Long Run Effects of Fortifying Grain Products with Folic Acid

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Abstract

How does prenatal nutrition affect children's long-run outcomes? This paper presents new empirical evidence from the 1996 folic acid fortification mandate for enriched grain products. Comparing cohorts exposed and unexposed to fortification across regions with different baseline folate deficiency, I find that in-utero fortification exposure shifts young adults' time from work to schooling: exposed young adults are 1.91-6.55 percentage points more likely to enroll in post-secondary schools, 1.22-4.17 p.p. less likely to work full time, and earn $1,280-$4,380 less per year. These effects are primarily driven by college-age cohorts. Based on a conservative back-of-the-envelope calculation, excluding long run human capital benefits would understate the net benefits of fortification by $1.85–$6.25 million per year. (JEL I18, J22, J24, N32, N52, Q18)

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1 Introduction

Early-life nutrition plays a critical role in long run human capital formation by supporting both physical and cognitive development (Ampaabeng and Tan, 2013; Portrait, Van Wingerden and Deeg, 2017). While evidence is mounting that early-life nutrition shocks have long-run effects on human capital, most economic studies focus on overall intake or macronutrients (Almond, Currie and Duque, 2018). Evidence on the long-run effects of micronutrients remains limited, despite their essential role in supporting key biological functions. This paper contributes new empirical evidence on the long run effects of prenatal nutrition on human capital outcomes by studying the folic acid fortification of enriched grain products in the late 90s.

Fortification is a cost-effective strategy to enhance access to micronutrients. The U.S. has a long history of fortifying foods with iodine, iron, and various vitamins. Folic acid fortification was the most recent effort to combat maternal deficiency in folate, a critical nutrient for neurodevelopment. Maternal folate deficiency, particularly concerning during pregnancy, can lead to severe birth defects and cognitive impairments in children (Roth et al., 2011; Irvine et al., 2022). To prevent these risks, the U.S. Food and Drug Administration (FDA) mandated the fortification of 40µg/100g of folic acid, i.e., the synthetic form of folate, in enriched grain products starting March 5, 1996. While public health literature widely recognizes the immediate benefits of folic acid fortification in reducing birth defects and improving infant health, its long-term effects on human capital remain underexplored.

I leverage geographical variation in pre-fortification birth defects tied to folate deficiency and the timing of folic acid fortification of grain products to assess the program's effect. Folic acid fortification effectively reduced folate deficiency (Wald et al., 2001), with greater benefits observed in regions with higher baseline deficiencies (Section 5.1). Folate is crucial for neural tube formation during the first trimester of pregnancy, and neurological damage during this stage is often irreversible. Thus, the effects of maternal exposure to folic acid fortification may manifest in later life stages. If fortification is effective, we should observe significant improvements in the outcomes of individuals exposed to folic acid fortification during early fetal development, particularly in regions with higher baseline folate deficiency. Due to the lack of large-scale data on maternal folate deficiency, I use the pre-fortification prevalence of birth defects tied to folate deficiency to capture maternal exposure to folic acid fortification. I then combine this spatial variation and the timing of fortification with the microdata on educational and labor outcomes from the American Community Survey (ACS), and estimate long-run effects of in-utero exposure to fortification using a cohort difference-in-differences (cohort-DiD)

approach.

I begin by documenting several first-stage facts: (i) measured folate content rose across a wide range of foods following fortification; (ii) dietary folate intake and serum folate concentrations increased; and (iii) the prevalence of folate-sensitive congenital anomalies declined, with larger reductions in places with higher baseline rates. Cohort difference-in-differences estimates indicate that in-utero exposure to fortification reallocated time from work toward schooling in young adulthood: post-secondary school enrollment increased by 1.91–6.55 percentage points (5.67–19.44% of the sample mean), full-time employment fell by 1.22–4.17 p.p. (2.68–9.17% of the sample mean), and annual earnings declined by $1,280–$4,380 (5.93–20.32% of the sample mean). These patterns are consistent with longer time spent in school.

Benchmarking against other nutrition policies, the implied human capital effects are comparable to those attributed to salt iodization and smaller than those estimated for the Food Stamps Program, a substantially larger intervention. To my knowledge, this is the first study to quantify the long-run human capital benefits of folic acid fortification. A conservative back-of-the-envelope calculation indicates that excluding these long-run gains from cost–benefit analyses would understate the program's net benefits by $1.85–$6.25 million per year.

This paper contributes to both economic and public health literature on food fortification. Most economic studies have examined the long-term benefits of salt iodization on cognitive development, health, and socioeconomic outcomes—such as improved cognitive performance and higher earnings (Feyrer, Politi and Weil, 2017; Serena, 2019; Adhvaryu et al., 2020; Huang, Liu and Zhou, 2020; Deng and Lindeboom, 2022*a*; Tafesse, 2022). One exception is (Niemesh, 2015), which finds that iron fortification of bread increased working-age adults' incomes and school enrollment and raised their children's long-run wages. In contrast, evidence on the human capital effects of folic acid fortification is scarce. Biologically, the case for folate is strong: unlike iodine and iron deficiencies—which primarily impair thyroid function and oxygen transport, respectively—folate deficiency directly disrupts neural development and can have more severe and lasting consequences. Folic acid fortification could therefore exert stronger effects on cognitive development and downstream outcomes such as schooling and earnings. Adoption has also been far less widespread globally than salt iodization or iron supplementation, particularly in developing countries, so causal evidence from the United States can inform policy design elsewhere. The public health literature has emphasized short-run health effects of folic acid supplementation (e.g., Wald et al., 2001; Quinlivan et al., 2002; Kancherla et al., 2022) and cost–benefit analyses of fortification (e.g., Grosse et al., 2005; Bentley et al., 2009;

Llanos et al., 2007). By examining long-run human capital outcomes, this study extends that work and provides new causal evidence on the broader developmental consequences of folic acid fortification.

More broadly, this paper also relates to the fetal origins literature. A large body of research shows that prenatal and early childhood nutritional conditions have enduring consequences. Adverse shocks, such as famine (Meng and Qian, 2006; Almond et al., 2007; Chen and Zhou, 2007; Meng and Qian, 2009; Lindeboom, Portrait and Van den Berg, 2010; Scholte, Van Den Berg and Lindeboom, 2015; Deng and Lindeboom, 2022*b*) and Ramadan fasting (Almond and Mazumder, 2011; Almond, Mazumder and Van Ewijk, 2015; Majid, 2015; Greve, Schultz-Nielsen and Tekin, 2017), have been linked to poorer health and labor market outcomes in adulthood. In contrast, positive interventions, such as breastfeeding (Fitzsimons and Vera-Hernández, 2022), iodine supplementation (Field, Robles and Torero, 2009; Araújo, Carrillo and Sampaio, 2021), and food assistance programs like WIC and food stamps (Hoynes, Page and Stevens, 2011; Rossin-Slater, 2013; Hoynes, Schanzenbach and Almond, 2016; Bailey et al., 2024), have been shown to support cognitive development and improve long-term socioeconomic outcomes. This study extends this literature by examining the long run impacts of prenatal exposure to a previously unstudied intervention.

The paper is organized as follows: Section 2 provides the policy backgrounds; Section 3 describes the data; Section 4 outlines the research design and discusses identifying assumptions; Section 5 presents results; Section 6 discusses robustness; and Section 7 concludes.

2 Background

2.1 Folate deficiency disorder and associated birth defects

Folate deficiency is a major cause of neural tube defects (NTDs), the most common congenital anomalies of the central nervous system (CNS) in newborns (Smithells et al., 1983). Severe NTDs, such as anencephaly, are typically fatal, with most affected infants dying before or shortly after birth.[1] Infants with less severe NTDs, like spina bifida, can survive into adulthood but often carry a high risk of lifelong physical and mental disabilities (Yi et al., 2011).[2] In the early 1990s, approximately 4,000 fetuses in the U.S. (about 1 in 1,000) were affected by NTDs annually, with one-third lost due to selective or spontaneous abortions (Cragan et al., 1995; Mersereau et al., 2004). Folate deficiency can also lead to other congenital CNS anoma-

[1] Infants with anencephaly are born without parts of the skull and brain.

[2] The backbone of infants with spina bifida does not close properly, leaving a section of the spinal cord and spinal nerves exposed to the outside without the protection of the backbone.

lies, such as hydrocephaly (Naz et al., 2016; Liu et al., 2018). These birth defects can develop as early as the first month of pregnancy when the neural tube begins to form, and failure to close the neural tube by the end of the first trimester can cause irreversible damage to the central nervous system (Obeid, Holzgreve and Pietrzik, 2013). While in-utero surgery may offer some palliative benefits, such neurological damage remains irreversible (Greene and Copp, 2014). Timely medical intervention is often difficult, as most ultrasound screenings occur in the second trimester—when anomalies are easier to detect—and many pregnant women in the U.S. lack adequate prenatal care (Blumenfeld, Siegler and Bronshtein, 1993).

2.2 Sources of folate

Folate exists naturally in foods such as beef liver, dark green leafy vegetables, beans, peas, nuts, and a variety of fruits and fruit juices. However, meeting the recommended intake during pregnancy through diet alone is challenging (Czeizel, 2000). Data from the National Health and Nutrition Examination Surveys (NHANES) III (1988–1994) show that women ages 15–49 consumed an average of 233.68 μg of folate per day, well below the 400 μg recommended by the U.S. Public Health Service for pregnant women. One reason dietary intake falls short is that natural food folate is unstable under typical cooking conditions, which can substantially reduce the amount ultimately absorbed, making it a less reliable way to improve folate status during pregnancy (McNulty and Pentieva, 2004).

Folate is also available from nutritional supplements, including over-the-counter folic acid tablets and multivitamins sold in pharmacies. Folic acid supplements are often prescribed during prenatal visits (Ray, Singh and Burrows, 2004). A key challenge, however, is low awareness of and adherence to supplementation recommendations (Toivonen et al., 2018). According to CDC guidance,[3] folic acid should be taken starting at least one month before conception. Yet about 50% of U.S. pregnancies are unintended (Finer and Zolna, 2016). From 1995 to 1998, only about 30% of U.S. women reported taking a daily vitamin containing folic acid, and fewer than 10% knew it should be taken before pregnancy (Petrini, Damus and Johnston, 1999). Access and affordability also pose barriers, particularly for low-income women (Czeizel, 2000). These constraints point to the need for a low-cost, preferably passive approach to ensure adequate folic acid intake among women who may become pregnant.

[3] See https://www.cdc.gov/ncbddd/folicacid/recommendations.html (accessed on 05/20/2022).

2.3 Folic acid fortification and other fortifications in the U.S.

The United States has a long history of using food fortification to improve public health. Salt iodization began in the 1920s, vitamin D fortification of milk followed in the 1930s, and flour and bread were enriched with B vitamins and iron in the 1930s and 1940s. The most recent effort, folic acid fortification of grain products, started in the 1990s. The first wave of grain fortification in the 1940s followed the identification of specific nutrient deficiency disorders in the U.S. In the early 1940s, the FDA issued the first standard of identity for enriched flour, requiring the addition of iron and B vitamins (niacin, thiamin, and riboflavin). By the 1950s, these standards extended to other cereal grain products, including bread, rice, macaroni, and noodles (Hutt, 1984; Committee on Use of Dietary Reference Intakes in Nutrition Labeling, 2004). Folic acid fortification is the most recent amendment to the standard of identity for enriched grain products and is widely regarded as one of the most successful public health initiatives in recent decades (Berry, Mulinare and Hamner, 2010).

