Adding Salt to the Womb: The Benefits of Salt Iodization from Infancy to Adulthood*

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Abstract

We estimate the effects of the 1924 introduction of iodized salt in the U.S. by exploiting pre-1924 geographic variation in iodine deficiency. Iodized salt reduced infant mortality by 1.1 deaths per 1,000 births (1.6%) for counties at the 75th percentile of iodine deficiency relative to the 25th percentile. These effects are concentrated in urban counties—where iodized salt was disproportionately available—explaining 1/3 of the decline in the urban-rural infant mortality gap in the 1920s. We show that the long-term effects on labor market outcomes are consistently large among urban-born individuals, reconciling conflicting results in the literature.

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1 Introduction

At the turn of the 20th century, one in six infants born in a city in the United States would not live until their first birthday. Infant mortality was high throughout the country during this period, but babies born in rural areas were 32 percent more likely to survive infancy. By 1930, urban infant mortality had fallen by 65 percent, representing a complete convergence in the mortality gap between infants born in cities and infants born in rural America. This drop in infant mortality in cities is a major factor in what is known as the "urban mortality transition," wherein urban mortality dropped broadly relative to rural mortality during the first half of the 20th century (Haines, 2001).

Economists and demographers have put forward several explanations for the urban mortality transition, with particular focus given to public health efforts to reduce food- and water-borne illness. Recent research has shown, however, that these efforts explain very little of the convergence between urban and rural mortality (Anderson et al., 2022). In fact, Anderson et al. (2022) find that municipal-level public health interventions can explain, at most, 10% of the decline in urban infant mortality between 1900 and 1940. On the other hand, changes in the nutritional content of food—particularly access to essential micronutrients such as iodine—have received relatively little attention in this literature, despite the importance of micronutrient intake for fetal development and infant survival (Zimmermann, 2011).

Because infant mortality accounted for a large share of deaths during this period, improvements in infant health were a major factor in the broader convergence in urban and rural mortality. In 1924, iodine was introduced to table salt in the United States, but access to

stabilized iodized salt—and the resulting improvement in iodine intake—was concentrated in urban areas. We use this natural experiment to study the effect of nationwide salt iodization on infant health outcomes and on urban–rural disparities in infant health.

Since iodine consumption is not randomly assigned across individuals, we study the effects of the nationwide adoption of iodized salt using 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).[1] Since iodine deficiency was historically the primary cause of goiter—an enlargement of the thyroid gland—variation in goiter incidence reflects variation in iodine deficiency.

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 find no evidence that our estimated effects are driven by changes in health that are unrelated to iodization or changes in the composition of births.

We further document that the aggregate reductions in infant mortality following iodiza-

[1]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. By 1945, the prevalence of goiter fell by around 90 percent based on comparisons of medical examinations of draft age men from WWI and WWII. This reduction is widely attributed to the introduction of iodized table salt in 1924 (Schiel and Wepfer, 1976).

tion are driven by reductions in urban areas, where the iodized salt was 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 in our sample.[2]

Although iodized salt improved infant survival, prior research suggests that rapid increases in iodine exposure may have posed short-term health risks for iodine-deficient adults. For instance, medical evidence dating back to the early introduction of iodized salt shows that rapid increases in iodine intake among iodine-deficient adults can disrupt thyroid function, a phenomenon known as iodine-induced hyperthyroidism (McClure, 1927, 1934). Consistent with this mechanism, Feyrer et al. (2017) document a temporary increase in thyroid-related deaths at the national level following iodization and provide suggestive evidence on its mortality consequences. We build on this work by providing direct empirical estimates of the mortality effects of salt iodization, finding a short-term increase in thyroid-related mortality in areas with higher iodine deficiency. We then quantify the resulting trade-off across age groups by showing that reductions in infant deaths offset the increase in adult deaths.[3]

The reductions in infant mortality we document imply meaningful improvements in early-life health for urban-born cohorts, raising the question of the long-run economic consequences of salt iodization. While prior work has examined the long-term effects of salt iodization, it does not account for the urban–rural differences in treatment exposure that we document, motivating a re-evaluation of these effects. The existing literature on the long-term effects

[2]The infant mortality rate in our sample closed by 8.5 infant deaths per 1,000 births between 1920 and 1929, which is around 16% (8.5 / 52.5) of the total convergence that occurred between 1900 and 1930 based on a sample of seven registration states where data are available for the entire period (Haines, 2001).

[3]In the era of modern medicine, increased monitoring of thyroid hormone levels of adults in historically iodine deficient areas may be appropriate.

of salt iodization in the United States has been mixed. For instance, Adhvaryu et al. (2020) find no long-run effects of salt iodization on the labor market outcomes of men, while Feyrer et al. (2017) find large effects on the cognitive ability of World War II enlistees.

The lack of measured improvement in men's labor market outcomes is puzzling given the expected relationship between childhood health, human capital, and labor market outcomes. We re-evaluate the long-term effects of salt iodization on labor market outcomes by accounting for the urban–rural differences in treatment exposure that prior work does not capture. Using 1950 Census data linked to the Social Security Numident file, we build on the analyses of Adhvaryu et al. (2020) by defining treatment at the sub-state level and evaluating urban-rural differences in treatment. This analysis reveals two important findings. First, the null results on labor market outcomes Adhvaryu et al. (2020) find for men may be driven by a lack of statistical power using state-level variation, and positive employment effects emerge when using sub-state variation.[4] Second, these positive long-run effects are driven by improved outcomes for men born in urban counties, which is consistent with our short-term effects on infant mortality. We further show that the positive effects on cognitive ability found in Feyrer et al. (2017) are larger for men from urban counties. Taken together, these findings demonstrate the important role of iodized salt in explaining the diverging outcomes for children born in urban versus rural counties in the 1920s from birth to adulthood.

Today, 145 countries have some legislation regarding salt iodization and around 88 percent of the world's population uses iodized salt. In Nigeria, the first African country to achieve national iodine sufficiency, a back-of-the-envelope calculation using our estimates suggest that infant mortality could have been up to 24% higher in the absence of their

[4]We can identify county of birth using the Numident.

iodine mandate.[5] Despite these improvements, however, iodine deficiency remains prevalent in many countries around the globe due to inadequate dietary supply (Zimmermann and Andersson, 2021). Today, iodine deficiency still affects 1.88 billion people worldwide, including 241 million children (Andersson et al., 2012). Even some high-income countries continue to suffer from iodine deficiency. As of 2018, half of German children were iodine deficient. Despite these troubling numbers, iodine consumption in Germany has fallen in recent years—even as salt consumption has risen—as awareness of the importance of iodine for health and human capital has diminished (Remer et al., 2022). 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. Notably, many of the low-income countries where iodine deficiency is most common today are also geographical areas with high heat and humidity, so additional care is needed to ensure that iodized salt that is distributed as part of global health efforts remains stable.

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 introduction of iodized table salt in the United States. Section 3 describes the data used in the paper. Section 4 discusses our empirical strategy in further detail. Section 5 presents our infant mortality results as well as robustness and placebo exercises. Section 6 presents our

[5]Nigeria had a total goiter rate (TGR) of 20% prior to iodization (WHO, 1996). This is around two thirds of the rate in Michigan which was 38% prior to iodization (Schiel and Wepfer, 1976). The simple goiter rate in Michigan based on WWI medical examinations was 11.4 per 1,000 men. The implied reduction in infant mortality at this level of goiter from our estimates is $0.259 \times 11.4 = 3.59$ fewer deaths. Re-scaling this based on the ratio of goiter prevalence between Michigan and Nigeria implies that iodization reduced infant mortality by 1.9 deaths per 1,000 births. Infant mortality fell by 8 deaths per 1,000 births in Nigeria five years following salt iodization in 1993.

findings on the short-term increase in adult mortality from the rapid reintroduction of iodine. Section 7 describes why we would expect to see the effects of salt iodization concentrated in urban areas and presents results on the heterogeneous treatment effects of salt iodization on infant mortality and long-run outcomes. 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 impair the cognitive development and survival of children. During the perinatal, neonatal, and early childhood periods, insufficient iodine intake can lead to hypothyroidism and other health issues. During pregnancy, serious iodine deficiency can lead to prematurity, congenital abnormalities, and stillbirth (Zimmermann, 2011). 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— was historically 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. This relationship has been documented in the medical literature, which finds a positive association between the degree of iodine deficiency and the size of the thyroid (Carle et al., 2014). 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).

Building on this relationship, we characterize the underlying severity of iodine deficiency using available measures of goiter prevalence. In populations with moderate to severe deficiency, we would expect a higher share of individuals with notable thyroid enlargements. For the median (80th percentile) state in the WWI examinations data, corresponding health surveys among school-age children indicate a goiter prevalence of 18% (56%).[6] These levels are characteristic of populations with moderate (severe) iodine deficiency (Carle et al., 2014). This suggests that meaningful, population-wide iodine deficiency remained prevalent in many parts of the U.S. prior to iodization, implying substantial potential gains from the intervention.

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 1 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 in 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 in Michigan and was estimated to affect around 6 percent of men living in Western states (Schiel and Wepfer, 1976).

[6]The median (mean) state roughly corresponds to Tennessee (Colorado). Goiter rates among school children are tabulated from (Olesen, 1926, 1929).

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 (Markel, 1987). 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

3.1 Health Outcomes

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 (groups 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.

The goiter rates tabulated from the WWI draft examinations are typically lower than those measured in contemporaneous health surveys. For example, a survey by the Colorado State Public Heath Service reported a prevalence of 26.5% among school-aged boys, whereas the prevalence reported in the WWI examinations was only 0.9% (Love and Davenport, 1920). The lower rates are likely due to the fact that these medical examinations were focused on identifying defects relevant for military classification rather than on measuring morbidity in the general population (Love and Davenport, 1920).[7] For instance, the Physical Examination Standards explicitly stated that "simple goiter, when sufficiently severe to interfere with the wearing of military collar" was a cause for rejection (Love and Davenport, 1920). As a result, many clinical cases of mild goiter that were not disqualifying may not have been recorded.[8] Although the absolute levels of goiter reported in the WWI examinations may be under-reported, the geographic variation across places closely resembles that found in health surveys from the same period (Olesen, 1926).

Our primary outcome of interest is the infant mortality rate (IMR). To measure our outcomes at the county-level we use birth and infant death data compiled by Bailey et al.

[7]Several other factors may have contributed to these lower rates, including the speed of draft medical examinations and the varying degrees of experience of military medical examiners in diagnosing thyroid disorders (Olesen, 1929).

[8]This interpretation is consistent with survey-based tabulations of goiter severity. For instance, the goiter rate in Colorado excluding cases classified as "slight" is 1.5%, which aligns much more closely with the WWI figures (Olesen, 1929).

(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 annual county-level vital events for 1,273 counties across 24 states, which account for 58% of the U.S. population in 1920. 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).

Fortunately, our primary outcome of interest, the infant mortality rate, is likely one of the more reliably measured health statics at the county-level during this period. This is because births and infant deaths are, in theory, more reliably recorded than population counts through the issuance of birth and death certifications which facilitates the tabulation of the IMR.[9] In order to calculate 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, our measures of annual birth rates may be subject to measurement error. To address this, we directly use reported birth rates at the state-level, 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 intercensal data we found covering our time period. These data come from "Vital Statistics Rates in the

[9]There are known issues with respect to the under-registration of births especially during this period (Eriksson et al., 2018), but we have no reason to suspect this would systematically vary by pre-1924 goiter rates.

United States 1900–1940" published by the United States Public Health Service (USPHS) and are available for 34 states for the years in our estimation period. To further address issues with the denominator, we supplement our analysis with counts of infant deaths and births as alternative population-agnostic measures at the county level.

For additional robustness analysis, we also digitize state-level deaths by cause of death and age at death between 1920–1928. These outcomes are taken from the Census Bureau's annual "Mortality Statistics" publication. We use these data to separately look at the impacts of salt iodization on mortality from causes of deaths that are potentially related to iodine deficiency. We also use these data to further quantify the potential adverse effects of iodization on adult mortality from thyroid-related causes described in Feyrer et al. (2017). Using a narrow window of data, Feyrer et al. (2017) provide descriptive evidence that the increase in thyroid-related deaths is the largest for the elderly. Unfortunately, the age groups used to record deaths change over the larger window we use to evaluate pre-trends. Therefore, we focus on deaths over 30 due to exophthalmic goiter as our primary measure of adult mortality from thyroid-related causes.[10]

Finally, we use data from the 1920 Census of Population, Agriculture, and Manufacturing schedules from the IPUMS National Historical Geographic Information System (NHGIS) (Manson et al., 2024). 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.

[10]This category includes deaths due to "Basedow's disease, Exophthalmic cachexia goitre, Graves's disease, insanity of Graves's disease, Parry's disease, Stokes's disease" — all of which are terms for Graves's disease or its complications.

3.2 Labor Market Outcomes

To study long-run labor market outcomes, we use the 1950 full count decennial Census (Ruggles et al., 2024) to observe outcomes for our cohorts which are between the ages of 21–30 in 1950. We construct three labor market outcomes of interest. The first is an indicator for whether or not an individual is participating in the labor force (either employed or unemployed and actively looking for work). The second outcome we study is employment which is equal to the labor force indicator except that it is also set to zero for individuals that are currently unemployed. Finally, we examine the intensive margin of labor supply using the number of weeks the individual worked in the prior year. Specifically, we construct an indicator for whether or not the individual worked at least 40 weeks in the previous twelve months conditional on working at least one week.[11]

To observe the section (groups of counties) of birth we link the 1950 Census to the National Archive's public version of the Social Security Numident file (NARA Numident), which contains county of birth. We obtain section of birth from the county of birth using the crosswalk provided by Love and Davenport (1920). The Numident file is a major component of the Social Security's Administrative Records System, a subset of which was transferred to NARA. The Numident contains information on date of death, date of birth, birthplace, as well as other background information for individuals that died between 1988 and 2005 (see Appendix A.1 for more details). We link the Numident to the 1950 full count decennial Census where we observe labor market outcomes. We are able to match 424,000 men and 67,000 women born in the US between 1920 and 1929 that appear in the 1950 Census to

[11] The weeks worked variable is only available for sample-line individuals in the 1950 Census, so we only observe this outcome for a 1-in-5 random sample.

the NARA Numident sample (see A.2 for more details). Given known issues with linking women related to name changes, we focus our attention on men in our analysis. The series of linkages required to construct this sample may introduce non-random selection into being successfully linked to the NARA Numident. To address non-representativeness we re-weight our (male) sample with propensity score weights (see Appendix A.2 for more details).

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 (Callaway et al., 2024). 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.

In our primary specifications, we estimate the following differences-in-differences model:

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

Here y_{ct} is an outcome of interest in county c in year t, $f(G_{s(c)})$ is a function of the pre-period goiter rate (cases per 1,000 men) in section (county group) $s(c)$. Our baseline specification uses the continuous measure $G_{s(c)}$, but we also use terciles of $G_{s(c)}$ in robustness checks. α_c are county fixed effects, μ_t are year fixed effects, and $\lambda_{r(c),t}$ 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. We cluster our standard errors at the section level.[12] In our robustness analysis using births or infant deaths in levels as outcomes, we estimate our model using Poisson Pseudo-Likelihood Maximization (PPML).

[12]Section are groups of counties at which pre-1924 goiter rates were measured.

4.1 Estimation Sample

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 during WWI. 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. 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. 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 substantially 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. 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.

5 Effects of Salt Iodization on Infants

We begin by discussing the impact of salt iodization on infant mortality at the county level. Figure 4 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 grows linearly until the end of our sample period in 1929—this trend makes sense as thyroid health of reproductive age women likely improved over time with more iodine exposure. 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).

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 1.6 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 1.6 percent or around 15 percent of the effect size found in Clay et al. (2014).

To assess robustness to alternative specifications, in columns (2) – (4) of Table 2 we show that our estimates are not sensitive to a variety of additional controls.[13] 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 additional 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

[13]We also exclude the year 1919 from our estimation sample in Figure A2 to confirm our results are not influenced by the 1918 influenza pandemic.

results are also robust to this alternate specification.

