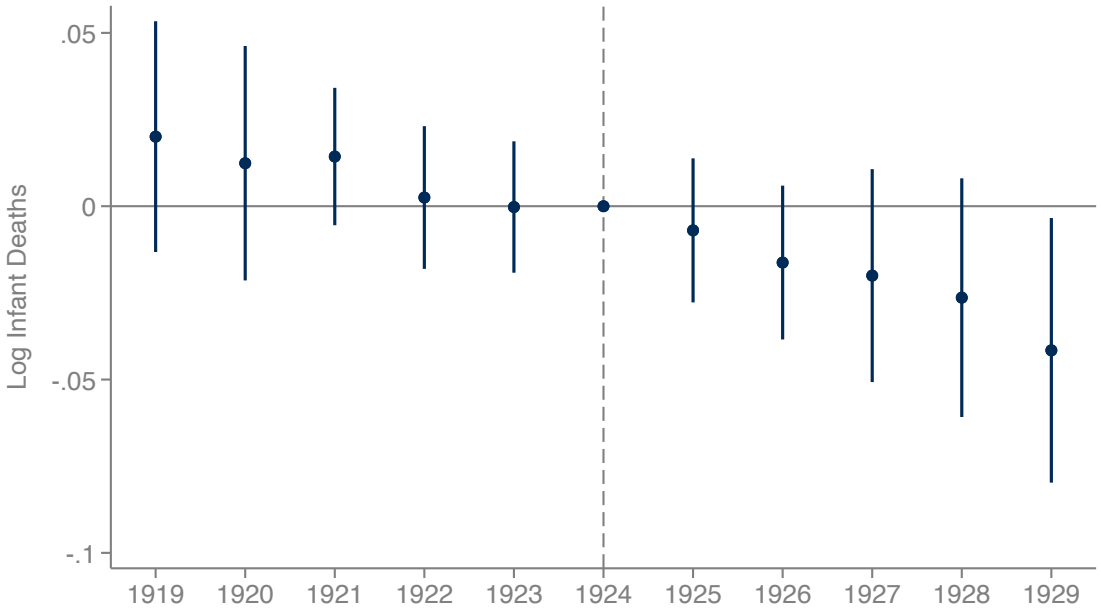


Figure A8: Impact on Log Infant Deaths. This figure displays coefficients from an event study of log deaths on goiter rates. The event study uses the same specification as Column (4) in Table A6, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend.

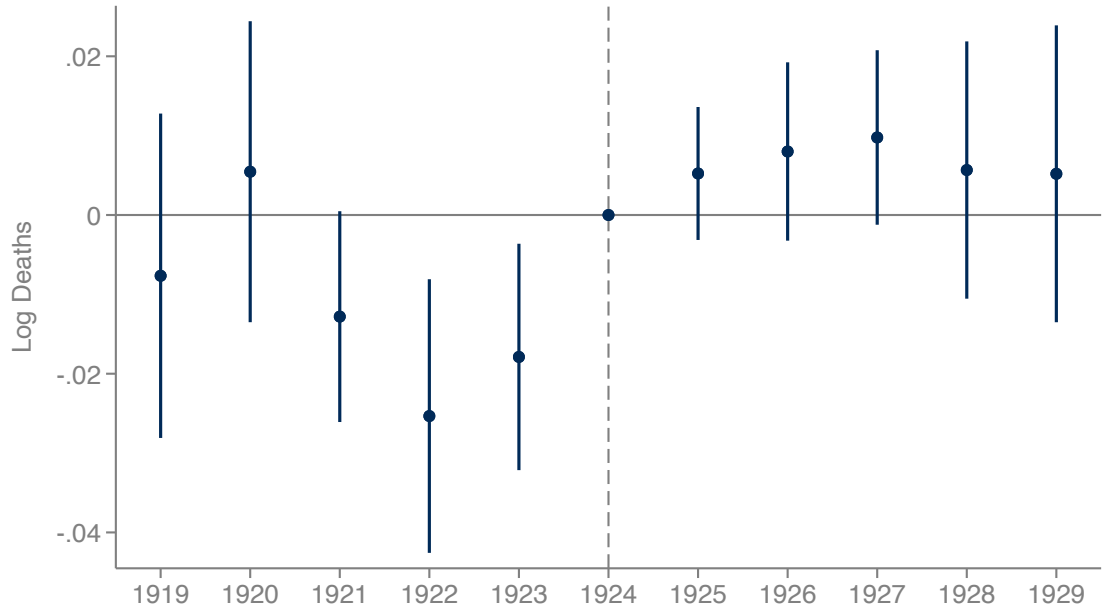


Figure A9: Impact on Log Deaths. This figure displays coefficients from an event study of log deaths on goiter rates. The event study uses the same specification as Column (4) in Table A7, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend.