As with earlier fortification campaigns, the folic acid policy was driven by accumulating evidence that folic acid prevents neural tube defects (NTDs). In October 1990, as part of the Nutrition Labeling and Education Act, Congress directed the FDA to evaluate the link between folic acid and NTDs and to develop a plan for adding folic acid to foods (Wright, 2003). On September 14, 1992, the United States Public Health Service (USPHS) recommended that all women of childbearing age consume 400 μg of folic acid daily to prevent NTDs. In response, on March 5, 1996, the FDA amended the standard of identity to require 140 μg/100 g of folic acid in enriched grain products by January 1, 1998 (Food and Drug Administration, 1996). In practice, fortification was largely completed by mid-1997 (Jacques et al., 1999). I therefore define event time as March 1996—the month the FDA authorized folic acid fortification. Because enriched wheat flour is used in many processed foods, fortification extended beyond breads and pastas; for example, some chips contain folic acid (Figure 1). Before the mandate, voluntary addition of folic acid was prohibited in standardized foods[4] and discouraged in other products to avoid overfortification and nutrient imbalances in the population (Food and Drug Administration, 1996, 2015).

[4] "Standardized foods" are products with a federal standard of identity, such as enriched grain products.

FIGURE 1: CHIPS WITH ENRICHED WHEAT FLOUR AS AN INGREDIENT

3 Data

3.1 Birth certificate data

The Vital Statistics Natality files cover all U.S. live births and report detailed birth outcomes (birth month/year, county of birth, birth weight, gestational age in weeks, and congenital anomalies) and maternal characteristics (age, race, Hispanic origin, education, and prenatal care use) (National Center for Health Statistics, 2003). I use these data for two purposes. First, I proxy baseline folate deficiency with the pre-fortification prevalence of folate-sensitive congenital anomalies and, combining gestational age with the policy's authorization and rollout dates, assign in-utero exposure at the cohort level (Section 4.2). Second, I test whether exposure to fortification changed the distribution of infant and maternal characteristics, to rule out compositional shifts as an explanation for the main results.

3.2 Outcome data

Outcome data are drawn from the American Community Survey Public-Use Microdata Sample (ACS PUMS), 2016–2023 (Ruggles and Williams., 2025). I focus on young adults because the earliest fully exposed cohorts, who were conceived after the March 1996 authorization and born in the fourth quarter of 1996, are in their twenties during these waves. The analysis sample includes individuals ages 19–29. I study two domains of human capital. For education, I examine high school completion (diploma or GED) and current enrollment in post-secondary education (college or graduate/professional school). For labor markets, I consider labor force participation, employment, full-time status, and annual earnings.

3.3 Other data

(i) Baseline county characteristics. I compile baseline county characteristics from multiple sources. Demographic data on race, gender, age, and total population come from the Intercensal Population Estimates (US Census Bureau, 1990). Birth and death rates, the unemployment rate, the value of products sold per farm, and average farm size come from the County and City Data Book (1988) (US Census Bureau, 2009). Transfer payments are from the Bureau of Economic Analysis's Regional Economic Information System (REIS) (Bureau of Economic Analysis, 1988). These data are combined with exposure data from birth certificates for balance test (Table A1)

(ii) Time-varying covariates. In the baseline specification, I control for local economic conditions using a Bartik-style unemployment measure, following Ganong and Liebman (2018) and East (2020). For each state of birth, I interact pre-policy sectoral employment shares from the BLS Quarterly Census of Employment and Wages (QCEW) with annual national changes in sectoral unemployment rates and sum across sectors to obtain a predicted state unemployment rate (US Bureau of Labor Statistics, 1989-2002). This approach mitigates concerns that fortification could mechanically influence the contemporaneous unemployment rate. I also include time-varying controls for potentially confounding policies: (i) Medicaid/SCHIP eligibility for pregnant women, as estimated by Hoynes and Luttmer (2011); (ii) mental health parity laws; (iii) an indicator for the first major AFDC waiver; and (iv) an indicator for the implementation of TANF. I first assign these exposures at the birth record level using the Natality files and then aggregate to the state–cohort level to align with the definition of fortification exposure.

4 Methods

4.1 Empirical strategy

An ideal empirical strategy would be a randomized controlled trial assigning pregnant women to receive folic acid supplements and following their children into adulthood to compare outcomes. This approach is not feasible at scale. Instead, I use the 1996 U.S. folic acid fortification of grain products as a natural experiment to estimate the long-run human capital effects of prenatal folic acid supplementation.

My approach parallels studies that leverage baseline regional disease prevalence to estimate the benefits of health interventions. For example, researchers have used baseline hookworm infection rates to study hookworm eradication (Bleakley, 2007), malaria prevalence to evaluate malaria eradication (Bleakley, 2010; Kuecken, Thuilliez and Valfort, 2021), measles in-

cidence to assess vaccination (Atwood, 2022), pneumonia rates to examine the introduction of sulfa antibiotics (Lazuka, 2020), and goiter prevalence to analyze salt iodization (Feyrer, Politi and Weil, 2017; Adhvaryu et al., 2020).

To identify the effects of folic acid fortification, I use a cohort DiD design that compares cohorts exposed and unexposed to the fortification in utero in states with high and low baseline CNS anomaly rates. My baseline regression model is:

$$Y_{istc} = \sum_{\tau \neq 1995} \beta_\tau \cdot \mathbf{1}\{\text{CNSA top quartile}\}_s \times \mathbf{1}\{t \in \tau\} + \mu_s + \lambda_t + \gamma_c + C_{istc} + \varepsilon_{istc} \quad (1)$$

where Y_{istc} represents the outcome for individual i who born in state s and quarter-and-year t recorded in survey year c, $\mathbf{1}\{\text{CNSA top quartile}\}_s$ is a dummy for the states with the highest quarter of baseline CNS anomaly rates,[5] $\mathbf{1}\{t \in \tau\}$ is a dummy for each birth cohort except the reference cohort 1995, μ_s is state-of-birth fixed effects to account for cohort-invariant unobserved heterogeneity, λ_t is quarter-and-year-of-birth fixed effects to control for cohort-specific shocks, γ_c is survey year fixed effects to control for unobservables related to age,[6] C_{ist} is a set of control variables including (i) individual characteristics, including gender, race dummies, and Hispanic origin, (ii) exposure measures of confounding policies including Medicaid/CHIP expansion, welfare reform, and state mental health parity laws (Section 3.3), and (iii) a Bartik-style measure of state-by-year unemployment rate to control for local economic conditions.

The coefficients β_τ are the primary parameters of interest, which capture the dynamic effects of in-utero exposure to folic acid fortification across cohorts. Because my outcome data does not identify who is folate-deficient, β_τ should be interpreted as intent-to-treat (ITT) effects. Below, I approximate treatment-on-the-treated (TOT) effects by scaling the ITT using the estimated pre-fortification share of folate-deficient women ages 19–45 from NHANES.

Several prior studies model treatment intensity using continuous, dose–response DiD designs. In this setting, that approach raises two concerns. First, the relationship between folate deficiency and cognitive development may be non-monotonic (e.g., thresholds or saturation), so interpreting a single slope from a continuous-dose model relies on functional-form and monotonicity assumptions that may not hold. Second, identification in dose–response DiD requires stronger parallel-trends conditions, i.e., parallel trends within each dose level and stable composition across dose cells, which are demanding here (Callaway, Goodman-Bacon

[5] This results in 14 high-exposure states, including IN, IA, KS, MD, MN, NE, NJ, NY, ND, RI, SD, TN, TX, and VT.

[6] Because we can obtain age from survey year and year of birth, controlling for age fixed effects is equivalent to controlling for survey year fixed effects.

and Sant'Anna, 2024). For transparency, the baseline specification adopts a binary design contrasting high- and low-baseline-risk states (defined by pre-fortification prevalence of folate-sensitive anomalies). I nonetheless report dose–response estimates in Section 5.5 as a robustness check; they are directionally consistent with the binary results but less precise, consistent with the concerns discussed above.

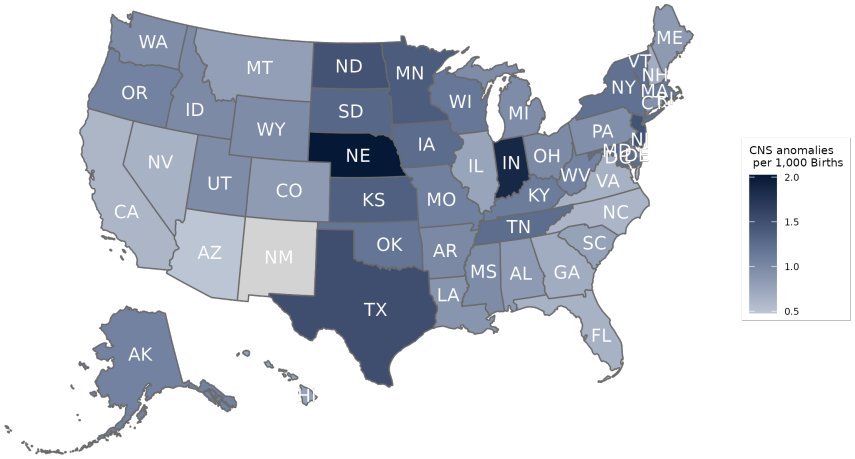
4.2 Exposure measure

I proxy exposure to folic acid fortification using baseline shares of infants diagnosed with central nervous system (CNS) anomalies. Birth certificates record five categories of CNS anomalies: spina bifida, anencephaly, hydrocephaly, microcephaly, and "other" CNS anomalies. Folate deficiency is a leading cause of neural tube defects (NTDs) (Wald et al., 2001). Spina bifida and anencephaly are the most common NTDs, and other NTD subtypes are grouped under "other" CNS anomalies. Folate deficiency can also contribute to hydrocephaly and microcephaly, directly or indirectly through NTDs (Abdel-Salam and Czeizel, 2000; Naz et al., 2016; Liu et al., 2018). Overall, the medical literature indicates that folic acid supplementation substantially reduces the risk of CNS anomalies.

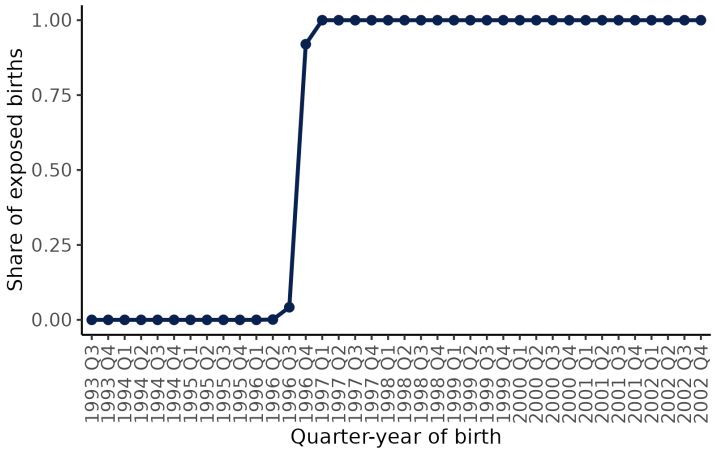
I define the baseline period as January 1989–June 1993. Most states began reporting congenital anomalies in 1989; the exceptions are Louisiana (1990), Nebraska (1990), Oklahoma (1991), New York (1993), and New Mexico (not reported during the study period). This window maximizes state coverage—all states and the District of Columbia except New Mexico. Limiting the baseline to the first half of 1993 ensures that cohorts born afterward have at least four pre-periods for the event-study analysis. Figure 2a presents the baseline CNS anomaly rates across states.