5.1 Robustness

5.1.1 Placebo Exercises

One 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 other common diseases as opposed to goiter as our "treatment" variable (i.e., we use rickets prevalence per 1,000 men as the treatment). Table 3 displays the estimated effects using these placebo treatments. When using the prevalence of these other diseases 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. Salt iodization should have no direct impact on family formation, but family formation and its correlates can relate to infant health through possibly confounding channels. If changes in unobserved determinants of family formation are driving the impacts on infant health we may expect to see changes in marriage and divorce rates. Figure A5 displays the estimated effects on marriage and divorce. Reassuringly, we see no evidence of pre-trends in these outcomes and no impact of iodization.

5.1.2 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 A1 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. Interestingly, the coefficients for the second and third terciles are similar, suggesting potential non-linear effects.

5.1.3 Changes in Fertility

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. We cannot directly measure county-level fertility rates due to challenges with estimating county-level intercensal adult population. To address these challenges and provide additional robustness for our main infant mortality results, we consider two alternative specifications: one using state-level vitality rates and another using county-level counts.

State-Level Estimates. Reliable county-level birth and infant death statistics allow us to evaluate the effects of salt iodization on infant mortality rates. However, measuring fertility requires reliable intercensal population estimates, which are only available at the

state-level through the Census Bureau's vitality statistics estimates.

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 5 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 4, column 1). However, we do estimate a significant effect by 1927 in our event study specification. Reassuringly, the state-level results are very similar in magnitude to our county-level results. Figure 6 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 4, column 3).

County Count Outcomes. We further address population measurement issues by using population-agnostic specifications with vitality counts. Table A2 displays county-level estimates on infant deaths estimated using Poisson Pseudo-Maximum Likelihood (PPML). Similar to our infant mortality rate analysis, we estimate that salt iodization reduced infant deaths by around 3–8 percent in counties with one standard deviation higher goiter rates relative to the mean county.[14] Table A3 does the same for births. We find no consistent evidence of a significant impact on births.

[14]This also addresses concerns that our results could be driven by changes in the misreporting of infant births that could theoretically be correlated with pre-period goiter rates. See Eriksson et al. (2018) for more details about underreporting of vital statistics in this period.

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

5.1.4 Robustness to Cause-Specific Mortality Definitions

To provide additional evidence that our infant mortality results are driven by salt iodization, we look at the effects of salt iodization on infant mortality separately for specific causes of death. If our main results truly reflect the effects of iodized salt, we would expect to see strong effects for some causes of death and no effects on others. We group the early infancy causes of death into iodine related and not iodine related categories and estimate separate specifications for each category. The causes most likely to be related to iodine deficiency include: "premature birth (not still-born); congenital debility, icterus, and sclerema; other diseases peculiar to early infancy" (Zimmermann, 2011). The non-iodine related causes include: "lack of care [and] injury at birth." We use our state-level data in these analyses given the lack of readily available data at finer levels of geography.

Figure 7A displays the state-level estimates on infant mortality for each category. We find that by 1928 salt iodization reduces infant mortality due to iodine related causes by around 1.2 deaths per 1,000 births in counties with a one standard deviation larger pre-period goiter rate. Reassuringly, the estimated effects are very similar to our coefficients for the overall infant mortality rate. Moreover, we find no effects on the infant mortality rate due to non-iodine related causes. We also examine the "premature birth (not still-born)" category separately in Figure 7B as it is the most common cause of death during

early infancy in our period and most clearly linked to iodine deficiency.[15] Again, we find significant reductions in infant mortality from this cause of around 1 death per 1,000 births. Together, these findings provide additional evidence that the improvements in infant health we estimate are being driven by salt iodization.

6 Short-Term Tradeoffs Between Adult and Infant Health

As early as 1927, doctors in Detroit, Michigan began alerting the medical community about possible negative health effects of salt iodization on adults (McClure, 1927, 1934). A sudden increase in iodine intake among individuals with long standing thyroid issues including those stemming from iodine deficiency can generate iodine-induced hyperthyroidism, otherwise known as the Jod-Basedow effect, which can be fatal. More recent work has documented that the increase in goiter-related deaths was greater in states with more iodine deficiency before salt iodization (Feyrer et al., 2017). In order to better characterize the trade-off between the welfare of children and the welfare of adults, we directly estimate the effects of salt iodization on goiter-related deaths using state-level data.

Figure 8A displays the estimated effect of salt iodization on deaths due to "exophthalmic goiter" for individuals over 30 at the state-level using our PPML specification. We find evidence of a significant increase in goiter deaths for this population peaking in 1925. The number of goiter deaths increases by 12.75% for states with a one standard deviation higher pre-period goiter rate between 1924–1926. We also report the estimated effects on the goiter

[15] We also separately estimated the effects on stillbirths using separate data. We omit these results due to know issues with the registration of stillbirths (United States, 1949, Chapter C). The point estimates suggest a small reduction in stillbirths, but the estimates are noisy preventing us from making definitive conclusions. These results are available upon request.

mortality rate (exophthalmic goiter deaths per 1,000 individuals) in Figure 8B, with a one standard deviation increase in pre-1924 goiter rates corresponding to an increase in 0.9 goiter-related deaths per 100,000 individuals. This estimate is about half of the effect that Feyrer et al. (2017) infer from national trends.

We see evidence of a decline in this effect after 1926, but we use the estimated effect on goiter-related mortality from 1924–1926 as a upper bound on the health costs of salt iodization on adults to compare with the reductions we see in infant mortality. At the mean pre-1924 goiter rate, salt iodization increased goiter-related mortality by 0.69 per 100,000 individuals and decreased infant mortality by 1.08 per 1,000 births. In 1926, the population of the United States was approximately 117.4 million individuals. Among reporting areas, the average birth rate in 1926 was 20.5 per 1,000 individuals, corresponding to a rough estimate of 2.4 million births. Thus, in 1926, salt iodization saved approximately 2,600 infants and killed 810 adults.[16]

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

[16]Even using the much larger estimates of goiter-related mortality from Feyrer et al. (2017) with which they estimate 2,340 excess deaths from goiter-related illness, more infants are saved, resulting in a net positive effect.

or grainier 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 A7. 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 "grainier" 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).

As late as 1955, farm households reported lower usage of iodized salt and were more likely to only use non-iodized salt (USDA, 1955). National representative surveys with questions about iodized salt consumption are not available before 1955, but in 1924 there were likely even greater gaps between urban and rural consumption since the first contemporary self-service grocery store[17] in the United States, Piggly Wiggly, did not open until 1916 (Eschner, 2017). Self-service grocery stores proliferated quickly in the early 1920s, but initially Piggly Wiggly would only establish franchises in towns with more than 5,000 residents (Tolbert, 2023). Methods to improve iodized salt stabilization have continued to improve in the now over 100 years since iodized salt's first introduction in the United States, but even today manufacturers in the United States iodize salt with iodide instead of iodate, which the World Health Organization recommends for better stability in humid conditions (NIH, 2024).

[17]Prior to the introduction of self-service grocery stores, households would purchase dry goods from grocery store clerks, who would retrieve the salt from large containers.

If the iodized salt in small fiber cans never made it to the tables of individuals living in rural areas in 1924, 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.[18]

Figure 9 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.[19] To further explore this relationship, we also plot urban-rural trends in infant mortality separately for counties with high and low prevalence of goiter (defined as being above or below the 25th percentile of the county-level distribution) in Figure 10. In this exercise, we define completely rural counties as those with zero percent of the population living in urban areas to sharpen the distinction. If iodization in urban counties is driving this convergence, we should see less urban convergence in low goiter prevalence counties that would not be as affected by iodization. Indeed, we find that in the high goiter counties there is a complete closing of the urban-rural gap by 1929 which begins to close following iodization. In the low goiter prevalence counties, there is also convergence in infant mortality but the gap only closes by around half. In both sets of counties, we see almost no change in

[18] If we knew when rural access to iodized salt improved, we could also look for a reversal in heterogeneous treatment effects. Unfortunately, it is unclear exactly when rural areas gained access over time, and it was likely much more gradual than the introduction of iodized salt to cities.

[19] Figure A10 shows this relationship at finer levels of disaggregation by urbanicity.

infant mortality rates in the rural counties – consistent with a lack of access to iodized salt.

We investigate the relationship between the urban-rural convergence in infant mortality rates and salt iodization more rigorously in the regressions presented in Table 5. Column 1 displays estimates from our baseline specification but adds additional interactions 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.[20] 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). This is consistent with finding in Love and Davenport (1920) who investigate differences in the prevalence of goiter in urban versus rural areas. They find that "[t]he end result of these complex of causes is, then, the practicable equality that there is between urban and rural districts." We view this as evidence against the explanation that these urban rural differences are driven by differences in baseline goiter rates.

The urban-rural heterogeneity in the effects of salt iodization may also indirectly contribute to other infant health disparities for groups that tend to live in urban areas. We

[20]Since sections include both urban and rural counties, characterizing the relationship between the how rurality of a section and its average goiter rate is unfortunately not meaningful.

run similar regressions to explore the relationship between iodization and other covariates of interest that are known to be correlated with urbanicity (occupation score and share foreign born, see Figure A11 for the raw relationships). In columns (2) and (3) of Table 5 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 and share foreign born, respectively. We find that the infant mortality reductions of salt iodization are also concentrated among counties in sections with above median occupation scores 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 and the native-born population.

Although there are no readily available county-level statistics of thyroid-related deaths in order to disaggregate our findings in Section 6, we can evaluate national patterns in goiter deaths by urbanicity. In Figure 12 we document a relative increase in Graves's disease deaths, which may be caused by iodine-induced hyperthyroidism, among the urban areas of the United States in the years immediately following iodization. This increase in Graves's disease deaths in urban places is particularly interesting given the overall trend of improving health in urban areas over this period. This spike in deaths is consistent with a medical literature that documents iodine-induced hyperthyroidism for those with long-standing iodine deficiencies.

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.[21] These findings indicate

[21]These findings also serve as a robustness check for our main findings on infant mortality rates, as the lack of an effect in rural areas where iodized salt was not available can be considered an additional placebo

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.[22] To contextualize these findings in the larger timeline of the urban mortality transition, for the seven states that consistently reported infant mortality starting in 1900, the ratio of urban to rural infant mortality fell from 1.48 in 1900 to 1.11 in 1920, and then further to 1.01 in 1930 (Haines, 2001). 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.

7.1 Long-Run Effects on Human Capital Revisited

Past work has studied the effects of salt iodization on human capital. Feyrer et al. (2017) use a sample of World War II Army enlistees and find positive effects of salt iodization on cognitive ability, using current county as a proxy for county of birth. Adhvaryu et al. (2020) use Census data and state of birth and document similar positive effects on income, labor force participation, and full-time work—driven by the effects of salt iodization on young women. Adhvaryu et al. (2020) are limited, however, by the lack of finer geography measures by which to define treatment.

test.

[22] 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 5 (column 1) which 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.

We build on these papers by linking the 1950 Census to the National Archive's public version of the Social Security Numident file, which allows us to observe county of birth for a large subsample of individuals born between 1920 and 1929. Since married women usually changed their last names, the quality of linkages for women overall is poor. For this reason, we restrict our analysis to men.[23] We begin by replicating the state-level analysis in (Adhvaryu et al., 2020). For comparability, we define four birth cohort groups. Those born in the years 1923 to 1924 are the "pre-iodization" cohorts, those born between 1925 to 1927 are the "during iodization" cohorts, those born in the 1928 and 1929 cohorts are "after iodization," and those born between 1920 to 1922 serve as pre-period cohorts to evaluate parallel trends. In columns (1)-(3) of Table 6, we replicate the null results for men using state-level variation. In columns (4)-(6), we redefine treatment at the section level. With this greater level of precision, we can detect statistically and economically significant effects for men on labor force participation and employment. The coefficients imply that men born in sections with a one standard deviation higher goiter rate had a 1.3 and 1.0 percentage point higher probability of being in the labor force and being employed, respectively. In Table 7, we fully interact our specification with an indicator for whether the individual was born in an urban county. Consistent with the effects we find on infant mortality, the positive effects on labor market outcomes are driven by men who were born in the urban counties where iodized salt was available.

We repeat the main analysis in Feyrer et al. (2017) using their data on enlistees during WWII. During the war, all Army enlistees were required to take an exam known as the Army

[23] Adhvaryu et al. (2020) also evaluate effects for women since they define treatment by state of birth, which is available in the 1950 Census without further linkages.

General Classification Test (AGCT). Individuals assigned to the Air Force generally had higher test scores on the AGCT because of the higher skill requirements of the Air Force.[24] Unfortunately, the AGCT scores themselves were not reliably recorded in the enlistment data so instead recruitment into the Air Force is used as a proxy for cognitive ability. As in Feyrer et al. (2017) we compare Air Force assignment rates among those that enlisted for individuals from high and low goiter areas before and after the introduction of iodized salt.[25] To examine urban–rural differences in the impacts of iodization, we fully interact their main specification with an indicator for whether or not an individual was born in an urban county.[26] Figure 13 displays the event study coefficients for the urban interaction. The triple difference-in-differences estimates imply that one standard deviation increase in the goiter rate increased the probability of being assigned to the Air Force by 3.81 percentage points (27 percent increase over the pre-period mean) for enlistees born in urban counties. Figure A13 displays the estimated effects for recruits born in rural counties. The estimated effect is also (marginally) positive, but the event study coefficients are noisy, limiting our ability to draw conclusions about the impacts on the rural born population. Consistent with our other findings, the positive effects on human capital are primarily driven by enlistees from urban counties.

[24] See Section 4 in Feyrer et al. (2017) for further discussion.

[25] County of birth is not observed in the enlistment data so we make the same assumption as Feyrer et al. (2017) that county of residence is the same as county of birth which may introduce some measurement error.

[26] Specifically we replicate column 4 in Table 3 of Feyrer et al. (2017). We make two changes to the sample used in Feyrer et al. (2017). First, we redefine the positive selection period for air force recruits to be between January 1940 to August 1945 based on Figure 6 in Feyrer et al. (2017). Second, we drop the 1928 cohort from our analysis because there are so few men from that cohort that enlisted during the positive selection months. This is primarily driven by the fact that the 1928 cohort was 17, the minimum required age for enlistment, at the end of the positive selection period.

At first glance, these effect sizes—and the effect sizes found in the literature as a whole—may seem implausibly large, since sections with one standard deviation higher goiter rates only had 0.65 percentage point higher incidence than the average section. However, goiter rates represent an extreme lower bound for the population affected by iodine deficiency, as many men may have been impacted by iodine deficiency during infancy and childhood without presenting with goiter in adulthood. For instance, the incidence of goiter among examined men in Michigan was only 3% but a local survey of Michigan school children found that 38% of children had goiter in 1924. Further, children exposed to iodine deficiency in utero may not go on to develop goiters, but would still experience potential cognitive deficits. Since there is not a clear mapping between goiter rates measured in adult men and the overall expected population of an area that may be experiencing adverse effects of iodine deficiency, the estimates we present should be interpreted as neither an intent-to-treat or a treatment-on-the-treated. Instead, goiter rates represent a proxy for underlying pathology that is best understood in relative terms (i.e., standard deviations).

Our findings reconcile the inconsistency between the the positive effects found on the cognitive scores of young men in Feyrer et al. (2017) and the null effects on labor market outcomes for these same cohorts of men in Adhvaryu et al. (2020). Using sub-state variation in iodine deficiency we find labor market effects on men that are consistent with the human capital improvements documented by Feyrer et al. (2017). Further disaggregating by men born in urban counties and men born in rural counties, we also see that salt iodization only improved outcomes for urban-born men; this important heterogeneity is missed by both Adhvaryu et al. (2020) and Feyrer et al. (2017).

8 Conclusion

In this paper, we provide the first estimates on the impact of nationwide salt iodization on infant health in the United States. 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 15 percent of the effect size of decreases in lead exposure (Clay et al., 2014). We also find evidence that deaths related to thyroid disease among adults increased the first few years after salt iodization in areas with higher goiter prevalence suggesting a trade-off between long-run reductions in infant mortality and short-term increases in adult mortality. However, our estimates suggest that the reductions in infant deaths due to iodization offset any additional adult deaths attributable to thyroid-related causes. Moreover, we find that these improvements in infant health are concentrated in urban counties where iodized salt was more widely distributed. Finally, we show the long-term effects of salt iodization found by past research are also driven by individuals born in urban areas, where children would have actually been exposed to the newly introduced iodized salt—reconciling prior mixed results. Our findings also contribute to our understanding of the factors behind the urban mortality transition during the 20th century.