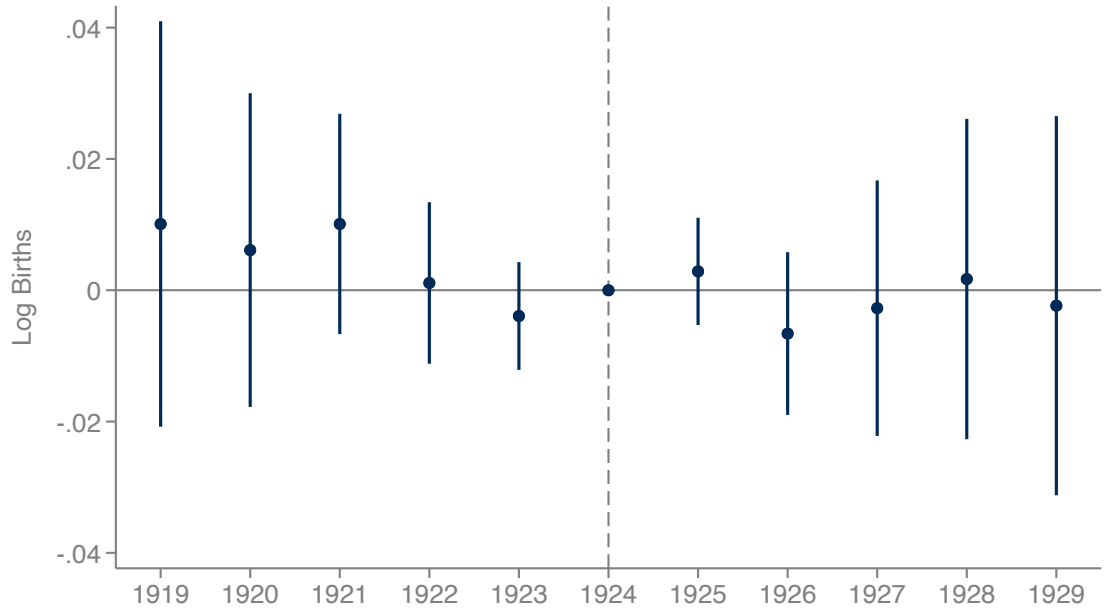


Figure A10: Impact on Log Births. This figure displays coefficients from an event study of log deaths on goiter rates. The event study uses the same specification as Column (4) in Table A8, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend.

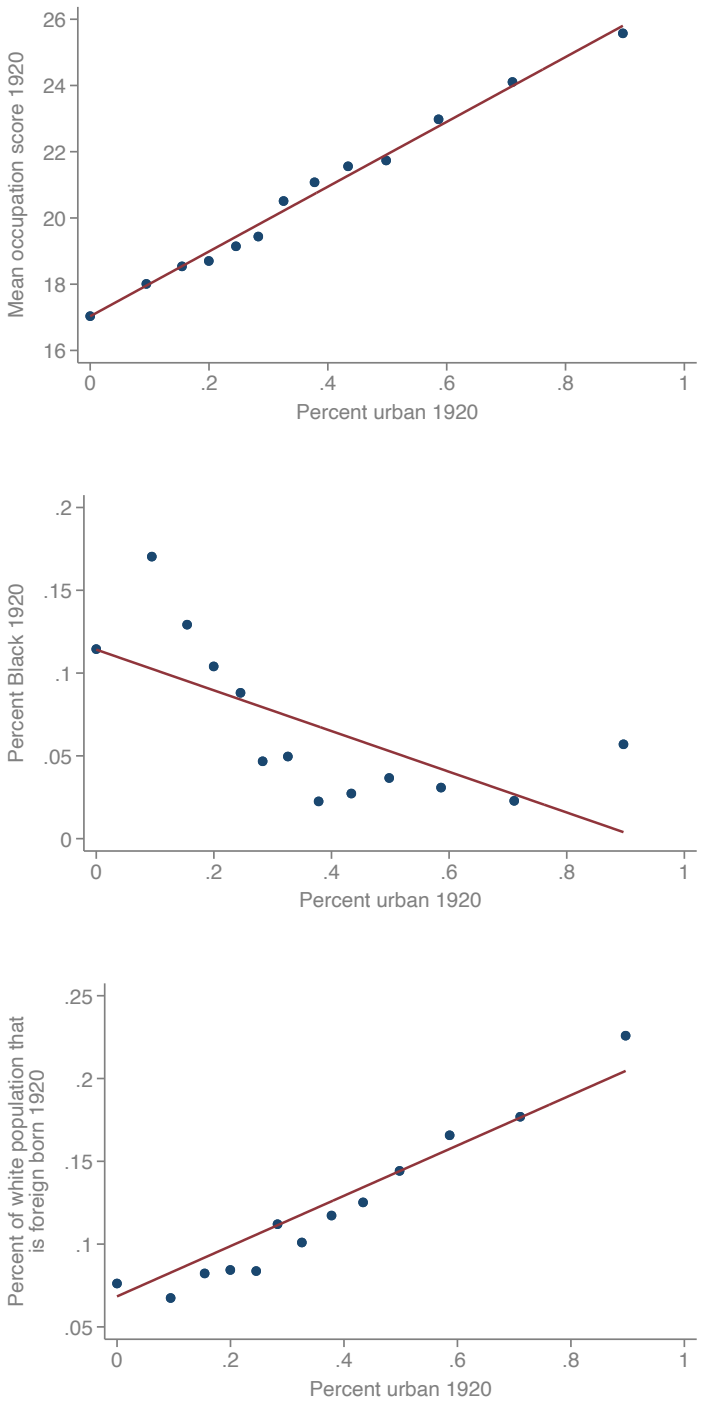


Figure A11: Correlates of Share Urban in 1920. This figure displays the relationship between the share of the population living in urban areas and other covariates in 1920 at the county level for the counties in our sample.