I determine exposure timing using weeks of gestation recorded on birth certificates. An infant is classified as exposed if the first trimester ends after March 1996 (the month fortification was authorized), since neural tube closure occurs by the first trimester and folic acid reduces the risk of CNS anomalies by helping the neural tube close properly. Next, I aggregate the birth-level exposure indicator to the quarter–year level. As shown in Figure 2b, the share exposed during the first trimester rises sharply starting with births in 1996 Q4. I therefore define individuals born from 1996Q4 onward as the exposed group. This pre–post timing, combined with spatial variation in baseline CNS anomaly rates, provides the key identifying variation in my empirical strategy.

I validate the exposure measure with the following two exercises. First, using NHANES III, I map state-level pre-fortification CNS anomaly rates to biomarkers of folate status and find a



(A) Baseline CNS anomaly rates by state



(B) Share of exposed births by quarter-and-year-of-birth

FIGURE 2: SPATIAL AND TEMPORAL VARIATION IN FORTIFICATION EXPOSURE

Notes: In Figure 2a, I compute baseline CNS anomaly rates from the restricted-use Natality files and aggregate them to the state of birth. The baseline window is January 1989–June 1993. In Figure 2b, a birth is classified as exposed if its first trimester ends after March 1996, when folic acid fortification was authorized. I then aggregate the birth-level exposure indicator to county–quarter–year cell averages.

negative association with both serum and RBC folate (Table 1). Second, I show that trends in CNS anomalies diverge by baseline risk: high-exposure regions exhibit larger post-fortification declines (Figure 5b; see Section 5.1).

4.3 Identifying assumptions

The validity of this empirical strategy relies on two assumptions: parallel trends and no anticipation. Parallel trends require that, absent folic acid fortification, average outcomes for high- and low-exposure groups would have evolved similarly across birth cohorts. Although this is not directly testable because we do not observe the counterfactual, I conduct partial checks of its plausibility. First, I examine whether exposure is correlated with pre-1989 county- or

TABLE 1: CORRELATION BETWEEN BASELINE CNS ANOMALY RATE AND FOLATE BIOMARKERS

	Serum foalte (1)	RBC folate (2)	Serum foalte (3)	RBC folate (4)
CNS anomaly rate	-0.5375**	-11.41**		
	(0.2607)	(4.572)		
1{CNSA top quartile}			-0.6059***	-10.55***
			(0.1854)	(3.171)
Observations	10,842	10,913	10,842	10,913
R^2	8e-04	0.0014	0.0021	0.0025
Est./Dep. var. mean	106.91%	105.42%	102.36%	101.52%

Notes: Dependent variables are individual-level folate measure. In parentheses are heteroskasticity-robust standard errors. Regressions are weighted by MEC final examination sample weights. Data source is public-use NHANES iii. Geographical identifiers that are not suppressed include 35 counties from 13 states. CNS anomaly rate is measured at state level.

state-level characteristics. A violation could arise if prenatal high-exposure areas systematically differ in factors that also influence long-run outcomes. For example, if poorer states happened to have higher CNS anomaly rates and economic conditions at birth independently affect adult outcomes, estimates might simply reflect underlying economic conditions rather than the effect of fortification. I then regress the exposure measure, $\mathbf{1}\{\text{CNSA top quartile}\}_s$, on these baseline characteristics. Table A1 shows that while some covariates are individually correlated with high exposure, as a whole they explain only about 30% of cross-state variation and less than 10% of cross-county variation, suggesting the high-exposure designation is largely orthogonal to observables and consistent with the parallel trends assumption.[7]

Second, I present dynamic effects using an event-study design to examine the presence of any pre-treatment trends. In most cases, we do not observe evidence of such trends. In Section 6, I add time-varying covariates to relax the unconditional parallel trends assumption underlying the baseline results. With these covariates included, the parallel trends assumption needs only to hold conditional on them.

The no-anticipation assumption requires (i) that mothers in high-exposure areas did not alter behavior in advance of fortification, and (ii) that food manufacturers did not begin fortifying before March 1996. In this setting, anticipatory responses are unlikely. (Petrini, Damus and Johnston, 1999) indicates low awareness of folic acid among women of childbearing age. Because the fortification mandate was motivated by scientific evidence and directed at food

[7] Fully explaining cross-state differences in baseline CNS anomaly rates is beyond the scope of this paper. Salient contributors likely include maternal health conditions, dietary patterns, and use of folic acid supplements (with possible roles for surveillance and reporting practices). Rather than model these determinants, I validate my research design using the partial test described above. While this test does not rule out all confounding, it increases confidence that the exposure measure is orthogonal to a wide set of observables and supports the plausibility of the parallel-trends assumption in the difference-in-differences design.

manufacturers, it likely had low salience for the general public. On the supply side, voluntary folic acid fortification was prohibited for standardized foods and discouraged elsewhere due to concerns about overfortification and nutrient imbalances (Food and Drug Administration, 1996, 2015). Consistent with these arguments, I do not observe any evidence for anticipatory behavior in my event-study results.

5 Results

5.1 Descriptive first-stage evidence

(i) Folate content in foods increased after folic acid fortification. First, folic acid content in foods rose markedly after fortification. Using the 1994–1996 and 1998 waves of the Continuing Survey of Food Intakes by Individuals (CSFII), I compare per-serving folic acid in the same foods before and after the mandate, based on USDA's recipe-based nutrient calculations. Because the CSFII records the reason for composition changes, including enrichment/fortification, reformulation, agricultural or processing modifications, and implementation of the Nutrition Labeling and Education Act. I can isolate changes attributable to fortification. As shown in Figure 3, folic acid increased across a wide range of products, from white bread to snack/cookie bars; in total, more than 350 basic food items show higher folic acid due to fortification (Anderson et al., 2001).

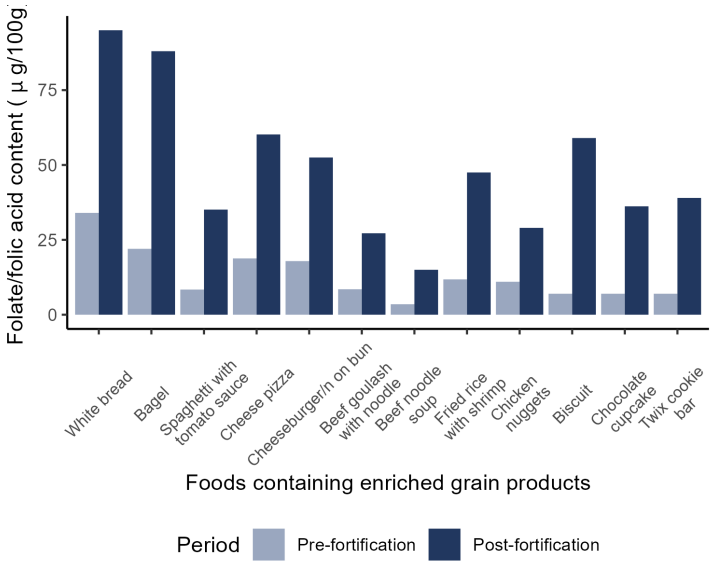
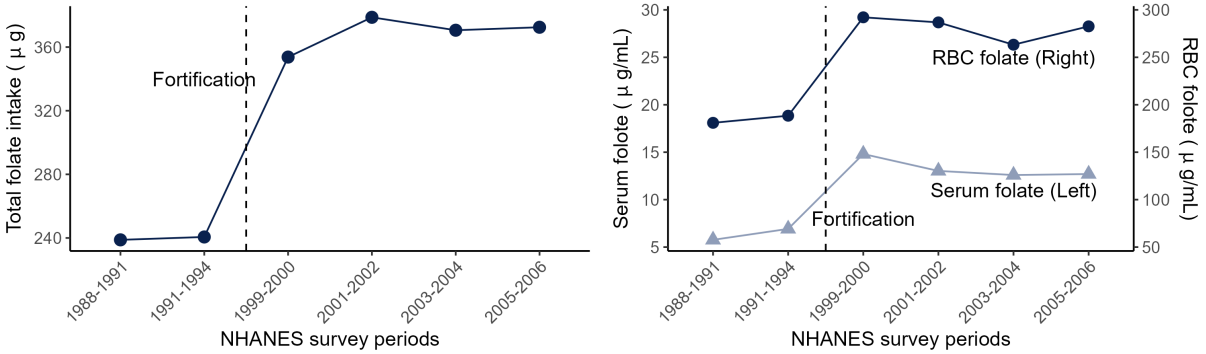


FIGURE 3: CHANGES IN FOLATE CONTENTS IN SELECTED FOODS DUE TO FORTIFICATION

Notes: Data on food folate content is from USDA Continuing Survey of Food Intakes by Individuals (CSFII) 1994-1996 and 1998. Folate content is estimated by USDA based on recipe. Changes in folate content in this graph are solely due to fortification.

(ii) Dietary folate intake and blood folate increased after folic acid fortification. Second,

using NHANES, I document sharp increases in dietary folate intake and blood folate among women ages 19–45 following fortification. Mean dietary intake rose by nearly 50% (Figure 4a). The share with intake below 400 μg/day fell from 98.65% to 69.87% ($\Delta = -28.78$ percentage points). These intake measures exclude folic acid from supplements and medications (Ahluwalia et al., 2016). Biomarkers show parallel gains: serum folate more than doubled and red blood cell (RBC) folate rose by nearly 50% (Figure 4b), indicating sustained improvements in folate status. Blood folate measurement methods were stable over 1999–2006 (Pfeiffer et al., 2012).



(A) Dietary folate concentrations before and after fortification

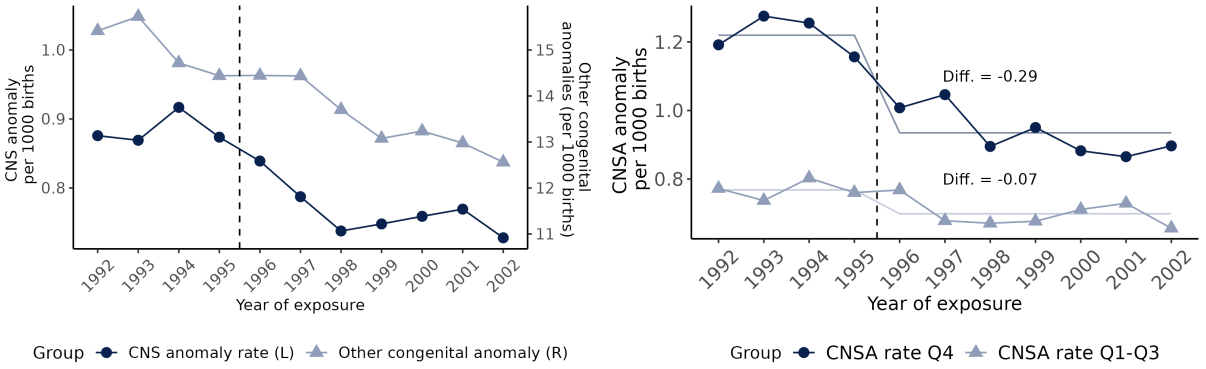
(B) Blood folate concentrations before and after fortification

Notes: Data is from harmonized NHANES data cleaned by Nguyen et al. (2023) to ensure comparability of folate measures across survey periods. Mobile examination center (MEC) final examination sample weights are used for all folate measures in all survey periods.

(iii) Congenital anomalies declined after folic acid fortification. Finally, Figure 5a shows that as folate intake and absorption rose, CNS anomaly rates declined. After a flat period from 1992 to 1996, incidence fell sharply following fortification. This pattern is unlikely to reflect broader healthcare improvements, as rates of other congenital anomalies remained stable over the same period. Figure 5b further compares trends by exposure (defining high exposure as the top quartile of baseline CNS anomaly rates): rates fell in both high- and low-exposure regions, with a larger decline in high-exposure areas, supporting the validity of the exposure measure used in my research design.