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 documenting 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. They also illuminate how limitations in food science technology can limit the effectiveness of public health interventions, with implications for urban-rural health and labor market disparities.

References

Adhvaryu, A., S. Bednar, T. Molina, Q. Nguyen, and A. Nyshadham (2020). When it rains it pours: The long-run economic impacts of salt iodization in the united states. *RESTAT 102*, 395–407.

Anderson, D. M., K. K. Charles, and D. I. Rees (2022). Reexamining the Contribution of Public Health Efforts to the Decline in Urban Mortality. *AEJ: AE 14*(2), 126–157.

Andersson, M., V. Karumbunathan, and M. B. Zimmermann (2012). Global Iodine Status in 2011 and Trends over the Past Decade. *The Journal of Nutrition 142*(4), 744–750.

Bailey, M., K. Clay, P. Fishback, M. R. Haines, S. Kantor, E. Severnini, and A. Wentz (2018). U.s. county-level natality and mortality data, 1915-2007. *ICPSR*.

Barreca, A., K. Clay, and J. Tarr (2014). Coal, Smoke, and Death: Bituminous Coal and American Home Heating.

Callaway, B., A. Goodman-Bacon, and P. H. C. Sant'Anna (2024). Difference-in-Differences with a Continuous Treatment. (arXiv:2107.02637).

Carle, A., A. Krejbjerg, and P. Laurberg (2014). Epidemiology of nodular goiter. influenc of iodine intake. *Best Practice Research Clinical Endocrinology Metabolism 28*(4), 465–479.

Clay, K., W. Troesken, and M. Haines (2014). Lead and Mortality. *RESTAT 96*(3), 458–470.

Eriksson, K., G. T. Niemesh, and M. Thomasson (2018). Revising Infant Mortality Rates for the Early Twentieth Century United States. *Demography 55*(6), 2001–2024.

Eschner, K. (2017). The bizarre story of piggly wiggly, the first self-service grocery store. *Smithsonian Magazine*.

Feyrer, J., D. Politi, and D. N. Weil (2017). The Cognitive Effects of Micronutrient Deficiency: Evidence from Salt Iodization in the United States. *JEEA 15*(2), 355–387.

Goldstein, J. R., M. Alexander, C. Breen, A. Miranda González, F. Menares, M. Osborne, M. Snyder, and U. Yildirim (2023). Censoc mortality file. V 3.0 [dataset], UC Berkeley.

Haines, M. R. (2001). The Urban Mortality Transition in the United States, 1800-1940. *Annales de démographie historique* (1), 33–64.

Helgertz, J., N. Ozder, S. Ruggles, J. R. Warren, C. A. Fitch, J. D. Hacker, M. A. Nelson, J. P. Price, E. Roberts, and M. Sobek (2024). IPUMS Multigenerational Longitudinal Panel. V 1.2 [dataset], IPUMS, Minneapolis, MN.

J.A. Clements (1947). *Iodized salt: Hearing before a subcommittee of the committee on Interstate and Foreign Commerce, House of Representatives, Eightieth Congress, first session, on H. R. 2717.* Number iv, 80 p. Washington, D.C.: GPO.

Kimball, O. P., J. M. Rogoff, and D. Marine (1919). The Prevention of Simple Goiter in Man. *JAMA 73*(25), 1873–1874.

Love, A. G. and C. B. Davenport (1920). *Defects Found in Drafted Men.* G.P.O.

Manson, S., J. Schroeder, D. V. Riper, K. Knowles, T. Kugler, F. Roberts, and S. Ruggles (2024). Ipums nhgis. V 19.0 [dataset], IPUMS, Minneapolis, MN.

Markel, H. (1987, February). "When it rains it pours": endemic goiter, iodized salt, and David Murray Cowie, MD. *American Journal of Public Health 77*(2), 219–229.

McClendon, J. F. (1924). Inverse Relation Between Iodine in Food and Drink and Goiter, Simple and Exophthalmic. *JAMA 82*(21), 1668–1672.

McClure, R. D. (1927). Experiences with the thyroid problem in a detroit clinic. *Annals of surgery 85*(3), 333–338.

McClure, R. D. (1934). Thyroid Surgery as Affected by the Generalized Use of Iodized Salt in an Endemic Goitre Region—Preventive Surgery. *Annals of Surgery 100*(5), 924–932.

Mclendon, J. F. (1939). *Iodine and the Incidence of Goiter*. University of Minnesota Press.

NIH (2024). Iodine fact sheet for health professionals.

Olesen, R. (1926). Distribution of endemic goiter in the United States as shown by thyroid surveys. *Public Health Reports, U.S. Public Health Service 41*(48), 2691–2716.

Olesen, R. (1929). Endemic goiter. *Public Health Bulletin, U.S. Public Health Service* (192).

Politi, D. (2010). The effects of the generalized use of iodized salt on occupational patterns in Switzerland. *Working Paper*.

Remer, T., Y. Hua, J. Esche, and M. Thamm (2022). The DONALD study as a longitudinal sensor of nutritional developments: Iodine and salt intake over more than 30 years in German children. *European Journal of Nutrition 61*(4), 2143–2151.

Ruggles, S., M. A. Nelson, M. Sobek, C. A. Fitch, R. Goeken, J. D. Hacker, E. Roberts, and J. R. Warren (2024). IPUMS Ancestry Full Count Data. V 4.0 [dataset], IPUMS, Minneapolis, MN.

Schiel, J. B. and A. J. Wepfer (1976). Distributional Aspects of Endemic Goiter in the United States. *Economic Geography 52*(2), 116–126.

Tolbert, L. C. (2023). *Beyond Piggly Wiggly : Inventing the American Self-Service Store*. University of Georgia Press.

United States (1949). *Historical Statistics of the United States 1789-1945; a Supplement to the Statistical Abstract of the United States*. Washington, D.C.: U.S. government printing office.

USDA (1955). Household food consumption survey. Technical report.

WHO (1996). Review of findings from 7-country study in africa on levels of salt iodization in relation to iodine deficiency disorders, including iodine-induced hyperthyroidism. *WHO/UNICEF/ICCIDD Consultation Report*.

Zimmermann, M. B. (2011). The role of iodine in human growth and development. *Seminars in Cell Developmental Biology 22*(6), 645–652. Anaphase Promoting Complex, Control of Mitosis, and Beyond Micronutrient and mineral supply in developmental biology.

Zimmermann, M. B. and M. Andersson (2021). Global Endocrinology: Global perspectives in endocrinology: Coverage of iodized salt programs and iodine status in 2020. *European Journal of Endocrinology 185*(1), R13–R21.

9 Tables

Table 1: Summary Statistics

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

	Infant Mortality Rate								
	(1) Simple Goiter	(2) Arthritis	(3) Epilepsy	(4) Scoliosis	(5) Gonococcus infection	(6) Obesity	(7) Syphilis	(8) Tuberculosis, pulmonary	(9) Rickets
Disease $\times \mathbb{1}\{t \in [1919, 1922]\}$	0.14	-1.06**	-0.62	-0.67	0.68	0.25	-0.20	-0.73	0.40
	(0.43)	(0.48)	(0.50)	(0.48)	(0.56)	(0.58)	(0.49)	(0.44)	(0.31)
Disease $\times \mathbb{1}\{t \in [1925, 1927]\}$	-0.62	-0.55	-0.19	0.06	-0.20	-0.47	-0.08	-0.02	0.45
	(0.42)	(0.40)	(0.39)	(0.38)	(0.46)	(0.56)	(0.43)	(0.35)	(0.72)
Disease $\times \mathbb{1}\{t \in [1928, 1929]\}$	-1.59***	-0.55	0.24	-0.30	-0.94	0.62	0.15	0.25	0.20
	(0.41)	(0.57)	(0.52)	(0.46)	(0.74)	(0.60)	(0.70)	(0.50)	(0.65)
Mean Outcome	69.93	69.93	69.93	69.93	69.93	69.93	69.93	69.93	69.93
R-sq	0.72	0.72	0.72	0.72	0.72	0.72	0.72	0.72	0.72
Obs.	13,596	13,596	13,596	13,596	13,596	13,596	13,596	13,596	13,596

Notes: The outcome variable is the infant mortality rate measured as the number of infants deaths per 1,000 population. The disease rates are all standardized so that the coefficients represent the estimated effect of a 1 standard deviation increase. All columns include year, county, and region-by-year fixed effects as well as 1920 county-level covariates interacted with a linear time trend. 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 4: State Level Estimates

	Infant Mortality Rate	Birth Rate
	(1)	(2)
Goiter $\times \mathbb{1}\{t > 1924\}$	-1.14	0.13
	(0.67)	(0.12)
Mean Outcome	72.80	21.84
R-sq	0.95	0.98
Obs.	239	239

Notes: The outcome variables are infant deaths per 1,000 births and births 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, 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 population. Standard errors are clustered at the state level.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$

Table 5: County Infant Mortality Rate – Heterogeneity

	Pct. Urban	Occupation Score	Pct. Foreign Born
	(1)	(2)	(3)
Goiter $\times \mathbb{1}\{t > 1924\}$	-0.26	-0.05	0.26
	(0.45)	(0.40)	(0.50)
Goiter $\times \mathbb{1}\{t > 1924\} \times$ Above Med.	-1.68**	-1.86***	-1.90***
	(0.69)	(0.60)	(0.71)
Mean Outcome	69.93	69.93	69.93
R-sq	0.72	0.72	0.72
Obs.	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.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$

Table 6: Long Run Impacts – State versus Section Variation

	State Variation			Section Variation		
	(1) In Labor Force	(2) Employed	(3) Worked 40 Weeks	(4) In Labor Force	(5) Employed	(6) Worked 40 Weeks
Goiter $\times \mathbb{1}\{t \in [1920, 1922]\}$	-0.002	-0.001	-0.004	-0.004	-0.003	-0.001
	(0.004)	(0.004)	(0.007)	(0.003)	(0.003)	(0.009)
Goiter $\times \mathbb{1}\{t \in [1925, 1927]\}$	0.003	0.004	-0.007	0.003	0.004	0.005
	(0.003)	(0.003)	(0.009)	(0.004)	(0.003)	(0.010)
Goiter $\times \mathbb{1}\{t \in [1928, 1929]\}$	0.005	0.006	0.004	0.013**	0.010*	0.011
	(0.005)	(0.005)	(0.008)	(0.005)	(0.005)	(0.009)
Mean Outcome	0.92	0.89	0.82	0.92	0.89	0.82
R-sq	0.03	0.04	0.04	0.03	0.04	0.05
Obs.	423,255	419,200	74,927	423,255	419,200	74,927

Notes: This regression displays the relationship between long-run labor market outcomes and salt iodization. *In Labor Force* is an indicator for whether or not the individual was in the labor force, *Employed* is defined similarly but is set to zero for individuals that are unemployed, and *Worked 40 Weeks* is an indicator for whether the individual worked at least 40 weeks in the prior year conditional on working at least 1 week (this variable is only available for sample line individuals i.e., a 1-in-5 random sample). In columns 1–3 the continuous goiter measure varies at the state of birth level. In columns 4–6 the continuous goiter measure varies at the section of birth level. Columns 1–3 include an indicator for whether the individual is recorded as Black as well as state of birth level controls for the percent of the population that is Black in 1920, the percent of the population that is female in 1920, and the state's latitude, each of which is interacted with the period of birth (1920–1922, 1923–1924, 1925–1927, 1928–1929). They also include state of birth and division-by-cohort fixed effects. Columns 4–6 include a similar set of controls except variables are measured at the section of birth level and they include section of birth instead of state of birth fixed effects. The sample is men in our linked 1950 Census sample. Observations are re-weighted by their propensity score (see Table A4). Standard errors are clustered at the state of birth level in columns 1–3 and the section of birth level in columns 4–6.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$

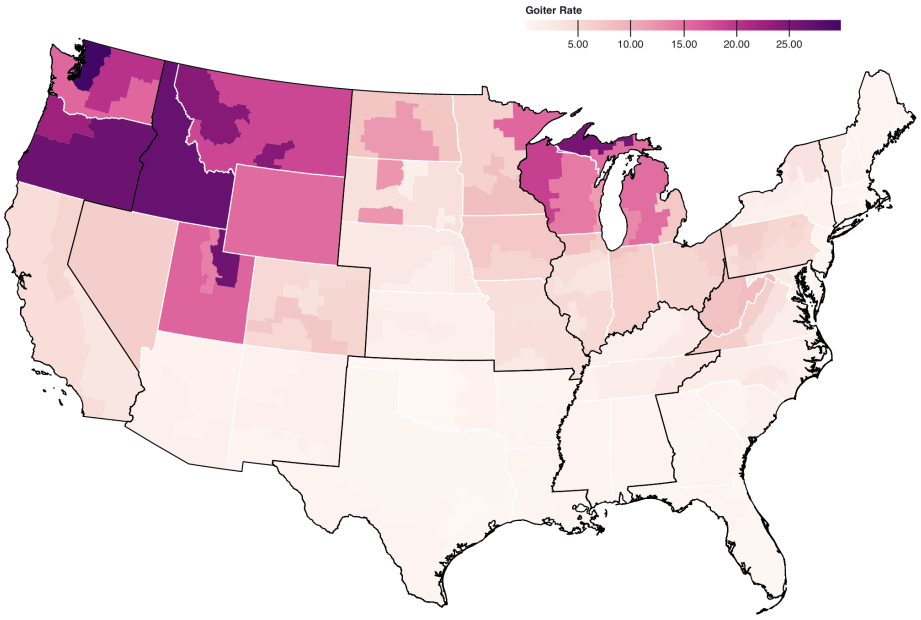
Table 7: Long Run Impacts — Urban–Rural Heterogeneity

	Pooled			Urban vs. Rural		
	In Labor Force	Employed	Worked 40 Weeks	In Labor Force	Employed	Worked 40 Weeks
Goiter $\times \mathbb{1}\{t \in [1920, 1922]\}$	-0.004	-0.003	-0.001	-0.004	-0.003	-0.012
	(0.003)	(0.003)	(0.009)	(0.006)	(0.006)	(0.016)
Goiter $\times \mathbb{1}\{t \in [1925, 1927]\}$	0.003	0.004	0.005	-0.003	-0.003	0.020
	(0.004)	(0.003)	(0.010)	(0.005)	(0.007)	(0.022)
Goiter $\times \mathbb{1}\{t \in [1928, 1929]\}$	0.013**	0.010*	0.011	-0.006	-0.014	0.014
	(0.005)	(0.005)	(0.009)	(0.009)	(0.009)	(0.024)
Goiter $\times$ Urban $\times \mathbb{1}\{t \in [1920, 1922]\}$				0.002	0.001	0.015
				(0.007)	(0.007)	(0.020)
Goiter $\times$ Urban $\times \mathbb{1}\{t \in [1925, 1927]\}$				0.010	0.010	-0.014
				(0.007)	(0.008)	(0.023)
Goiter $\times$ Urban $\times \mathbb{1}\{t \in [1928, 1929]\}$				0.025**	0.030***	0.000
				(0.012)	(0.011)	(0.027)
Mean Outcome	0.92	0.89	0.82	0.92	0.89	0.82
R-sq	0.03	0.04	0.05	0.03	0.04	0.05
Obs.	423,255	419,200	74,927	423,255	419,200	74,924

Notes: This regression displays the relationship between long-run labor market outcomes and salt iodization. See Table 6 for a description of the outcomes and controls used. Columns 1–3 are the same as columns 4–6 in Table 6. Columns 4–6 interact each covariate with an urban indicator which is set to one if the percentage of people living in an urban area in the individual's county of birth is above the median of the national county-level distribution in 1920. Observations are re-weighted by their propensity score (see Table A4). Standard errors are clustered at the section of birth level.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$

10 Figures

(A) Entire U.S.



(B) Estimation Sample

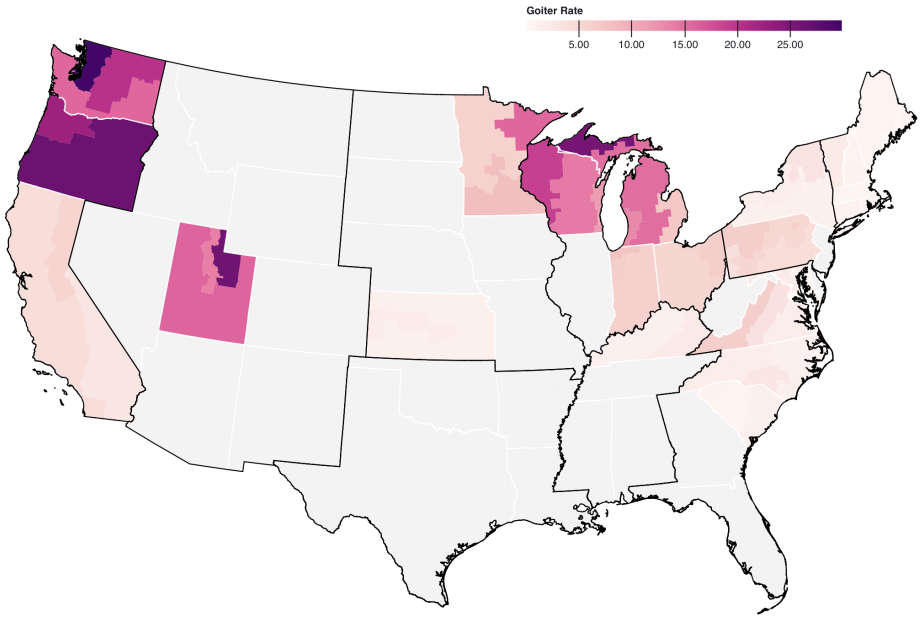
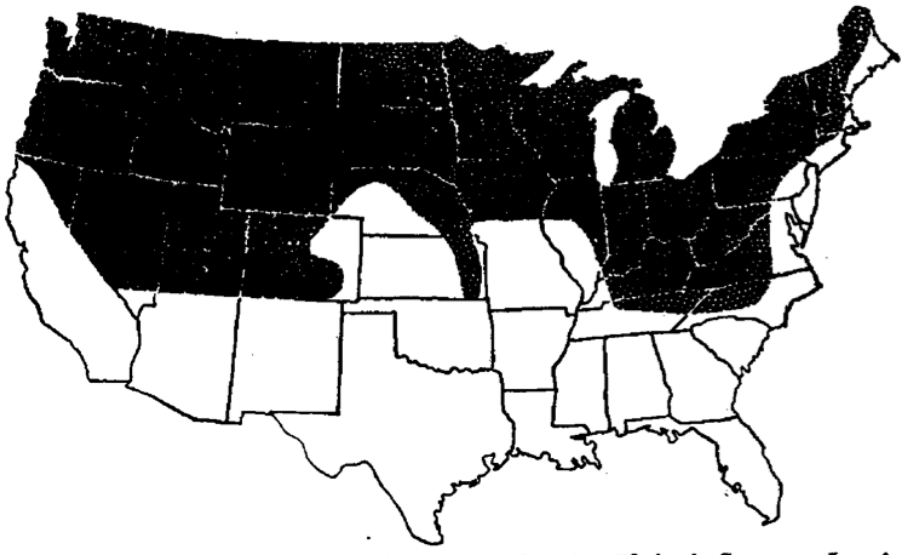


Figure 1: Goiter Rates. This figure displays rates of simple goiter per 1,000 inspected soldiers at the section level from Love and Davenport (1920). State boundaries are denoted by the white lines and Census divisions are denoted by the black lines. Panel A displays goiter rates for all sections in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

(A) Iodine Content of Drinking Water



(B) Endemic Goiter

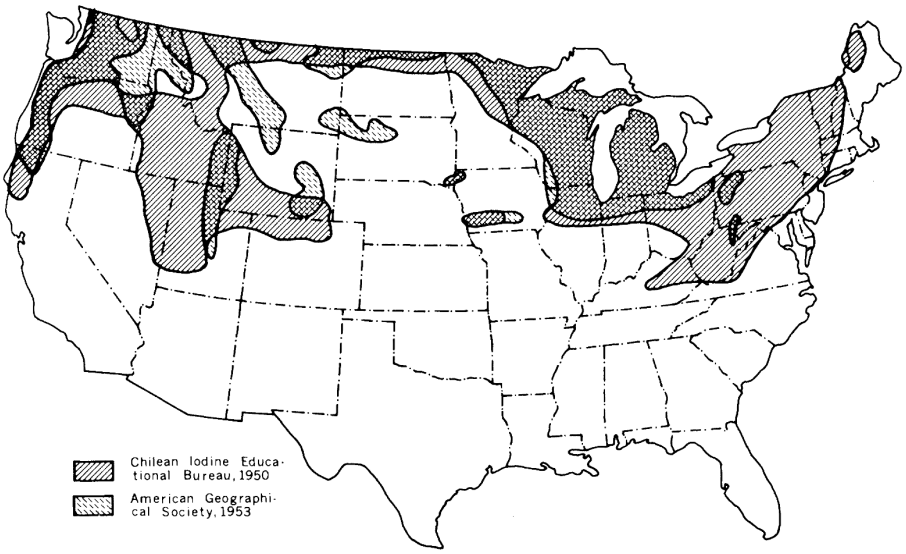


Figure 2: Iodine and Goiter. Panel (A) of this graph displays areas of the United States with high and low levels of iodine in drinking water. Areas marked in black have low levels of iodine (1 to 22 parts iodine per hundred billion parts water). Source: McClendon (1924). Panel (B) displays areas of endemic goiter from two independent surveys during the 1950s. Source: Schiel and Wepfer (1976).

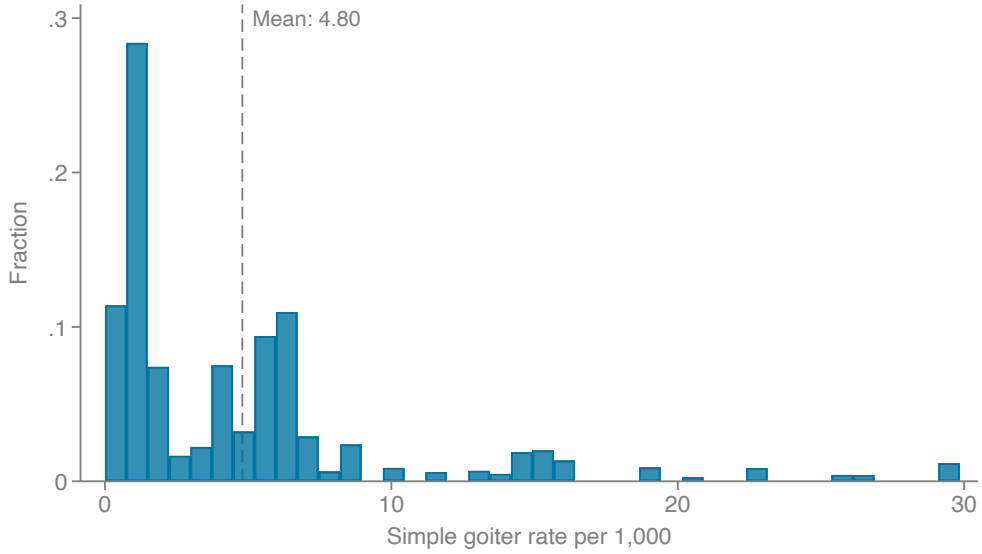


Figure 3: Distribution of Goiter Rates. This graph displays the distribution of simple goiter rates across sections in our estimation sample. Each section is weighted by its 1920 population.

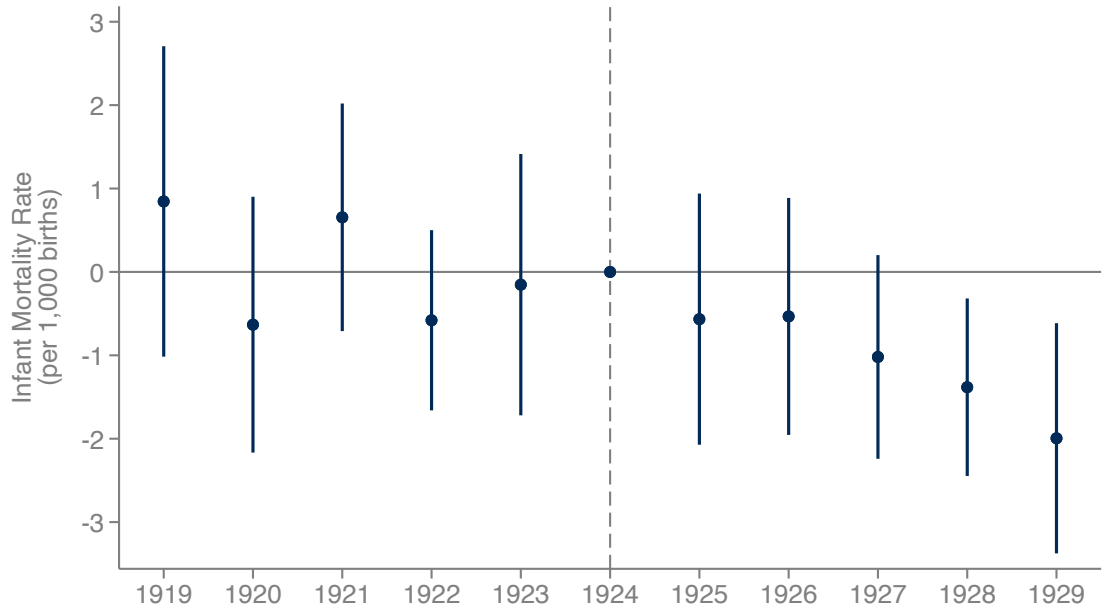


Figure 4: Infant Mortality Rate. This figure displays coefficients from an event study of infant mortality rates on goiter rates. The event study uses the same specification as Column (1) in Table 2, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend.

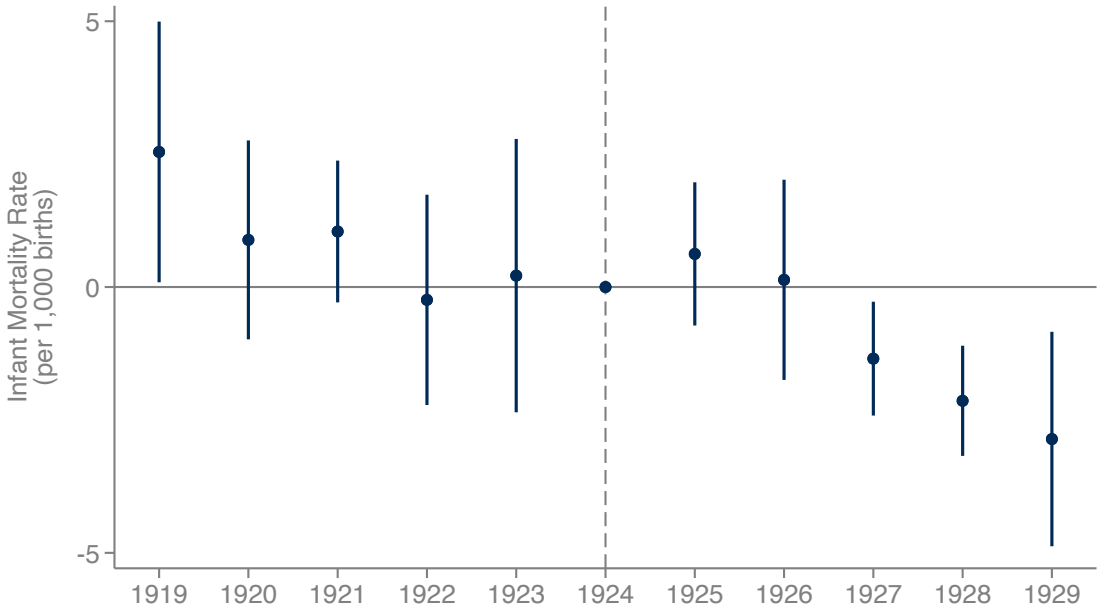


Figure 5: State Level Infant Mortality. This figure displays coefficients from an event study of infant mortality rates on goiter rates. The event study uses the same specification as Column (1) in Table 4, which includes state fixed effects, year fixed effects, region-by-year fixed effects, and 1920 state covariates interacted with a linear time trend.

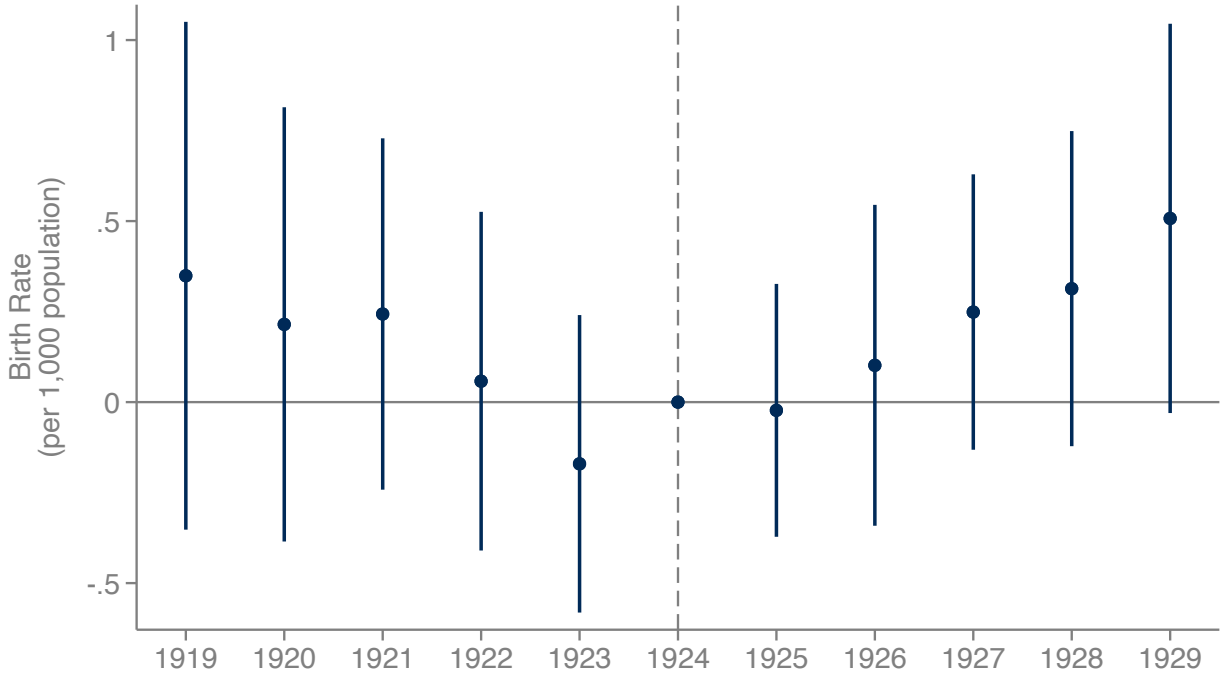
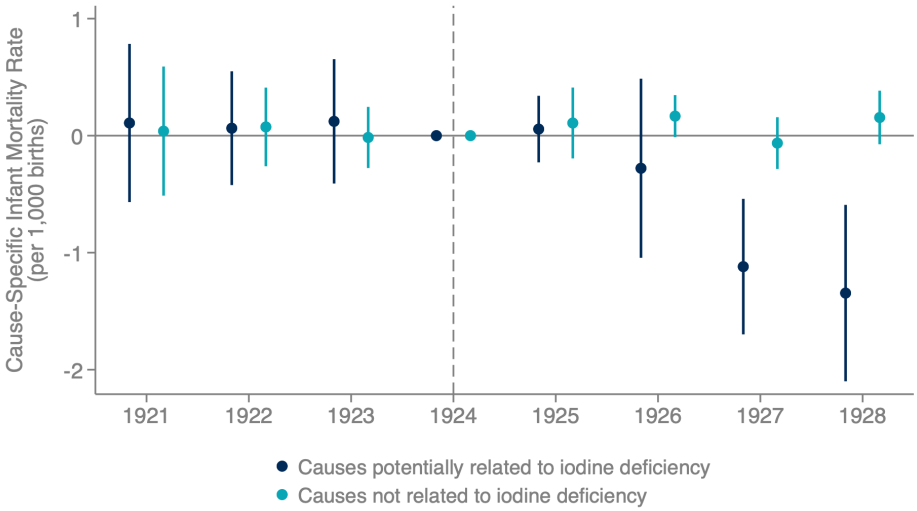


Figure 6: State Level Births. This figure displays coefficients from an event study of birth rates on goiter rates. The event study uses the same specification as Column (3) in Table 4, which includes state fixed effects, year fixed effects, region-by-year fixed effects, and 1920 state covariates interacted with a linear time trend.