5.2 Long run effects on school enrollment

Figures 6a-6b display cohort-specific estimates of the effects of in utero exposure to folic acid fortification on (1)the likelihood of earning a high school diploma or GED and (2) the likelihood of enrolling in post-secondary schools (college or graduate/professional school). Figure 6a shows no detectable effect of in utero exposure to folic acid fortification on the probability of earning a high school diploma or GED. The null is plausible given the high baseline: over 90%



(A) CNS and non-CNS anomaly rates

(B) CNS anomaly rates in high- and low-exposure regions

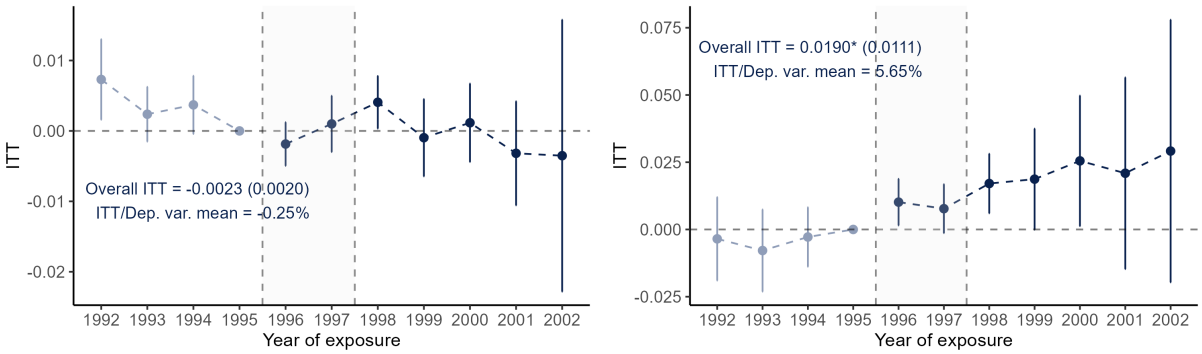
FIGURE 5: TRENDS IN CONGENITAL ANOMALY RATES

Notes: The unit of CNS Anomaly rate is cases per 1,000 births. High exposure is defined as the top quartile baseline CNS anomaly rate.

of young adults already hold a high school diploma or GED, leaving little room for additional gains even if exposure improved cognitive ability. Bailey et al. (2024) observe a similar pattern for food stamps: early-life exposure to food stamps increased college attendance and years of schooling but did not raise high school or GED completion.

Figure 6b shows that young adults exposed to fortification in utero are 1.9 p.p. more likely to enroll in post-secondary schools (5.65% at the sample mean), with no noticeable evidence of pre-treatment trends. Coefficients for the fully exposed cohorts (1998–2002) are generally larger than those for the partially exposed cohorts (1996–1997): partially exposed cohorts are 0.77–1.02 percentage points (p.p.) more likely to enroll, though the 1997 estimate is somewhat imprecise; fully exposed cohorts (1998-2000) are 1.71–2.55 p.p. more likely to enroll. Estimates for the 2001 and 2002 cohorts are larger but imprecise. I summarize the overall effects of fortification exposure in Table A2 and the cohort-specific effects in Table A3.

In Section 5.1, I use NHANES to estimate that 98.65% of women aged 19–45 were folate-deficient before fortification, and that the share deficient fell by 28.78 percentage points afterward. I use these figures to bound treatment-on-the-treated (TOT) effects from the reduced-form ITT estimates. Let $p_{\text{def}}^{\text{pre}} = 0.99$ and $\Delta p_{\text{def}} = 0.29$. If all previously deficient women benefited, the implied treatment rate is $p_{\text{def}}^{\text{pre}}$, yielding a lower bound $\text{TOT}_L = \text{ITT}/p_{\text{def}}^{\text{pre}}$. If only those whose deficiency was corrected benefited, the relevant treatment rate is Δp_{def}, yielding an upper bound $\text{TOT}_U = \text{ITT}/\Delta p_{\text{def}}$. Thus, $\text{TOT} \in \left[\text{ITT}/0.99,\ \text{ITT}/0.29\right]$, with the lower (upper) bound corresponding to broader (narrower) treatment incidence assumptions. Therefore, the TOT of in-utero fortification exposure on post-secondary school enrollment lies between 1.91 and 6.55 p.p. (5.67%-19.44% at the sample mean).



(A) High school diploma or GED

(B) Post-secondary school enrollment

FIGURE 6: LONG RUN EFFECTS OF FOLIC ACID FORTIFICATION ON YOUNG ADULTS' EDUCATIONAL OUTCOMES

Notes: The figure plots cohort-specific (dynamic) estimates with 95% confidence intervals. The treated group is the births whose first trimester ended after the March 1996 authorization of folic acid fortification and who were born in states in the top quartile of baseline CNS anomaly rates. I control for state-of-birth fixed effects; quarter-by-year-of-birth and survey-year fixed effects; and controls for gender, race, Hispanic origin, Medicaid eligibility, exposure to mental-health parity laws and welfare reforms, and a Bartik-style measure of local unemployment rate. The shaded region denotes cohorts with partial exposure. The unit of observation is individuals. Regressions and dependent-variable means are weighted by the IPUMS person weight. Standard errors are clustered by state of birth.

5.3 Long run effects on labor supply

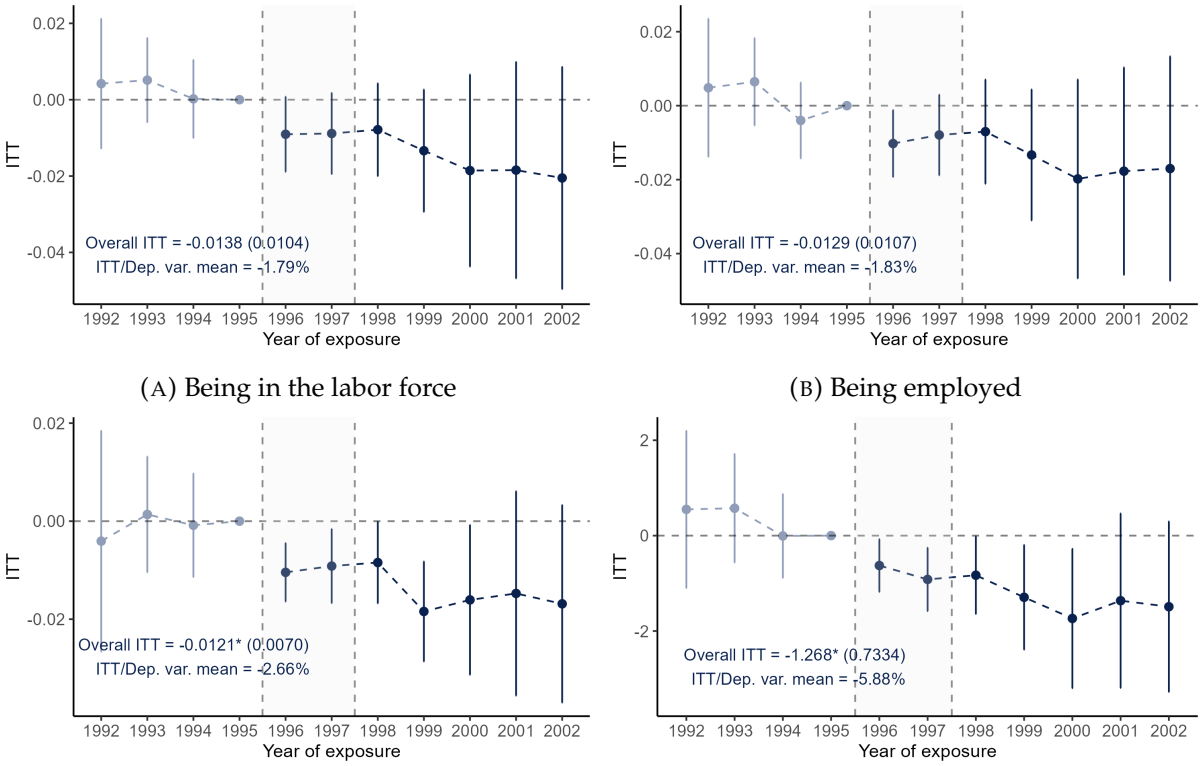
For young adults, a higher labor supply does not necessarily indicate better human capital; if they invest more in education, they allocate less time to work. Consistent with this mechanism, I find declines in labor supply that align with the rise in post-secondary school enrollment.

Figures 7a–7d plot event-study estimates for labor force participation, employment, an indicator for full-time work (1 if usual hours worked per week $\geq$ 40 hours), and annual earnings.[8] Across outcomes, coefficients for exposed cohorts are generally negative, though post-event estimates for participation and employment are somewhat imprecise. Overall, exposed young adults are 1.21 p.p. less likely to work full time (2.66% at the sample mean) and earn \$1,270 less annually (5.88% at the sample mean). Consistent with school enrollment results, coefficients are generally larger for fully exposed cohorts (1998–2002) than for partially exposed cohorts (1996–1997): partially exposed cohorts are 0.92–1.04 p.p. less likely to work full time and earn \$630–\$920 less per year; fully exposed cohorts (1998-2000) are 0.84–1.61 p.p. less likely to work full time, and earn \$830–\$1,740 less per year (Table A3).

Using the pre-fortification share of folate-deficient women (99%) and the post-fortification reduction (29%) as treatment rates, the implied TOTs indicate that in-utero fortification exposure reduces the probability of full-time work by about 1.22–4.17 p.p. (2.68%-9.17% at the sample mean) and reduces annual earnings by \$1,280–\$4,380 per year (5.93%-20.32% at the

[8] I use annual earnings in levels rather than logs because a nontrivial share of young adults report zero earnings.

sample mean).



(A) Being in the labor force

(B) Being employed

(C) Working full-time

(D) Annual earnings ($1,000)

FIGURE 7: LONG RUN EFFECTS OF FOLIC ACID FORTIFICATION ON YOUNG ADULTS' LABOR SUPPLY

Notes: The figure plots event-study estimates with 95% confidence intervals. The treated group consists of births whose first trimester ended after the March 1996 authorization of folic acid fortification and who were born in states in the top quartile of baseline CNS anomaly rates. I control for state-of-birth fixed effects; quarter-by-year-of-birth and survey-year fixed effects; and controls for gender, race, Hispanic origin, Medicaid eligibility, exposure to mental-health parity laws and welfare reforms, and the local unemployment rate. The shaded region denotes cohorts with partial exposure. The unit of observation is the individual. Regressions and dependent-variable means are weighted by the IPUMS person weight; percentile calculations are weighted by the number of births. Standard errors are clustered by state of birth.

5.4 Heterogeneity by age group

Individuals ≤ 22 are typically still making post-secondary enrollment decisions, whereas those > 22 are more attached to the labor market. Figure A1 presents estimates separately for these two groups (measured at the time of the survey): the effects are driven by the younger group—larger increases in enrollment alongside stronger declines in full-time work and hours; estimates for those > 22 are smaller and generally imprecise. This pattern is consistent with a schooling–work trade-off: exposure raises the return to education, so those still at the enrollment margin shift time toward school. It also suggests that labor-market gains for the older group may emerge later, once education investments made earlier translate into productivity and earnings.

5.5 Dose response

Figures A2a–A2c re-estimate the models using the continuous baseline CNS anomaly rate as the exposure proxy. This specification traces out a (potentially nonlinear) dose–response between exposure intensity and outcomes. The patterns are broadly consistent with the main findings: higher baseline deficiency is associated with higher post-secondary school enrollment and lower full-time work and earnings, but the estimates are noisier. That likely reflects (i) measurement noise in the continuous proxy, which pushes effects toward zero, and (ii) the fact that very small differences in baseline deficiency may not meaningfully change behavior. In contrast, the main approach (Q4 vs. the rest) delivers cleaner, more precise estimates.

5.6 Fertility selection

This section evaluates whether the long-run effects of fortification reflect fertility selection. Tables A4 and A5 show no detectable impact of fortification exposure on birth outcomes or on the distribution of most maternal characteristics. The one exception is an increase in the share of mothers aged ≤ 22. Figure A3, however, indicates that this rise follows a broader upward trend rather than a discrete change at fortification. If anything, a larger share of younger mothers would bias our estimates toward zero, given their higher baseline risk of adverse outcomes. The persistence of our main effects in the presence of this compositional shift suggests the true impact of in-utero fortification exposure is likely understated. Overall, these results argue against fertility selection as the primary driver of my findings.

6 Robustness

This section shows that the estimated effects on post-secondary school enrollment, full-time employment, and annual earnings are robust to multiple checks. A placebo randomization further indicates that the main estimates are unlikely to be driven by random noise.

(i) Alternative model specifications. I begin by testing robustness to alternative model specifications. Figures B1a–B1c compare the baseline with four variants: (i) a parsimonious model with only state-of-birth, quarter-by-year-of-birth, and survey-year fixed effects (omitting demographics and other time-varying covariates); (ii) the baseline excluding time-varying covariates; (iii) the baseline replacing separate state and survey-year effects with state-of-birth-by-survey-year fixed effects; and (iv) the baseline adding state-of-residence fixed effects. Conclusions are unchanged across specifications.

(ii) Alternative exposure thresholds. To assess sensitivity to the exposure definition, I vary

the baseline-risk cutoff used to define treated states. The baseline uses the top quartile (top 25%) of pre-eixisting CNS anomaly rates. I re-estimate the models using alternative thresholds—top 30% and top 20%—holding all other specification choices fixed. As shown in Figures B2a–B2c, the point estimates remain stable in sign and magnitude, and the confidence intervals largely overlap the baseline estimates. This pattern indicates the results are not driven by an arbitrary cutoff. Precision moves as expected with the number of treated states (slightly tighter at 30%, slightly looser at 20%), but the qualitative conclusions are unchanged.

(iii) Sharper comparision. I compare young adults born in states in the top versus bottom quartiles of baseline CNS anomaly rates. Excluding the middle quartiles sharpens the exposure contrast, reduces attenuation from measurement error, and sidesteps functional-form concerns associated with pooling medium- and low-exposure states in the baseline. Figures B3a–B3c show larger, more precisely estimated effects than in the baseline. Event-study pre-trends are flat in this extreme-groups comparison, further supporting parallel trends.

(iv) Excluding the 2020 data. I exclude 2020 to mitigate bias from the pandemic-related spike in nonresponse. ACS response rates were 94.7% (2016), 93.7% (2017), 92.0% (2018), 86.0% (2019), 71.2% (2020), 85.3% (2021), 84.4% (2022), and 84.7% (2023). The 2020 rate is substantially lower than in other years, and post-pandemic rates remain below pre-pandemic levels but are stable through 2023. Figures B4a–B4c show that excluding 2020 does not alter my conclusions.

(v) Randomization test. Finally, to test robustness to random noise, I run a randomization (placebo) exercise. I recompute ATT estimates after randomly reassigning treatment status 1,000 times while preserving its empirical distribution across regions. Figures B5a–B5c show that the main estimates lie well into in the tails of the placebo distributions, suggesting that they are unlikely to be driven by chance.

7 Magnitudes, economic significance, and policy comparison

7.1 Benchmarking magnitudes

To gauge magnitude, I benchmark the long-run education effects of folic acid fortification against two micronutrient policies (salt iodization and iron fortification of bread) and the Food Stamp Program. Because these policies differ in target populations, timing, exposure definitions, and outcomes, the exercise is illustrative rather than a strict apples-to-apples comparison.

For salt iodization, I draw on Adhvaryu et al. (2020), which estimates in-utero exposure

using a continuous proxy (baseline goiter prevalence) and report effects on years of schooling and income for adults aged 39-60. Moving exposure from the 25th to the 75th percentile increases years of schooling by 0.0712 years for women (about 0.63% of the mean; ITT) and 0.0313 years for men (about 0.27% of the mean; ITT) (Table 6 in Adhvaryu et al. (2020)). They also find income increases of 14.9% for women (ITT) and 2.88% for men (ITT) following salt iodization (Table 4 in Adhvaryu et al. (2020)).

For iron fortification, Niemesh (2015) estimate that moving from zero to a full 19 years of exposure at a one-standard-deviation difference in iron consumption implies a 0.05-year increase in schooling (ITT) among ages 22–50 (imprecisely estimated), and a 2.9% increase in total income controlling for years of schooling (Table 7 in Niemesh (2015)).

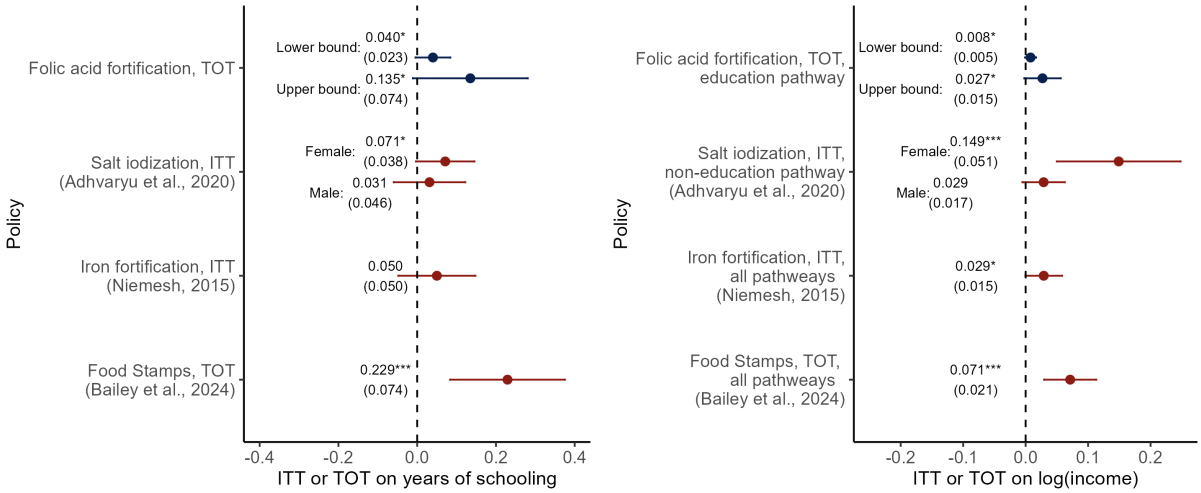
For the Food Stamp Program, Bailey et al. (2024) estimate that full exposure—from conception through age five—increases years of schooling by 0.2294 years (TOT), and raises labor income by 7.125% (TOT).

For folic acid fortification, I translate my post-secondary school enrollment effects into years of schooling using the age ≤ 22 estimate in Figure A1. I assume a 60% college completion rate and that non-completers exit after one year, so an additional enrollee contributes $(0.40 \times 1 + 0.60 \times 4) = 2.8$ years on average. Multiplying by the ITT enrollment effect of 0.014 yields an ITT schooling gain of $2.8 \times 0.014 = 0.0392$ years. To bound the treatment-on-the-treated (TOT), I scale by the first-stage exposure range from Section 5.1, i.e, pre-fortification deficiency share $= 0.99$ and post-fortification decline $= 0.29$, which implies TOT $\in \left[\frac{0.0392}{0.99}, \frac{0.0392}{0.29}\right] \approx [0.040,\ 0.135]$ years. Standard errors scale by the same factors. Starting from the enrollment standard error 0.0077, the ITT years standard error is $0.0077 \times 2.8 = 0.0216$. For the bounds, $0.0077 \times 2.8/0.99 \approx 0.0218$ and $0.0077 \times 2.8/0.29 \approx 0.0743$.

Comparing adult income effects across interventions is more challenging due to the lack of estimates on non-education channels, such as health, which likely account for a large share of long-run income gains from early-life nutrition (Bailey et al., 2024). Using a conservative 20% return to college education (Hoekstra, 2009; Zimmerman, 2014), the implied increase in later adult income operating through schooling alone is TOT $\in \left[\frac{0.0392\times0.20}{0.99}, \frac{0.0392\times0.20}{0.29}\right] \approx [0.8\%,\ 2.7\%]$. Because this excludes non-education pathways, it should be viewed as a lower bound. For example, Bailey et al. (2024) conclude that schooling accounts for roughly one-third of the income gains from early-life nutrition; health and other channels plausibly explain the remainder.

Figure 8 summarizes the comparisons. For years of schooling (Figure 8a), folic acid forti-

fication's effect is comparable to salt iodization for women and smaller than the Food Stamp Program (FSP). Yet fortification is far more cost-effective: it yields roughly 17.5–59% of FSP's schooling gains at ≈ 0.01% of FSP's annual cost (≈ $3 million (Grosse et al., 2005) vs ≈ $29 billion (Food and Nutrition Service, 2005)). For earnings (Figure 8b), the fortification effect is smaller when viewed only through the schooling channel.



(A) Years of schooling (B) Log(income)

FIGURE 8: COMPARING LONG RUN EFFECTS ON HUMAN CAPITAL ACROSS NUTRITION INTERVENTIONS

Notes: The figure plots point estimates for each nutrition intervention's effect on years of schooling and log(income), with 95% confidence intervals. The folic acid estimate converts its effect on post-secondary school enrollment into years of schooling and log(income); all other values are regression coefficients taken directly from the cited studies. Adhvaryu et al. (2020) estimate the effect of prenatal exposure to salt iodization using baseline goiter prevalence as a continuous proxy for iodine deficiency; Niemesh (2015) estimate the effect of prenatal exposure to iron fortification of bread using estimated iron consumption as a proxy for iron deficiency. For comparability, both are rescaled to reflect a shift from the 25th to the 75th percentile of the corresponding exposure measure. Bailey et al. (2024) report the effect of exposure to Food Stamp from conception to age five.

7.2 Monetizing the long run human capital gains

How much does the shift from work to school in early adulthood translate to longer-run human capital gains? I take a conservative benchmark for prime-age annual earnings of $53,996 (full-time workers aged 30-50, estimated using ACS IPUMS 2016-2023) and a 40-year working horizon with a 7% discount rate (US Office of Management and Budget, 2003). Based on my estimate for earnings via the schooling channel, i.e., 0.8% to 2.7%, The present value (PV) annuity factor is $\Phi(40, 0.07) = \frac{1-(1+0.07)^{-40}}{0.07} \approx 13.33$. Thus the PV gain per treated person from the schooling channel is $\Delta \text{PV}_{\text{HC}} \approx (0.008 \text{ to } 0.027) \times \$53,996 \times 13.33 \approx \$5,758 \text{ to } \$19,433$.

To express this at a cohort scale, consider 1,109,368 births in high-exposure states (the average number of births in the 14 high-exposure states from 1996 to 2002). Using the first-stage exposure change of 29% (Section 5.1) as a treated share, about 321,717 births are ef-

fectively exposed at the TOT margin. The cohort PV from the schooling channel is then Benefit $\approx 321,717 \times (\$5,758 \text{ to } \$19,433) \approx \$1.85$ to $\$6.25$ million. This calculation suggests that the net benefits of fortification would be underestimated by \$1.85–\$6.25 million per year if omitting its long run human capital benefits.