(A) Iodine vs. Non-iodine Related Causes



(B) Premature Birth

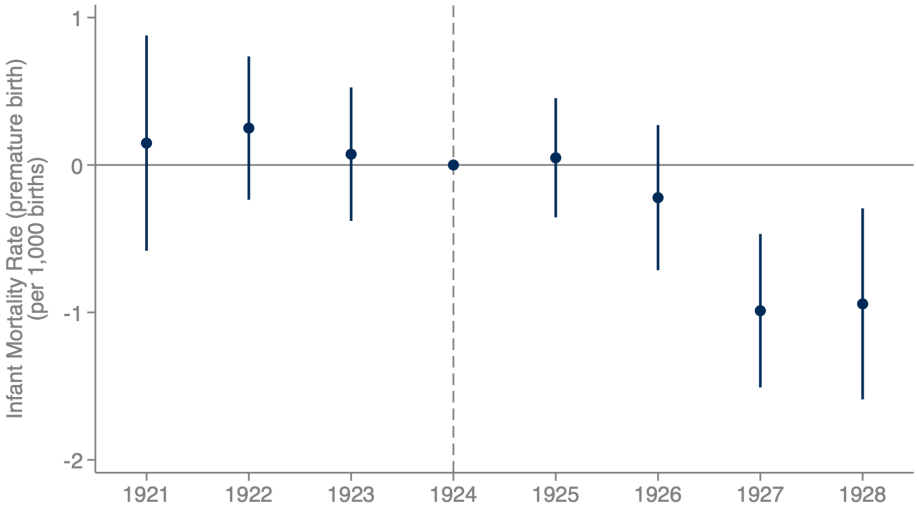
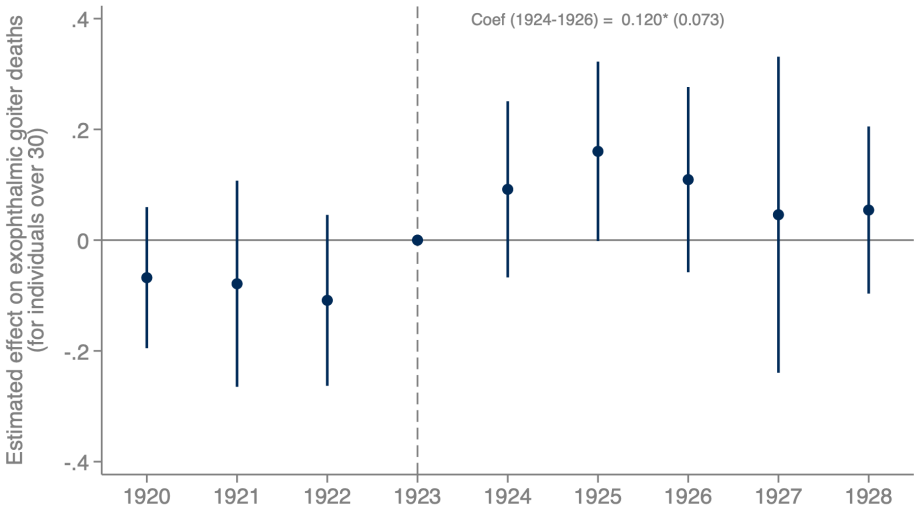


Figure 7: State Level Cause-Specific Infant Mortality. Panel (A) of this figure displays coefficients from an event study of the infant mortality rate on goiter rates separately for causes of death that are potentially related to iodine deficiency. The iodine deficiency related categories include: "premature birth (not still-born); congenital debility, icterus, and sclerema; other diseases peculiar to early infancy." The non-iodine related categories include: "lack of care; injury at birth." The event study uses the same specification as Column (3) in Table 4, which includes state fixed effects, year fixed effects, region-by-year fixed effects, and 1920 state covariates interacted with a linear time trend. Panel (B) of this figure displays coefficients from an event study of the infant mortality rate due to "premature birth (not still-born)" causes on goiter rates. The event study uses the same specification as Column (4) in Table 4, which includes state fixed effects, year fixed effects, region-by-year fixed effects, and 1920 state covariates interacted with a linear time trend.

(A) Goiter Deaths over 30 – PPML



(B) Goiter Mortality Rate

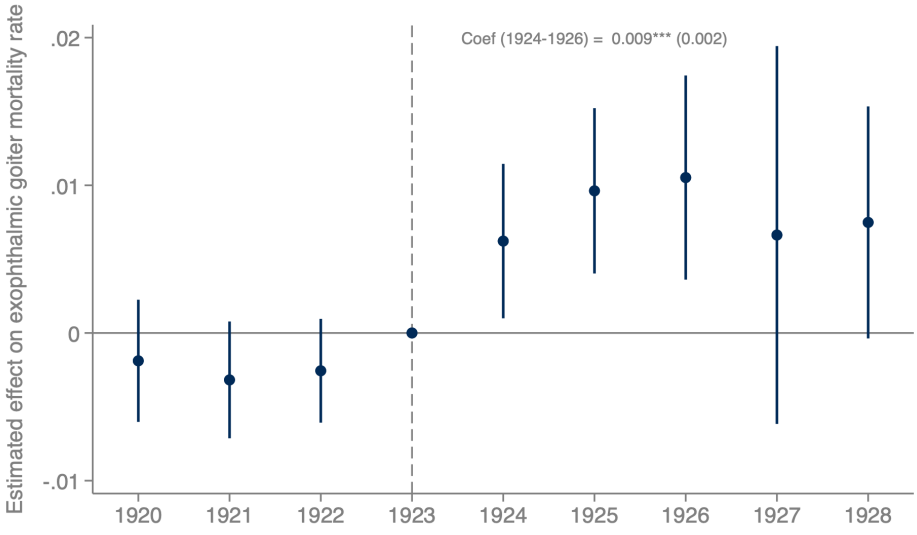


Figure 8: State Level Goiter Related Deaths. Panel (A) of this figure displays coefficients from an event study of state-level goiter deaths for individuals over 30 on goiter rates estimated using Poisson Pseudo-Maximum Likelihood. Panel (B) of this figure displays coefficients from an event study of goiter mortality rates (goiter deaths per 1,000 individuals) on goiter rates.

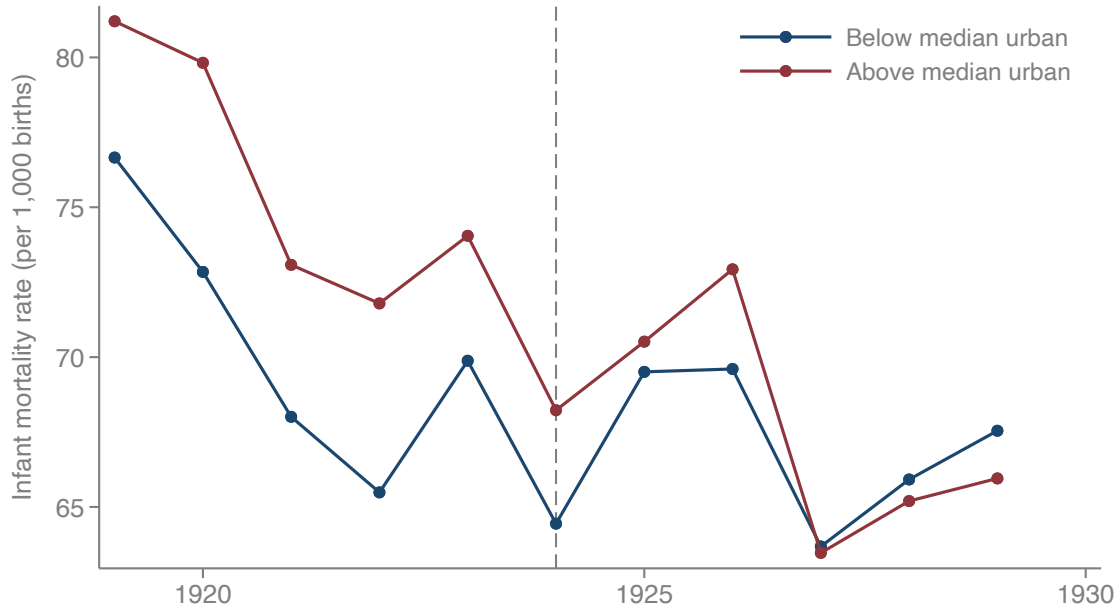
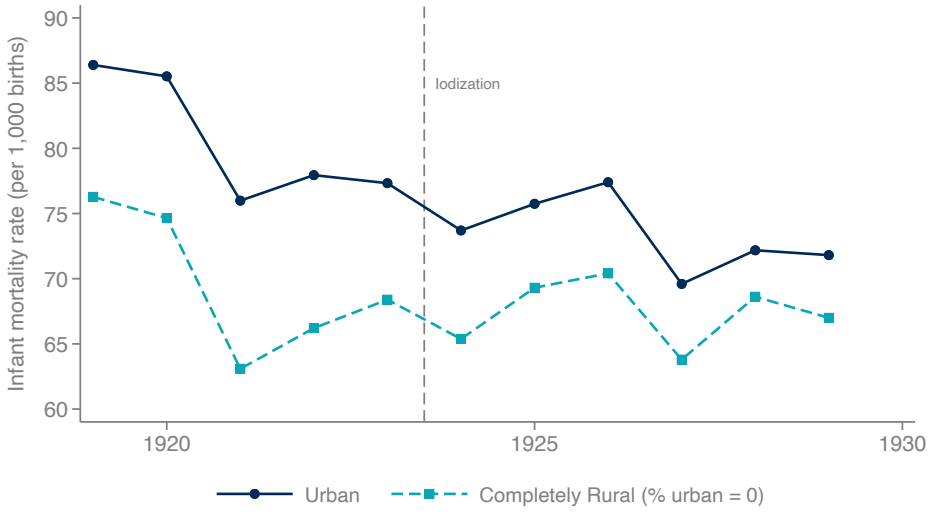


Figure 9: Trends in Urban-Rural Infant Mortality. This figure displays trends in infant mortality between urban and rural counties in our estimation sample. Urban and rural status are based on whether or not the percent of the population living in an urban area is above or below the median level of the national county distribution in our sample.

(A) Low Goiter Prevalence



(B) High Goiter Prevalence

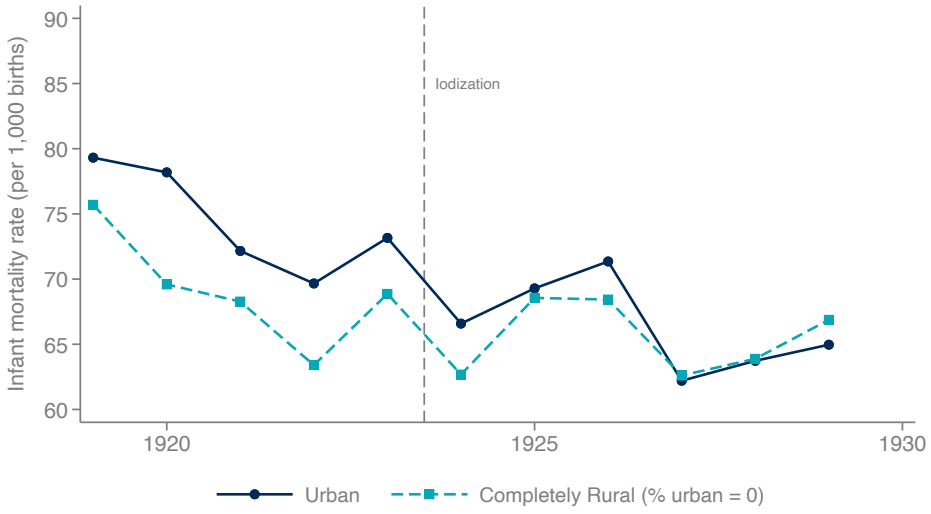


Figure 10: Trends in Urban-Rural Infant Mortality by Goiter Prevalence. Panel (A) of this figure displays the infant mortality rate in counties with high goiter prevalence (defined as greater than the 25th percentile i.e. 1.22 per 1,000 men) separately for urban and rural counties (rural here is defined as counties with zero percent of the population living in urban areas). Panel (B) displays the same but for low goiter prevalence counties.

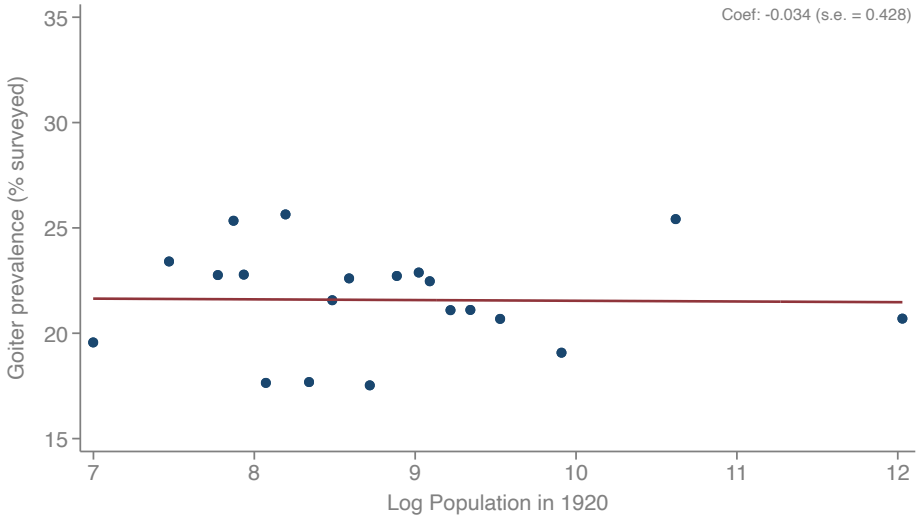
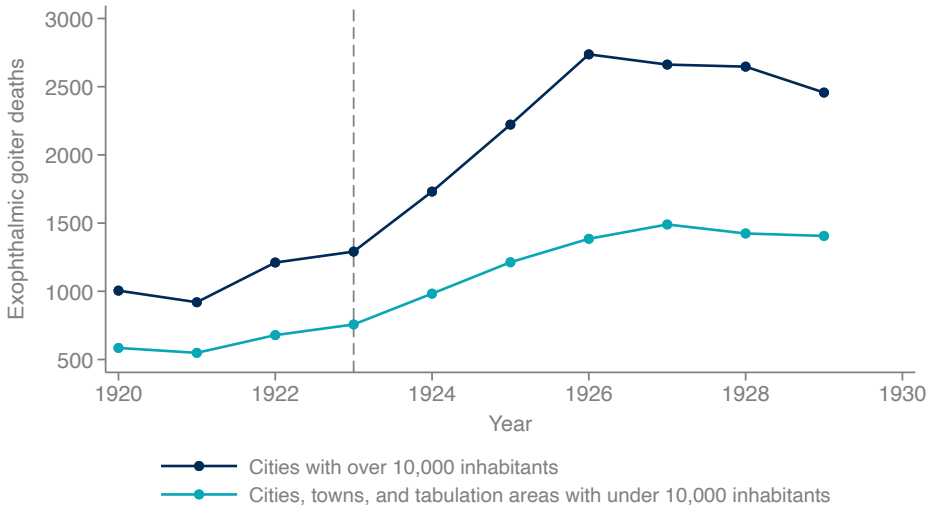


Figure 11: Relationship between Goiter Prevalence and Population. This figure displays a binscatter of the prevalence of goiter on the log population controlling for state fixed effects. The data are based on surveys of goiter prevalence across 130 localities in four states (Massachusetts, Connecticut, 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.

(A) Entire U.S.



(B) Estimation Sample

Figure 12: Urban vs. Rural Goiter Deaths. Panel (A) of this figure displays the number of deaths in the U.S. caused by exophthalmic goiter separately by cities with over 10,000 inhabitants and other tabulation areas with less than 10,000 inhabitants. Panel (B) displays the ratio between the deaths in the two types of areas.

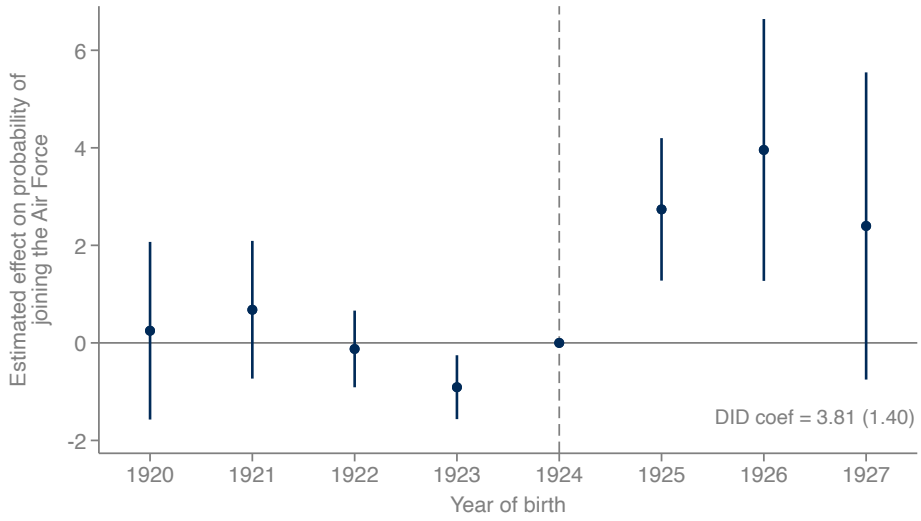


Figure 13: Long Run Impacts on Cognitive Ability – Urban Areas. This figure displays coefficients on the urban interaction from an event study of the probability of joining the Air Force on goiter rates fully interacted with an indicator for being born in an urban county. The event study uses the same specification as Column (4) in Table 3 of Feyrer et al. (2017). See Section 7.1 for more details.

A Data

A.1 NARA Numident

The NARA Numident records contain nearly complete death coverage for Social Security Number (SSN) holders that died between 1988 and 2005. The NARA Numident has been harmonized and linked to the 1940 Census by the CenSoc Project (Goldstein et al., 2023). We use their CenSoc–Numident file and link it forward to the 1950 Census using the IPUMS Multigenerational Longitudinal Panel (MLP) project's linkages (Helgertz et al., 2024).

Since the NARA Numident is double truncated, we restrict to individuals that died between the ages of 68 and 76, which are the ages of death we can observe for all of our cohorts. This accounts for 42% of deaths observed in the NARA Numident for the 1920 cohort and 65% for the 1929 cohort. The earliest age of death we observe reliably for the 1920 cohort is 68 in 1988 and the latest age of death we observe reliably for the 1929 cohort is 76 in 2005. To assuage concerns regarding selection from this restriction, we estimate a Gompertz model, which accounts for the double-truncation in the Numident data, to look at the effects of salt iodization on longevity. We find no evidence of differences in longevity conditional on survival to age 66 by iodized salt exposure. Full results of this analysis are available upon request.

A.2 Numident–Census Linkage

To construct our long-run analysis sample we start with all individuals in the 1950 Census that are born in the U.S. between 1920 and 1929 which leaves us with 23 million individuals. We exclude people born in Alaska and Hawaii. We match 6.4 million (27%) of these indi-

viduals to the 1940 Census using the IPUMS MLP crosswalk. We are able to match 44% of men and 12% of women. Lastly, we match 424,000 men (3.8% of original sample) and 67,000 (0.6% of original sample) women to the NARA Numident sample using the CenSoc linkages. This means we are able to match around 8.5% of the men that we observe in both the 1950 and 1940 Census to the NARA Numident. This match rate is low because of the constraint in the NARA Numident where we only have good coverage of individuals that died between 1988 and 2005. Adjusting the match rates to account for the truncated mortality coverage gives around a 20-30% match rate which is consistent with the typical rates for historical record linkages (see CenSoc record linkage documentation for further discussion of this issue).

Table A4 displays balance regressions of an indicator for being successfully linked to the NARA Numident on a host of Census covariates for our 1950 Census sample. We find that the probability of being successfully linked is not balanced across these covariates and this is particularly pronounced for women in our sample. Given known issues with linking women related to name changes, we focus our attention on men in our analysis. To address non-representativeness we re-weight our (male) sample with propensity score weights from a probit regression with the same set of covariates. Columns 4–6 display the same balance regressions re-weighting our linked sample. We still reject that our sample is representative of the overall 1950 sample. However, the re-weighting significantly improves the representativeness of our sample. In the sample of men, the F-statistic goes from 334 to 6 when re-weighting.

B Figures and Tables

B.1 Appendix Tables

Table A1: Impact on Infant Mortality Rate — Discrete Treatment

	Infant Mortality Rate			
	(1)	(2)	(3)	(4)
Goiter Tercile (=2)				
$\times \mathbb{1}\{t > 1924\}$	-2.39***	-2.32***	-2.82***	-2.34***
	(0.78)	(0.83)	(0.68)	(0.77)
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 A2: Impact on Infant Deaths – PPML

	Infant Deaths			
	(1)	(2)	(3)	(4)
Goiter $\times \mathbb{1}\{t > 1924\}$	-0.03**	-0.04***	-0.08***	-0.04**
	(0.02)	(0.01)	(0.02)	(0.02)
Mean Outcome	70.93	70.93	70.41	70.93
Division-Year FE		✓		
State-Year FE			✓	
Control Quintile × Year FE				✓
R-sq				
Obs.	13,596	13,596	13,585	13,596

Notes: The outcome variable in columns (1) – (4) is the 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, county fixed effects, 1920 county level covariates interacted with a linear time trend, and includes region-by-year fixed effects. The estimation period is 1919-1929. Standard errors are clustered at the section level.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$

Table A3: Impact on Births – PPML

	Births			
	(1)	(2)	(3)	(4)
Goiter $\times \mathbb{1}\{t > 1924\}$	-0.01	-0.02*	-0.04*	-0.01
	(0.01)	(0.01)	(0.02)	(0.01)
Mean Outcome	954.93	954.93	948.47	954.93
Division-Year FE		✓		
State-Year FE			✓	
Control Quintile × Year FE				✓
R-sq				
Obs.	13,596	13,596	13,585	13,596

Notes: The outcome variable in columns (1) – (4) is 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, county fixed effects, 1920 county level covariates interacted with a linear time trend, and includes region-by-year fixed effects. The estimation period is 1919-1929. Standard errors are clustered at the section level.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$

Table A4: Long Run Impacts – Sample Balance

	No weights			Re-weighted		
	(1) Pooled	(2) Male	(3) Female	(4) Pooled	(5) Male	(6) Female
Black	-0.010***	-0.015***	-0.002***	0.093***	-0.017*	0.058***
	(0.001)	(0.001)	(0.000)	(0.023)	(0.009)	(0.012)
1923-1924	0.005***	0.011***	-0.002***	-0.047***	-0.000	-0.070***
	(0.000)	(0.001)	(0.000)	(0.013)	(0.002)	(0.017)
1925-1926	0.005***	0.011***	-0.002***	-0.117***	-0.000	-0.189***
	(0.000)	(0.001)	(0.000)	(0.020)	(0.002)	(0.026)
1927-1929	0.005***	0.011***	-0.001**	-0.193***	0.003	-0.323***
	(0.000)	(0.000)	(0.000)	(0.013)	(0.002)	(0.020)
Urban status	-0.001***	-0.001	-0.001***	0.002	0.001	-0.022**
	(0.000)	(0.001)	(0.000)	(0.009)	(0.004)	(0.008)
Farm status	0.000	0.001	0.001***	0.027***	0.001	0.023**
	(0.000)	(0.001)	(0.000)	(0.008)	(0.005)	(0.009)
Born: East south central	-0.005***	-0.010***	-0.001**	0.090***	-0.005	0.106***
	(0.001)	(0.002)	(0.000)	(0.033)	(0.023)	(0.026)
Born: Middle atlantic	-0.006***	-0.012***	-0.001***	-0.040	-0.007	-0.004
	(0.001)	(0.002)	(0.000)	(0.034)	(0.028)	(0.029)
Born: Mountain	0.005**	0.010***	-0.000	0.037	0.015	0.057**
	(0.002)	(0.004)	(0.000)	(0.031)	(0.028)	(0.027)
Born: New england	-0.001	-0.002	0.001	-0.045	-0.004	-0.021
	(0.003)	(0.005)	(0.000)	(0.039)	(0.036)	(0.034)
Born: Pacific	0.001	0.002	-0.001***	0.027	0.010	0.020
	(0.002)	(0.005)	(0.000)	(0.037)	(0.036)	(0.026)
Born: South atlantic	-0.005***	-0.010***	-0.001**	0.094***	-0.018	0.086***
	(0.002)	(0.003)	(0.000)	(0.029)	(0.031)	(0.020)
Born: West north central	0.003**	0.006**	-0.000	0.005	0.008	0.006
	(0.001)	(0.003)	(0.000)	(0.022)	(0.015)	(0.021)
Born: West south central	-0.003**	-0.007**	-0.000	0.076**	-0.005	0.100***
	(0.002)	(0.003)	(0.000)	(0.032)	(0.026)	(0.027)
Relat. to head: Spouse	-0.026***	-0.009***	0.014***	-0.145***	0.011	-0.395***
	(0.002)	(0.002)	(0.001)	(0.021)	(0.032)	(0.032)
Relat. to head: Child	-0.005***	0.002***	0.016***	-0.012	0.001	-0.201***
	(0.001)	(0.000)	(0.001)	(0.027)	(0.003)	(0.028)
Relat. to head: Parent	-0.012***	-0.001*	0.008***	0.064***	0.006	-0.121***
	(0.001)	(0.001)	(0.001)	(0.024)	(0.005)	(0.028)
Relat. to head: Parent-in-law	-0.017***	-0.010***	0.003***	0.172***	-0.002	-0.002
	(0.001)	(0.001)	(0.000)	(0.030)	(0.004)	(0.032)
Occ: Farm	0.006***	0.000	-0.001***	-0.023	-0.006	0.045
	(0.001)	(0.001)	(0.000)	(0.047)	(0.007)	(0.066)
Occ: Semi-skilled	0.002***	0.002***	0.002***	-0.009	-0.003	0.003
	(0.000)	(0.000)	(0.000)	(0.045)	(0.004)	(0.059)
Occ: Unskilled	0.001	-0.000	-0.000**	0.041	-0.006	0.036
	(0.001)	(0.001)	(0.000)	(0.048)	(0.006)	(0.063)
Occ: Other/NA	-0.005***	-0.005***	-0.001***	0.022	-0.002	-0.013
	(0.000)	(0.001)	(0.000)	(0.043)	(0.005)	(0.057)
Both parents in hhld.	0.009***	0.012***	0.005***	0.100***	0.013**	0.085***
	(0.000)	(0.001)	(0.000)	(0.023)	(0.006)	(0.011)
Lives in Midwest	0.002**	0.003*	0.000**	-0.008	-0.002	0.036
	(0.001)	(0.002)	(0.000)	(0.034)	(0.023)	(0.031)
Lives in South	-0.003***	-0.006***	-0.001***	-0.054**	0.012	0.002
	(0.001)	(0.001)	(0.000)	(0.025)	(0.027)	(0.023)
Lives in West	-0.004***	-0.007***	-0.000*	-0.060	-0.015	-0.016
	(0.001)	(0.002)	(0.000)	(0.037)	(0.029)	(0.035)
Married	-0.003***	0.001***	-0.016***	0.123***	0.003*	0.167***
	(0.001)	(0.000)	(0.001)	(0.027)	(0.002)	(0.037)
Num. children	0.001***	0.000	0.002***	0.000	0.002*	0.002
	(0.000)	(0.000)	(0.000)	(0.004)	(0.001)	(0.005)
Lives in birth state	0.007***	0.014***	0.001***	0.012	0.009*	0.024*
	(0.000)	(0.001)	(0.000)	(0.009)	(0.005)	(0.013)
R-sq	0.01	0.01	0.01	0.11	0.00	0.26
Obs.	23,260,331	11,263,389	11,996,942	23,260,331	11,263,389	11,996,942
F-statistic	381.22	333.84	475.38	922.11	6.07	3703.85

Notes: This table displays regressions of an indicator for whether an individual in our 1950 Census sample is successfully linked to a county of birth in the Numident against a set of covariates separately by gender. Columns 1–3 show the raw relationship and columns 4–6 show the relationship when individuals are re-weighted. For each individual we obtain a propensity score $\Pr_i(L_i = 1|\mathbf{X}_i)$ from a probit regression with the same covariates in the table (we run separate probit regressions for men and women). Each individual then receives a weight equal to $(1 - \Pr_i(L_i = 1|\mathbf{X}_i))/\Pr_i(L_i = 1|\mathbf{X}_i)$. Standard errors are clustered at the state of birth level.*** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$

B.2 Appendix Figures

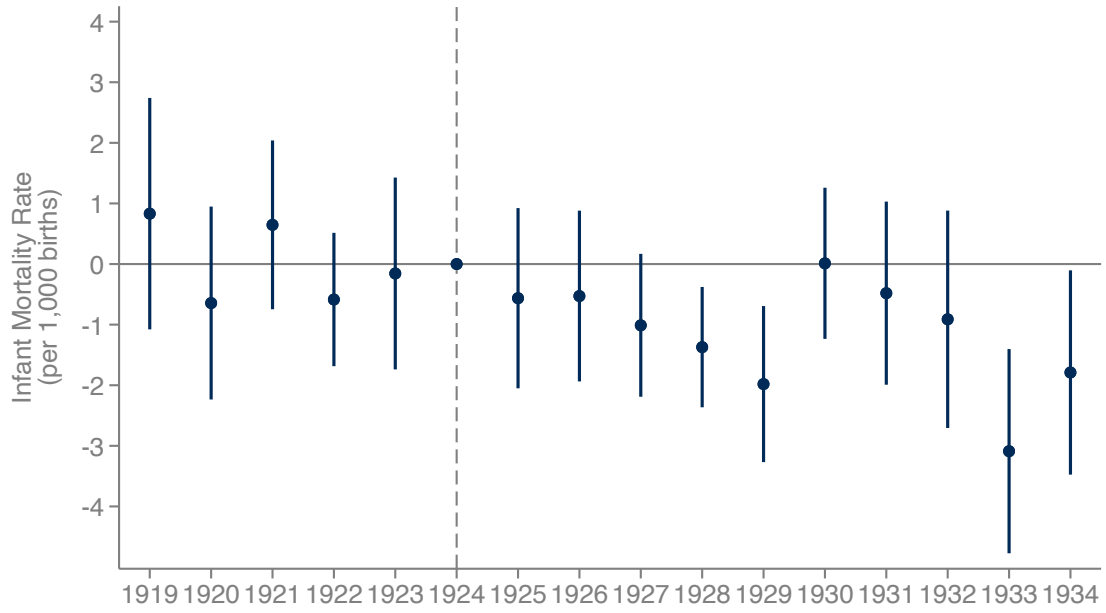


Figure A1: Infant Mortality Rate All Years. This figure displays coefficients from an event study of infant mortality rates on goiter rates. The event study uses the same specification as Column (1) in Table 2, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend. The estimation period is 1919 – 1934.