8 Conclusion

This paper provides the first evidence on the long-run human-capital effects of folic acid fortification of enriched grains. Exploiting the 1996 authorization of fortification and cross-state differences in baseline folate deficiency—proxied by pre-policy CNS anomaly rates—I compare adjacent birth cohorts within states to identify in-utero exposure. The results show a consistent reallocation from work to school in early adulthood: exposed individuals are more likely to enroll in post-secondary schools, less likely to work full time, and earn slightly less in the short run. These effects are concentrated among college-age cohorts. Estimates are stable across a wide set of alternative specifications, including control sets, exposure thresholds, and sample selection.

I benchmark these estimates against other nutrition interventions (salt iodization, iron fortification) and the Food Stamp Program. The years of schooling impacts are comparable to those documented for salt iodization and smaller than those for the Food Stamp Program—a much larger program. However, because folic acid fortification costs a tiny fraction of the Food Stamp Program's budget, it is much more cost-effective.

The findings of this paper align with the broader fetal-origins literature: well-timed early-life interventions can generate long run human capital gains. Given the low unit cost of fortification and its minimal reliance on behavior change, similar or larger benefits are plausible in settings where folate deficiency is more prevalent and access to supplements is limited, including many low- and middle-income countries. For policymakers operating under tight budget constraints, targeting severe micronutrient deficiencies via fortification can deliver favorable benefit–cost ratios relative to broad food subsidies.

One limitation is the absence of outcomes beyond young adulthood. Tracking human capital trajectories over the life course would provide a more complete assessment of fortification's effects. At present, few public datasets jointly include comparable human capital measures across states, birth cohorts straddling fortification, and place of birth. Building linked data—connecting birth records to administrative education, earnings, and health files or to longitudinal surveys—would help fill this gap and enable stronger tests of persistence.

Future work should follow exposed cohorts into early childhood to examine cognitive development, which can be measured by standardized test scores, grade progression, or special education placement, and into later adulthood to assess completed schooling, sustained full-time employment and earnings, occupation, and family formation. It should also quantify non-educational channels, such as health, that may mediate long-run income gains. Extending the analysis to fortification implementation in low- and middle-income countries would offer a stronger test of the external validity of this paper, given that the baseline folate deficiency and program coverage in these countries are often substantially different from the U.S. (McLean, de Benoist and Allen, 2008).

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Appendix

A Figures and tables

TABLE A1: BALANCE TEST

	$\mathbf{1}\{\text{CNSA top quartile}\}_s$		
	State-level	County Level	
	(1)	(2))	(3)
Demographic features			
Share of black (%), 1988	-0.0023	-0.0007	-0.0007
	(0.0153)	(0.0009)	(0.0036)
Share of female (%), 1988	0.0145	0.0341***	0.0341
	(0.2684)	(0.0084)	(0.0237)
Share of under 5 (%), 1988	0.2604	0.0812***	0.0812
	(0.2354)	(0.0124)	(0.0660)
Share of over 65 (%), 1988	-0.0061	-0.0281***	-0.0281
	(0.1058)	(0.0057)	(0.0317)
Birth rate (%), 1988	-0.0223	-0.0129***	-0.0129
	(0.0585)	(0.0028)	(0.0187)
Death rate (%), 1988	0.1621	0.0566***	0.0566*
	(0.2671)	(0.0114)	(0.0301)
Log population, 1988	0.0930	-0.0620***	-0.0620*
	(0.1198)	(0.0076)	(0.0340)
Economic conditions			
Transfer income p.p. (million $), 1988	-0.3067	0.0288	0.0288
	(0.5091)	(0.0321)	(0.2770)
Income p.p. (million $), 1985	107.9	36.61***	36.61
	(110.4)	(5.999)	(22.93)
Federal funds p.p. (million $), 1986	-234.2*	-7.814*	-7.814
	(135.3)	(4.400)	(9.496)
Unemployment rate (%), 1986	-0.0555	-0.0088**	-0.0088
	(0.0662)	(0.0037)	(0.0202)
Agriculture			
Value of produces sold per farm (million $), 1987	-4.560	-0.5773***	-0.5773
	(3.019)	(0.1024)	(0.4570)
Average farm size (million acres), 1987	152.6	-3.731	-3.731
	(181.6)	(9.547)	(15.16)
State FEs			Y
Observations	49	3,000	3,000
R^2	0.3209	0.0849	0.0849
Dep. var. mean	0.2941	0.2933	0.2933

Notes: Regressions are weighted by population of 1988. Data on share of black, share of female, share of under 5, share of the over 65, and population are from County Intercensal Estimates; data on birth rate, death rate, value of produces sold per farm, and average farm size are from County Databook 1988; data on transfers is from Bureau of Economic Analysis, Regional Economic Information System (REIS); unemployment data is from Bureau of Labor Statistics. $^{***}p < 0.01$, $^{**}p < 0.05$, and $^{*}p < 0.1$.

TABLE A2: REGRESSION RESULTS OF STANDARD DD ESTIMATES

	Educational outcomes		Labor outcomes			
	High school diploma or GED	post-secondary school enrollment	Being in the labor force	Being employed	Working full-time	Annual earnings ($1,000)
	(1)	(2)	(3)	(4)	(5)	(6)
1{CNSA top quartile} × Exposed cohorts	-0.0023	0.0190*	-0.0138	-0.0129	-0.0121*	-1.268*
	(0.0020)	(0.0111)	(0.0104)	(0.0107)	(0.0070)	(0.7334)
Observations	1,992,608	1,992,608	1,992,608	1,992,608	1,992,608	1,992,608
R^2	0.0123	0.1441	0.0417	0.0514	0.1168	0.1840
Dep. var. mean	0.9332	0.3369	0.7665	0.7017	0.4548	21.5536
Est./Dep. var. mean	-0.25%	5.65%	-1.79%	-1.83%	-2.66%	-5.88%

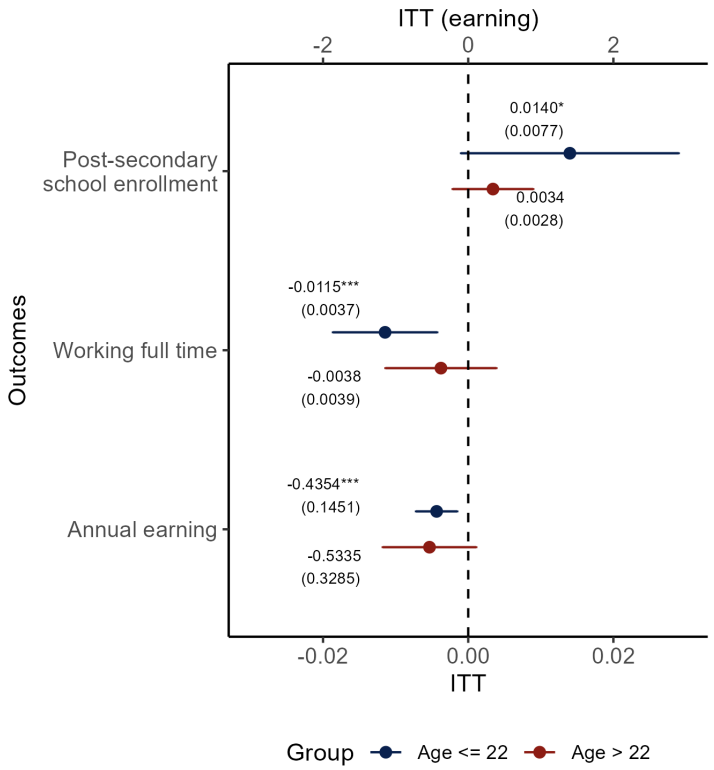
Notes: The table presents standard DD estimates and their standard errors. The treated group consists of births whose first trimester ended after the March 1996 authorization of folic acid fortification and who were born in states in the top quartile of baseline CNS anomaly rates. I control for state-of-birth fixed effects; quarter-by-year-of-birth and survey-year fixed effects; and controls for gender, race, Hispanic origin, Medicaid eligibility, exposure to mental-health parity laws and welfare reforms, and the local unemployment rate. The shaded region denotes cohorts with partial exposure. The unit of observation is the individual. Regressions and dependent-variable means are weighted by the IPUMS person weight; percentile calculations are weighted by the number of births. Standard errors are clustered by state of birth. $^{***}p < 0.01$, $^{**}p < 0.05$, and $^{*}p < 0.1$.

TABLE A3: REGRESSION RESULTS OF EVENT STUDY ESTIMATES

	Educational outcomes		Labor outcomes			
	High school diploma or GED	post-secondary school enrollment	Being in the labor force	Being employed	Working full-time	Annual earnings ($1,000)
	(1)	(2)	(3)	(4)	(5)	(6)
$\mathbf{1}\{\text{CNSA top quartile}\} \times \mathbf{1}\{t = 1992\}$	0.0073**	-0.0035	0.0048	0.0042	-0.0041	0.5502
	(0.0029)	(0.0079)	(0.0095)	(0.0087)	(0.0115)	(0.8409)
$\mathbf{1}\{\text{CNSA top quartile}\} \times \mathbf{1}\{t = 1993\}$	0.0024	-0.0078	0.0065	0.0051	0.0014	0.5743
	(0.0020)	(0.0078)	(0.0060)	(0.0056)	(0.0060)	(0.5806)
$\mathbf{1}\{\text{CNSA top quartile}\} \times \mathbf{1}\{t = 1994\}$	0.0037*	-0.0028	-0.0040	0.0002	-0.0008	-0.0065
	(0.0021)	(0.0056)	(0.0052)	(0.0052)	(0.0054)	(0.4487)
$\mathbf{1}\{\text{CNSA top quartile}\} \times \mathbf{1}\{t = 1996\}$	-0.0019	0.0102**	-0.0102**	-0.0091*	-0.0104***	-0.6266**
	(0.0016)	(0.0044)	(0.0046)	(0.0050)	(0.0030)	(0.2815)
$\mathbf{1}\{\text{CNSA top quartile}\} \times \mathbf{1}\{t = 1997\}$	0.0010	0.0077	-0.0079	-0.0089	-0.0092**	-0.9181***
	(0.0020)	(0.0046)	(0.0056)	(0.0054)	(0.0039)	(0.3389)
$\mathbf{1}\{\text{CNSA top quartile}\} \times \mathbf{1}\{t = 1998\}$	0.0041**	0.0171***	-0.0070	-0.0079	-0.0084*	-0.8299*
	(0.0019)	(0.0056)	(0.0072)	(0.0062)	(0.0042)	(0.4142)
$\mathbf{1}\{\text{CNSA top quartile}\} \times \mathbf{1}\{t = 1999\}$	-0.0010	0.0187*	-0.0133	-0.0134	-0.0184***	-1.293**
	(0.0028)	(0.0096)	(0.0090)	(0.0082)	(0.0052)	(0.5598)
$\mathbf{1}\{\text{CNSA top quartile}\} \times \mathbf{1}\{t = 2000\}$	0.0012	0.0255**	-0.0198	-0.0186	-0.0161**	-1.737**
	(0.0028)	(0.0124)	(0.0137)	(0.0128)	(0.0078)	(0.7453)
$\mathbf{1}\{\text{CNSA top quartile}\} \times \mathbf{1}\{t = 2001\}$	-0.0032	0.0209	-0.0177	-0.0185	-0.0147	-1.364
	(0.0038)	(0.0182)	(0.0143)	(0.0145)	(0.0106)	(0.9326)
$\mathbf{1}\{\text{CNSA top quartile}\} \times \mathbf{1}\{t = 2002\}$	-0.0035	0.0291	-0.0170	-0.0205	-0.0169	-1.490
	(0.0098)	(0.0249)	(0.0155)	(0.0148)	(0.0103)	(0.9116)
Observations	1,992,608	1,992,608	1,992,608	1,992,608	1,992,608	1,992,608
R^2	0.0124	0.1441	0.0514	0.0417	0.1168	0.1840
Dep. var. mean	0.9332	0.3369	0.7017	0.7665	0.4548	21.5536

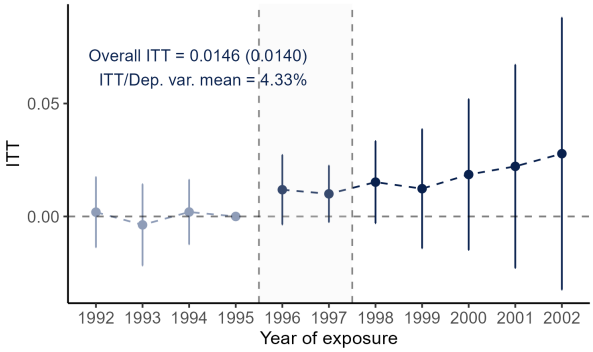
Notes: The table presents event study estimates and their standard errors. The treated group consists of births whose first trimester ended after the March 1996 authorization of folic acid fortification and who were born in states in the top quartile of baseline CNS anomaly rates. I control for state-of-birth fixed effects; quarter-by-year-of-birth and survey-year fixed effects; and controls for gender, race, Hispanic origin, Medicaid eligibility, exposure to mental-health parity laws and welfare reforms, and the local unemployment rate. The shaded region denotes cohorts with partial exposure. The unit of observation is the individual. Regressions and dependent-variable means are weighted by the IPUMS person weight; percentile calculations are weighted by the number of births. Standard errors are clustered by state of birth. $^{***}p < 0.01$, $^{**}p < 0.05$, and $^{*}p < 0.1$.

FIGURE A1: HETEROGENEITY BY AGE GROUP

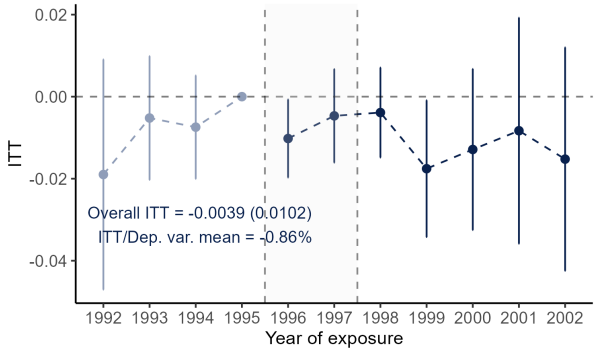


Notes: The table presents point estimates of the effects of in-utero fortification exposure by age group and their 95% confidence intervals. The treated group consists of births whose first trimester ended after the March 1996 authorization of folic acid fortification and who were born in states in the top quartile of baseline CNS anomaly rates. I control for state-of-birth fixed effects; quarter-by-year-of-birth and survey-year fixed effects; and controls for gender, race, Hispanic origin, Medicaid eligibility, exposure to mental-health parity laws and welfare reforms, and the local unemployment rate. The shaded region denotes cohorts with partial exposure. The unit of observation is the individual. Regressions and dependent-variable means are weighted by the IPUMS person weight; percentile calculations are weighted by the number of births. Standard errors are clustered by state of birth.

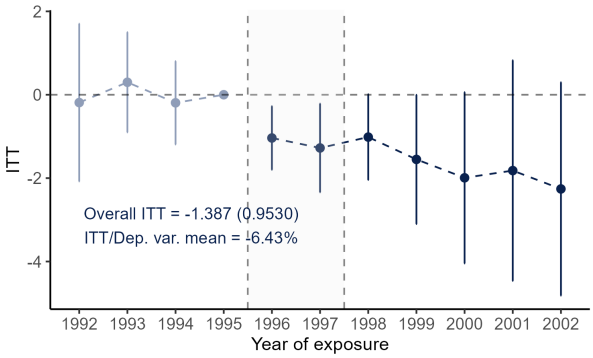
FIGURE A2: DOSE-RESPONSE RESULTS



(A) post-secondary school enrollment



(B) Working full time



(C) Annual earnings ($1,000)

Notes: The figure plots event-study estimates with 95% confidence intervals. The shaded region denotes cohorts with partial exposure. The unit of observation is the individual. Regressions and dependent-variable means are weighted by the IPUMS person weight; percentile calculations are weighted by the number of births. Standard errors are clustered by state of birth.

TABLE A4: IMPACTS OF EXPOSURE TO FOLIC ACID FORTIFICATION ON BIRTH OUTCOMES

	Birth weight (grams)	Low birth weight (1 if birth weight < 2500)	Gestation weeks	Preterm (1 if grestation weeks < 37)
	(1)	(2)	(3)	(4)
1{CNSA top quartile} $\times$ Exposed cohorts	-0.3562	0.0002	0.0129	0.0013
	(2.956)	(0.0006)	(0.0179)	(0.0014)
Observations	84,017	84,017	84,017	84,017
R^2	0.8532	0.7881	0.7817	0.7270
Dep. var. mean	3320	0.0748	38.8453	0.1141
Est./Dep. var. mean	-0.01%	0.24%	0.03%	1.12%

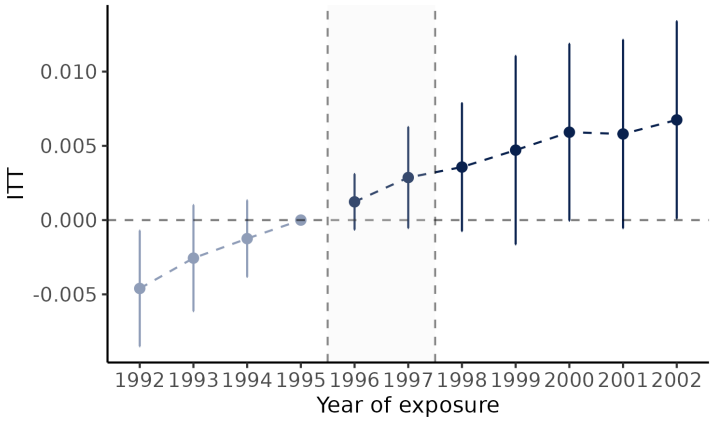
Notes: The table presents standard DD estimates and their standard errors. . I aggregate natality records to county-by-quarter-of-birth cells and merge a state-level exposure measure to each cell. The treated group consists of births whose first trimester ended after the March 1996 authorization of folic acid fortification and who were born in states in the top quartile of baseline CNS anomaly rates. I control for county-of-birth fixed effects, quarter-by-year-of-birth fixed effects, and time-varying covariates capturing Medicaid eligibility, exposure to mental-health parity laws and welfare reforms, and the local unemployment rate. Regressions and dependent-variable means are weighted by the number of birth in each cell. Standard errors are clustered by state of birth. $^{***}p < 0.01$, $^{**}p < 0.05$, and $^{*}p < 0.1$.

TABLE A5: IMPACTS OF EXPOSURE TO FOLIC ACID FORTIFICATION ON COMPOSITION OF MATERNAL CHARACTERISTICS

	Black	Age ≤ 22	$23 \leq$ Age ≤ 29	Education $<$ college	Unmarried	Indequate prenatal care
	(1)	(2)	(3)	(4)	(5)	(6)
1{CNSA top quartile} $\times$ Exposed cohorts	-0.0006	0.0057**	-0.0047	0.0021	0.0037	0.0146
	(0.0032)	(0.0024)	(0.0055)	(0.0063)	(0.0096)	(0.0126)
Observations	84,017	84,017	84,017	83,905	84,017	84,017
R^2	0.9871	0.9308	0.6128	0.9477	0.9122	0.7810
Dep. var. mean	0.1508	0.2697	0.3745	0.5486	0.3262	0.2379
Est./Dep. var. mean	-0.43%	2.10%	-1.26%	0.38%	1.13%	6.12%

Notes: The table presents standard DD estimates and their standard errors. . I aggregate natality records to county-by-quarter-of-birth cells and merge a state-level exposure measure to each cell. The treated group consists of births whose first trimester ended after the March 1996 authorization of folic acid fortification and who were born in states in the top quartile of baseline CNS anomaly rates. I control for county-of-birth fixed effects, quarter-by-year-of-birth fixed effects, and time-varying covariates capturing Medicaid eligibility, exposure to mental-health parity laws and welfare reforms, and the local unemployment rate. Regressions and dependent-variable means are weighted by the number of birth in each cell. Standard errors are clustered by state of birth. $^{***}p < 0.01$, $^{**}p < 0.05$, and $^{*}p < 0.1$.

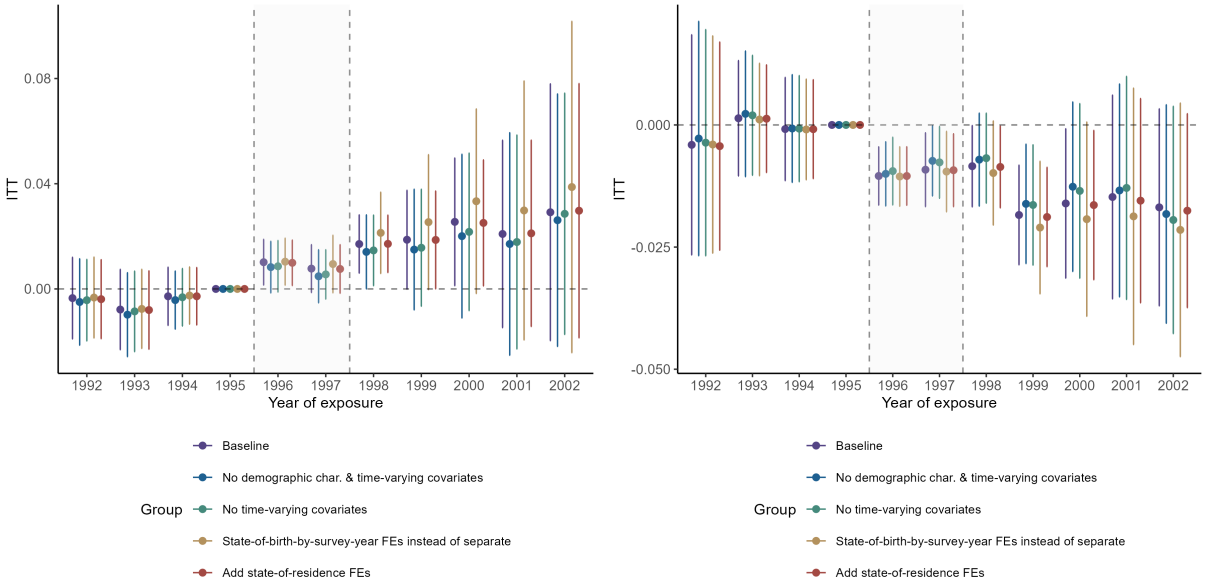
FIGURE A3: DYNAMIC EFFECTS OF EXPOSURE TO FOLIC ACID FORTIFICATION ON THE SHARE OF MOTHERS ≤ 22



Notes: The table presents standard DD estimates and their standard errors. . I aggregate natality records to county-by-quarter-of-birth cells and merge a state-level exposure measure to each cell. The treated group consists of births whose first trimester ended after the March 1996 authorization of folic acid fortification and who were born in states in the top quartile of baseline CNS anomaly rates. I control for county-of-birth fixed effects, quarter-by-year-of-birth fixed effects, and time-varying covariates capturing Medicaid eligibility, exposure to mental-health parity laws and welfare reforms, and the local unemployment rate. Regressions and dependent-variable means are weighted by the number of birth in each cell. Standard errors are clustered by state of birth. $^{***}p < 0.01$, $^{**}p < 0.05$, and $^{*}p < 0.1$. The shaded region denotes cohorts with partial exposure.