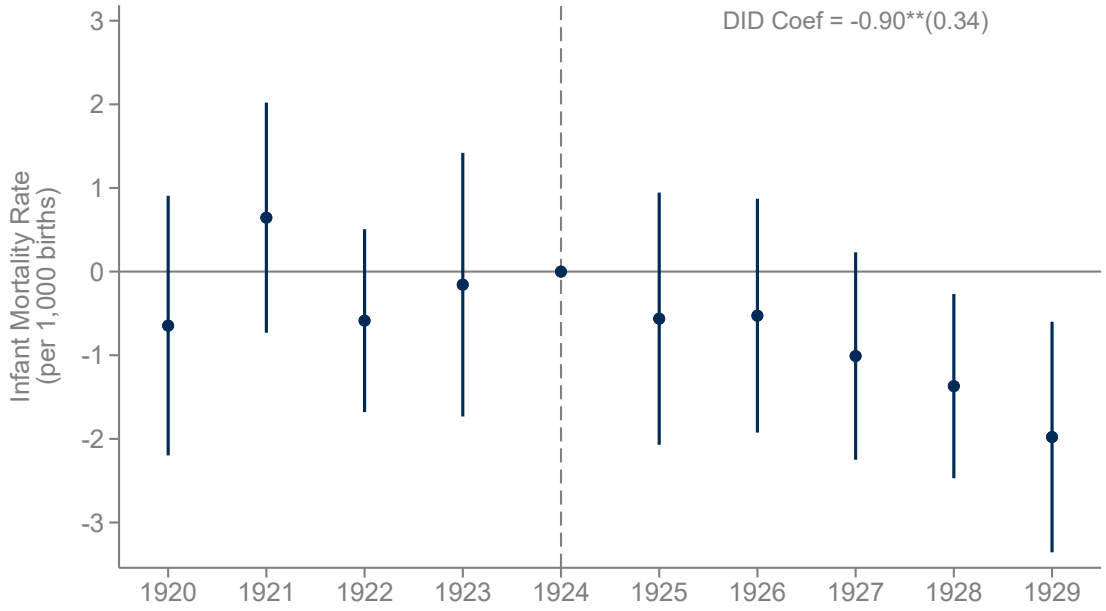


Figure A2: Infant Mortality Rate – Excluding Influenza Pandemic. This figure displays coefficients from an event study of infant mortality rates on goiter rates. The event study uses the same specification as Column (1) in Table 2, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend. The estimation period is 1919 – 1934. The specification excludes 1919 to avoid interactions with the influenza pandemic.

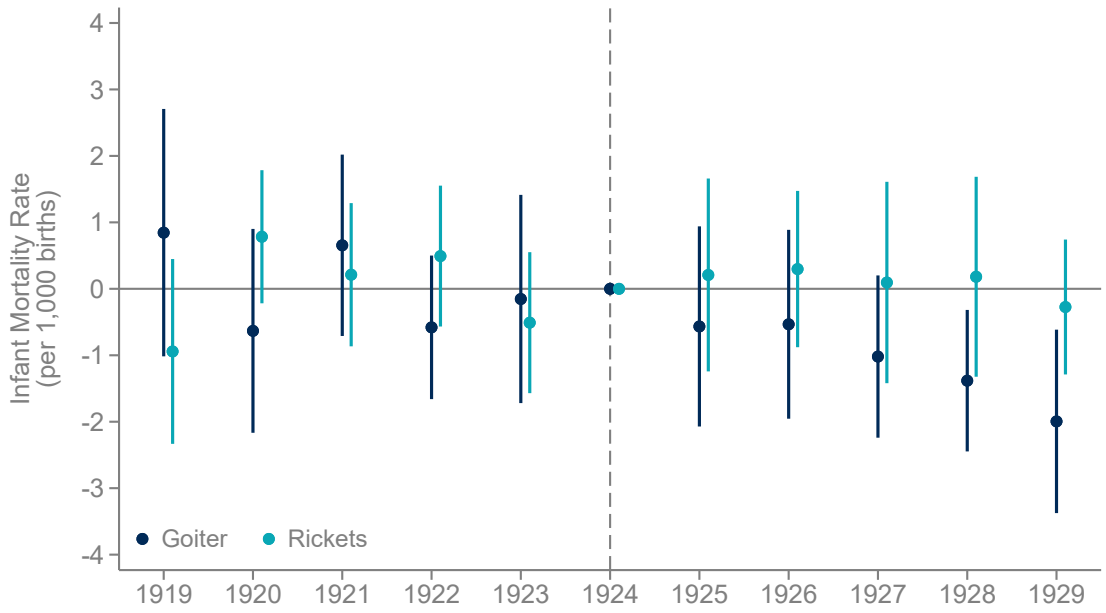


Figure A3: Infant Mortality Rate Rickets Placebo. This figure displays coefficients from an event study of infant mortality rates on Rickets rates. The event study uses the same specification as Column (1) in Table 2, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend.

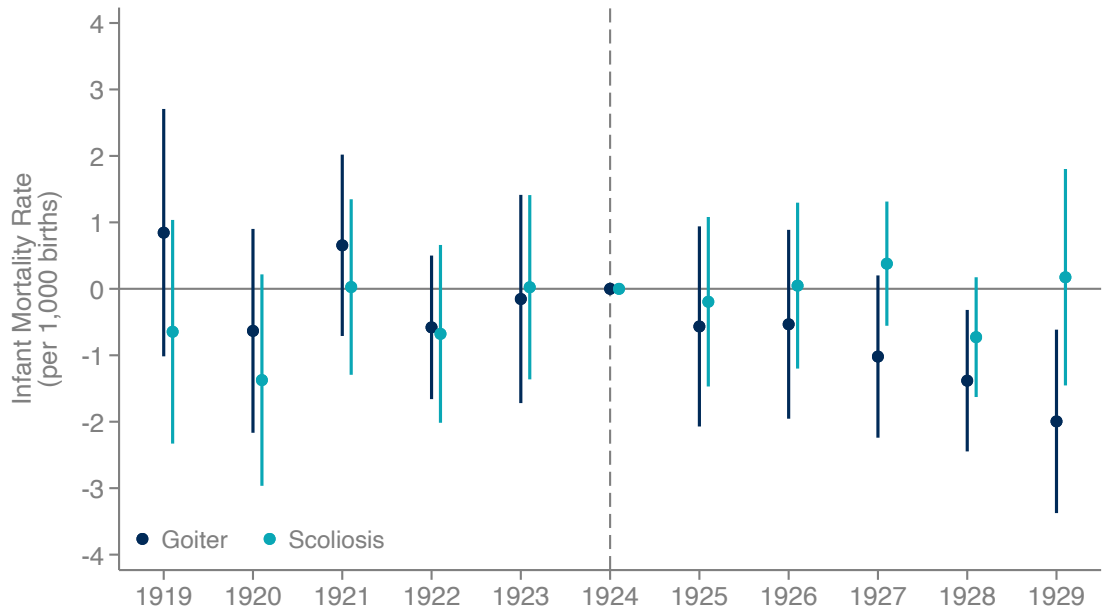


Figure A4: Infant Mortality Rate Scoliosis Placebo. This figure displays coefficients from an event study of infant mortality rates on Scoliosis rates. The event study uses the same specification as Column (1) in Table 2, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend.

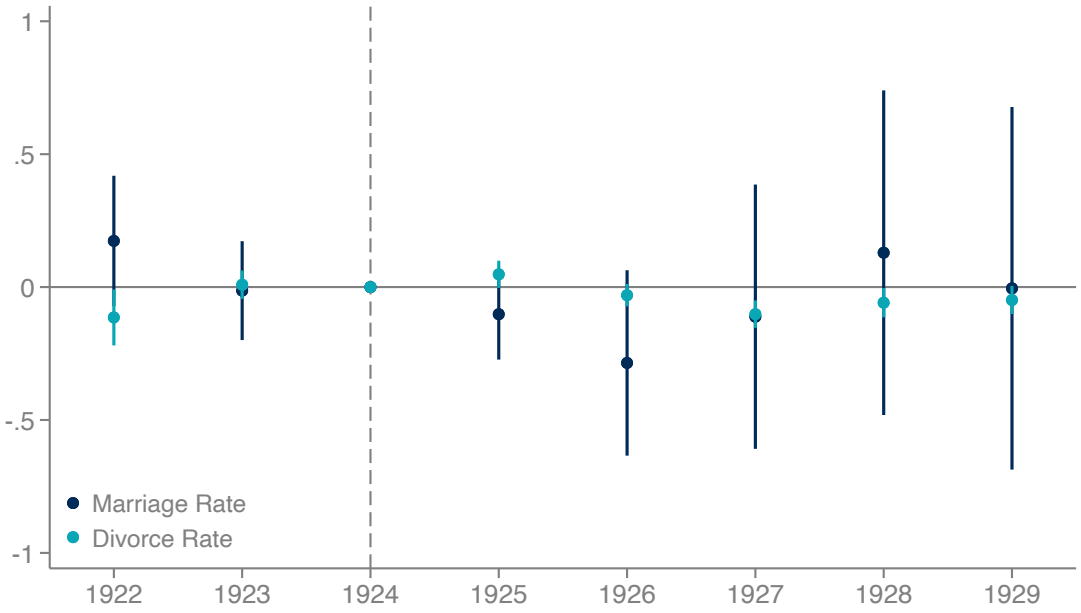


Figure A5: Marriage and Divorce Placebo. This figure displays coefficients from an event study of marriage and divorce rates on goiter rates. The event study uses the same specification as Column (1) in Table 2, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend.

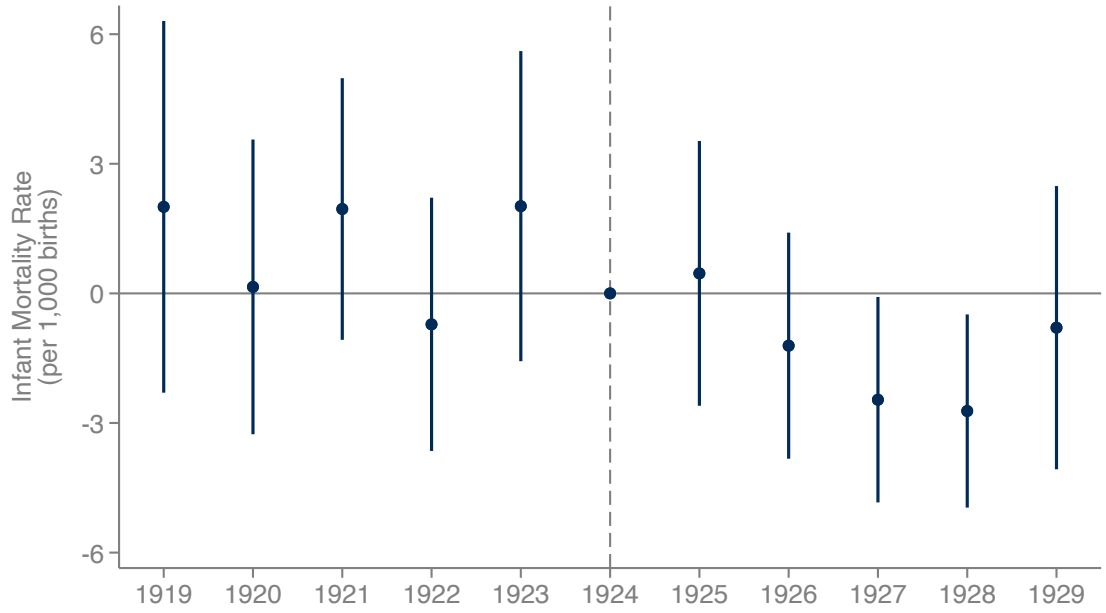


Figure A6: Infant Mortality Rate Discrete Treatment. This figure displays coefficients from an event study of infant mortality rates on an indicator for being in the top tercile of the within-Census division goiter distribution. The event study uses the same specification as Column (1) in Table A1, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend.

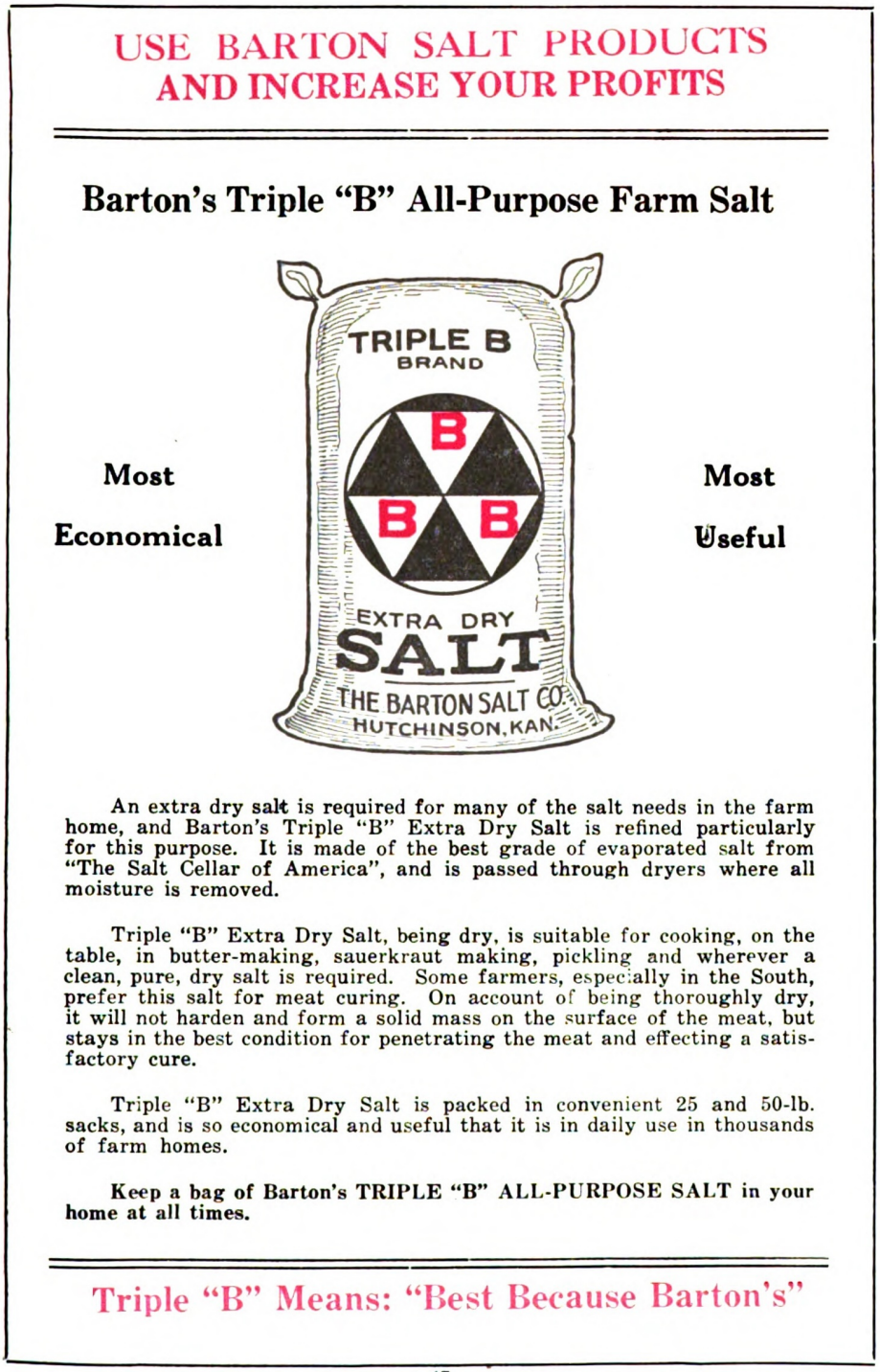


Figure A7: Barton Salt Advertisement. This advertisement displays a 25–50 lb. bag of salt that was typically purchased by farmers for home use. Salt sold in this bulk quantity was typically packaged in cloth bags which were not as effective in keeping out moisture as the smaller tin cans that were used for smaller scale distribution.

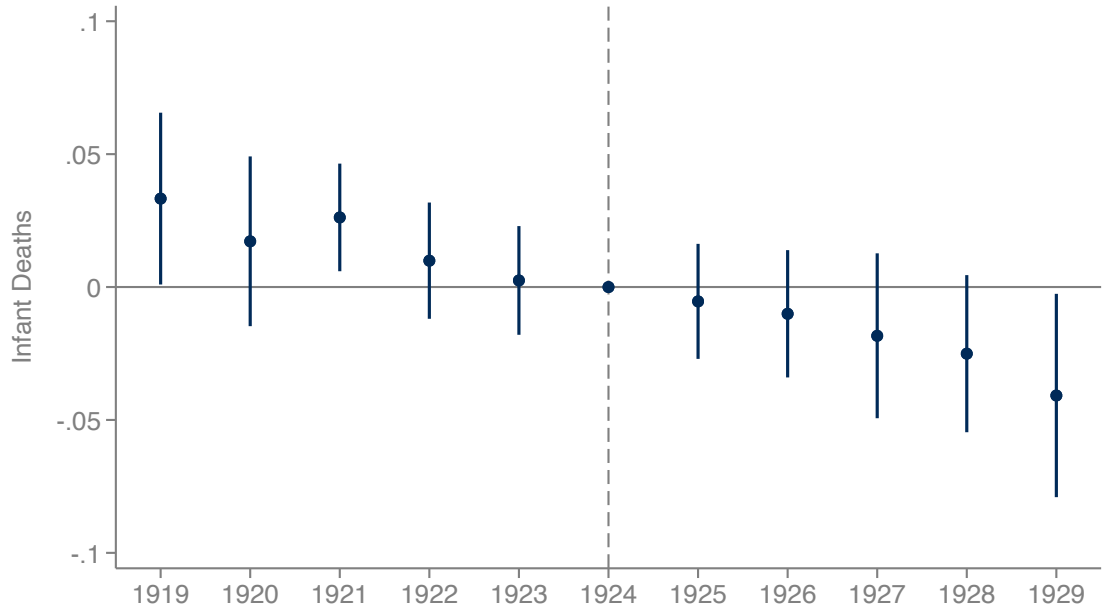


Figure A8: Impact on Infant Deaths – PPML. This figure displays coefficients from an event study of infant deaths on goiter rates. The event study uses the same specification as Column (4) in Table A2, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend.

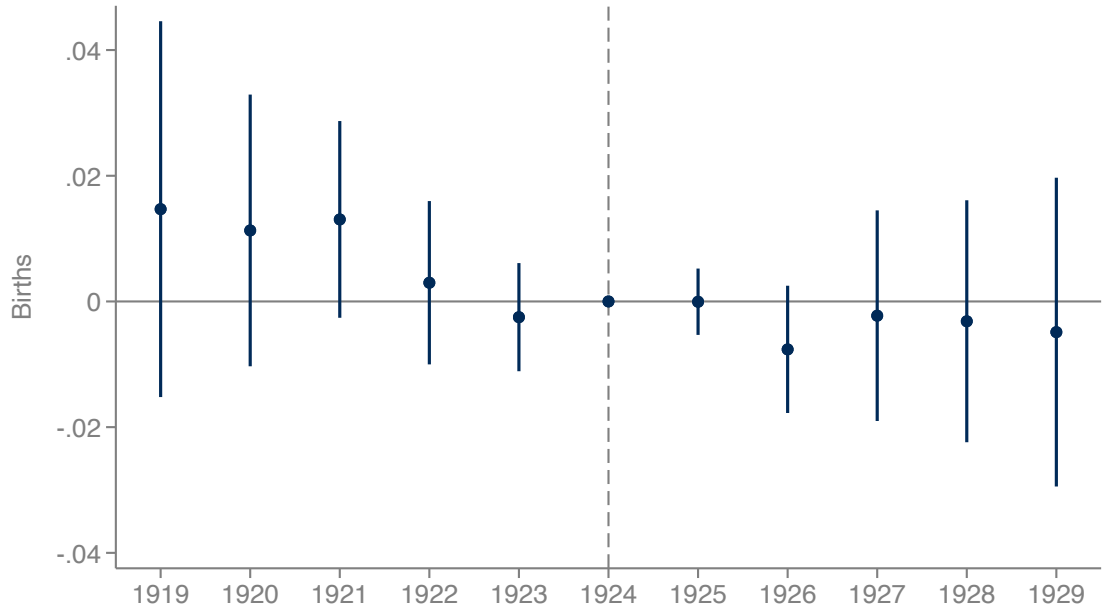


Figure A9: Impact on Births – PPML. This figure displays coefficients from an event study of births on goiter rates. The event study uses the same specification as Column (4) in Table A3, which includes county fixed effects, year fixed effects, region-by-year fixed effects, and 1920 county covariates interacted with a linear time trend.

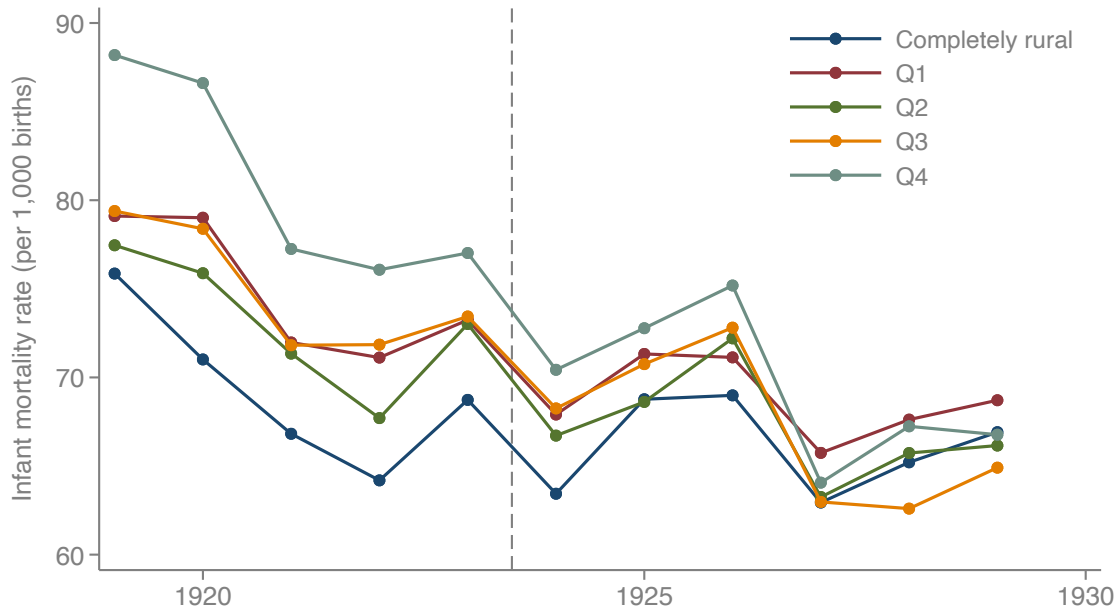


Figure A10: IMR by Urban Status. This figure displays the infant mortality rate separately for different groups of counties based on the percent of the population that is living in an urban area in 1920. The 'completely rural' bin is counties where zero percent of the population lives in an urban area. The Q1–Q4 bins are quartiles of counties with non-zero population in urban areas.

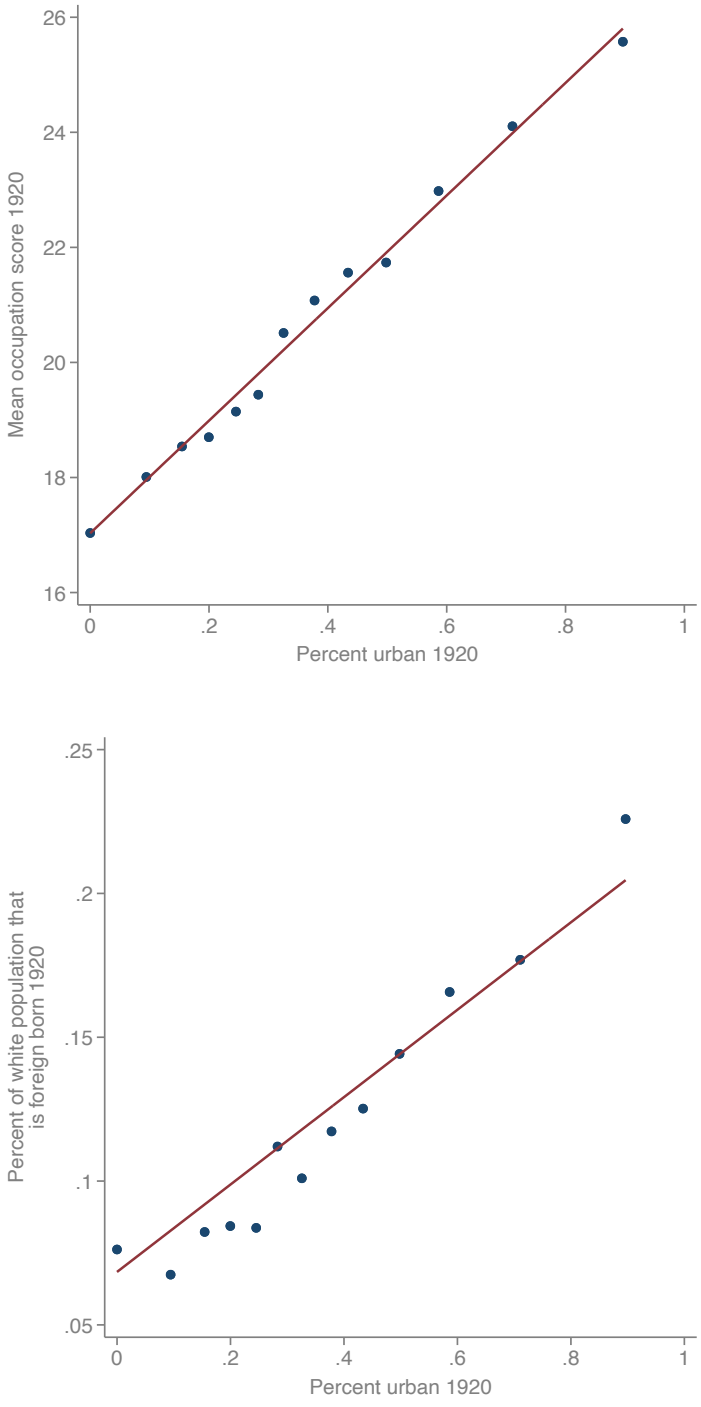


Figure A11: Correlates of Share Urban in 1920. This figure displays a binned scatterplot showing the relationship between the share of the population living in urban areas and other covariates in 1920 at the county level for the counties in our sample.

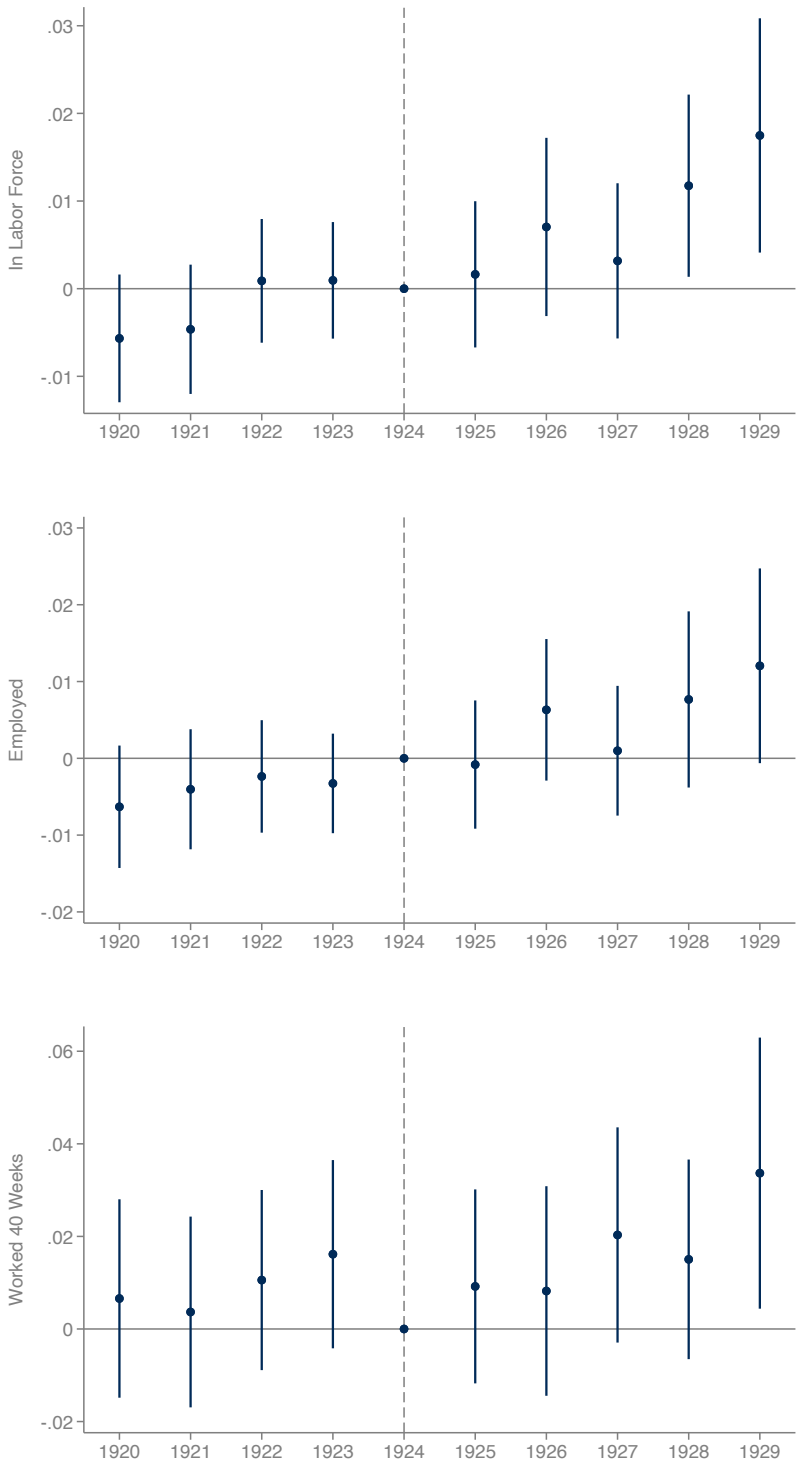


Figure A12: Long-Run Labor Market Impacts. This figure displays coefficients from an event study of various labor market outcomes on goiter rates. The event study uses the same specification as columns 4–6 in Table 6.

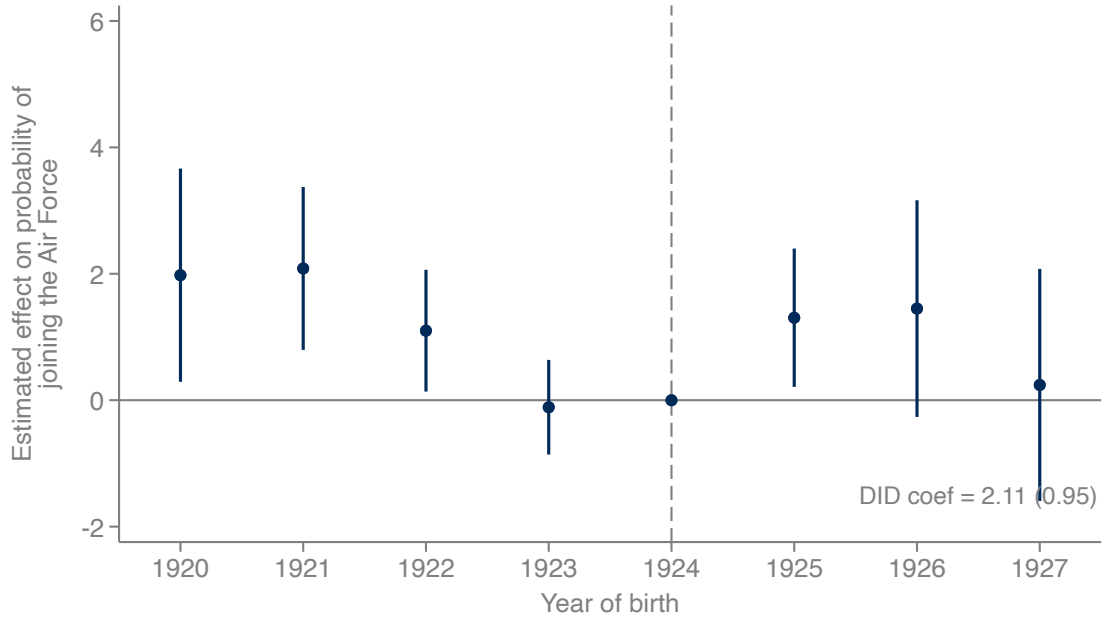


Figure A13: Long Run Impacts on Cognitive Ability – Rural Areas. This figure displays coefficients on the rural interaction from an event study of the probability of joining the Air Force on goiter rates fully interacted with an indicator for being born in an rural county. The event study uses the same specification as Column (4) in Table 3 of Feyrer et al. (2017). See Section 7.1 for more details.