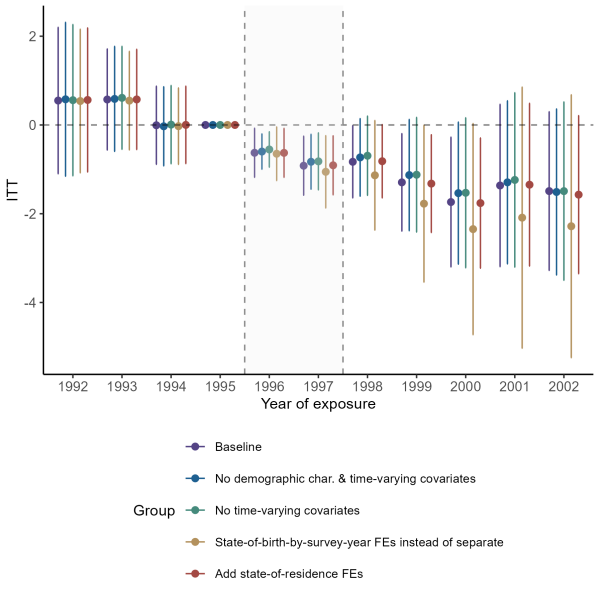
B Robustness checks and falsification tests

FIGURE B1: ROBUSTNESS TO ALTERNATIVE MODEL SPECIFICATIONS



(A) post-secondary school enrollment

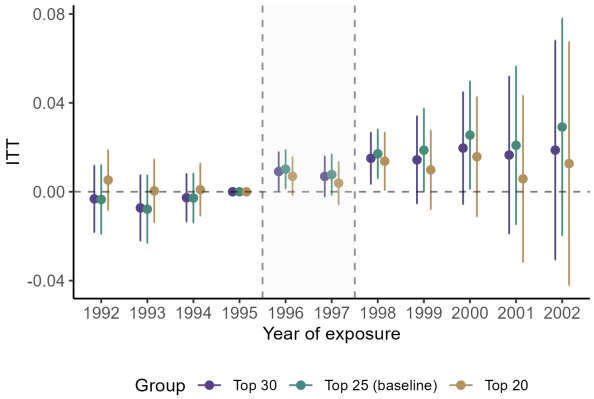
(B) Working full time



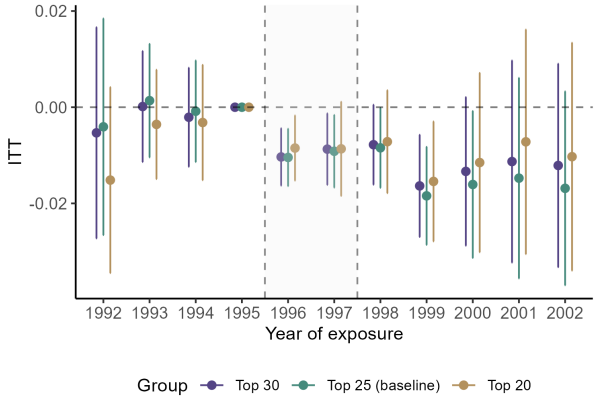
(C) Annual earning

Notes: The figure plots event-study estimates with 95% confidence intervals. The treated group consists of births whose first trimester ended after the March 1996 authorization of folic acid fortification and who were born in states in the top quartile of baseline CNS anomaly rates. The shaded region denotes cohorts with partial exposure. The unit of observation is the individual. Regressions and dependent-variable means are weighted by the IPUMS person weight; percentile calculations are weighted by the number of births. Standard errors are clustered by state of birth.

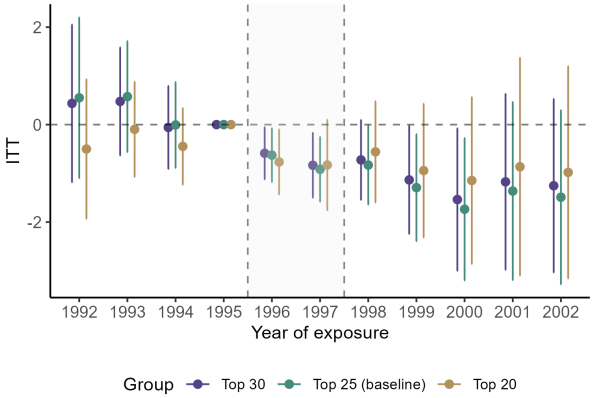
FIGURE B2: ROBUSTNESS TO ALTERNATIVE EXPOSURE THRESHOLDS



(A) post-secondary school enrollment



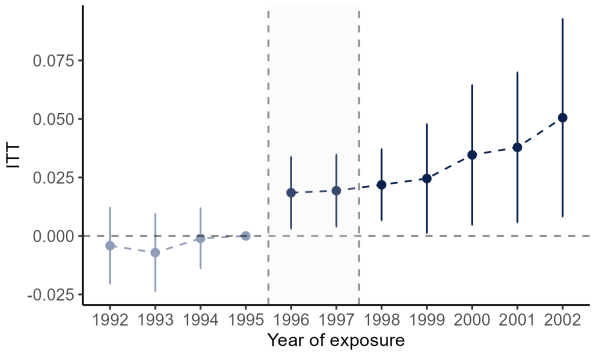
(B) Working full time



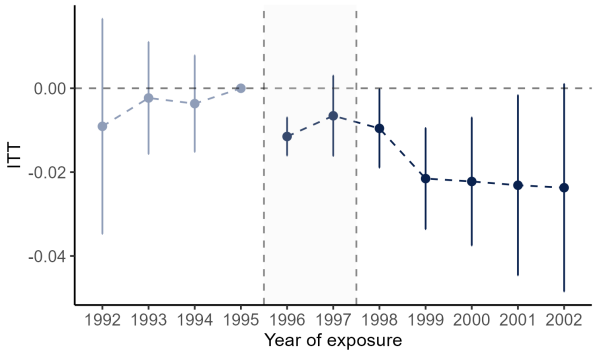
(C) Annual earning

Notes: The figure plots event-study estimates with 95% confidence intervals. The treated group consists of births whose first trimester ended after the March 1996 authorization of folic acid fortification and who were born in states in the top 30%, top quartile (25%), or top 20% of baseline CNS anomaly rates. The shaded region denotes cohorts with partial exposure. The unit of observation is the individual. Regressions and dependent-variable means are weighted by the IPUMS person weight; percentile calculations are weighted by the number of births. Standard errors are clustered by state of birth.

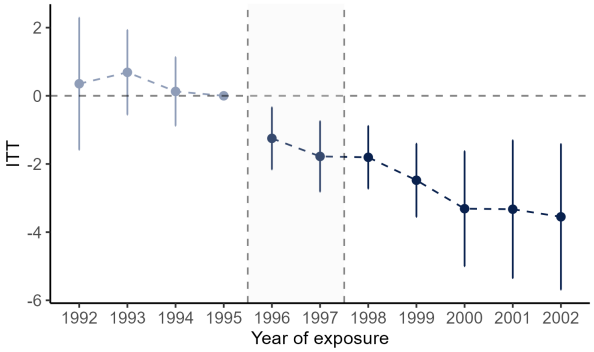
FIGURE B3: STATES WITH Q4 VS. Q1 EXPOSURE



(A) post-secondary school enrollment



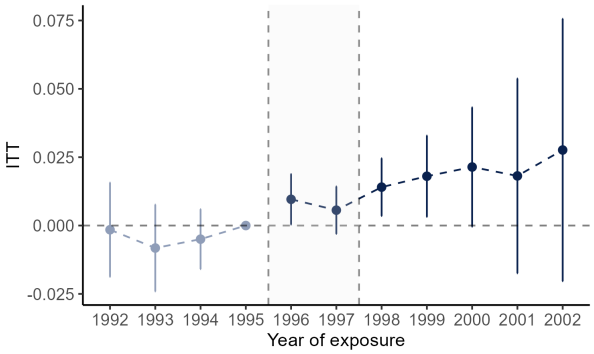
(B) Working full time



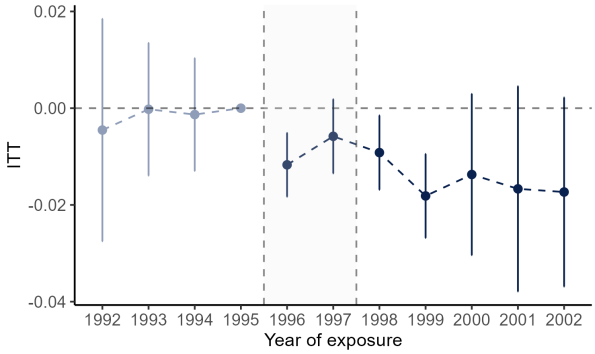
(C) Annual earning

Notes: The figure plots event-study estimates with 95% confidence intervals. The treated group consists of births whose first trimester ended after the March 1996 authorization of folic acid fortification and who were born in states in the top quartile of baseline CNS anomaly rates. The shaded region denotes cohorts with partial exposure. The unit of observation is the individual. Regressions and dependent-variable means are weighted by the IPUMS person weight; percentile calculations are weighted by the number of births. Standard errors are clustered by state of birth.

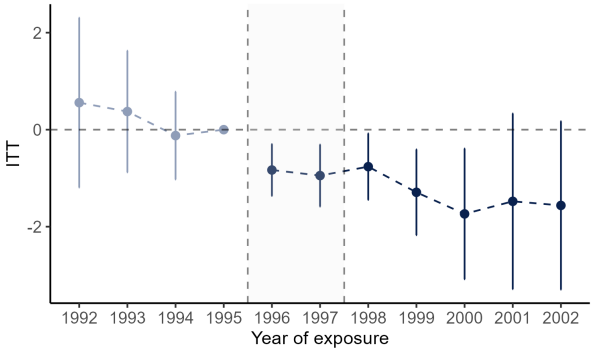
FIGURE B4: DROP ACS IPUMS 2020



(A) post-secondary school enrollment



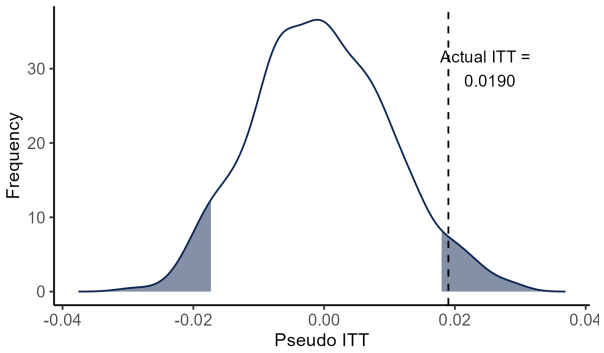
(B) Working full time



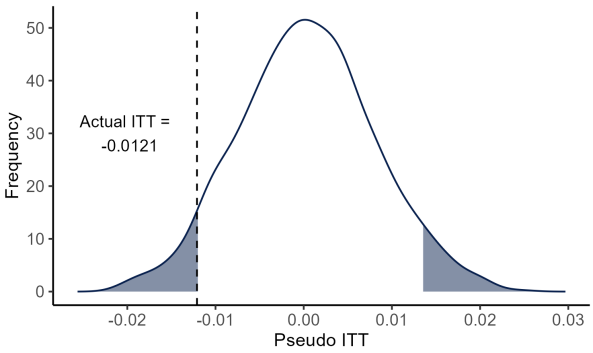
(C) Annual earning

Notes: The figure plots event-study estimates with 95% confidence intervals. The treated group consists of births whose first trimester ended after the March 1996 authorization of folic acid fortification and who were born in states in the top quartile of baseline CNS anomaly rates. The shaded region denotes cohorts with partial exposure. The unit of observation is the individual. Regressions and dependent-variable means are weighted by the IPUMS person weight; percentile calculations are weighted by the number of births. Standard errors are clustered by state of birth.

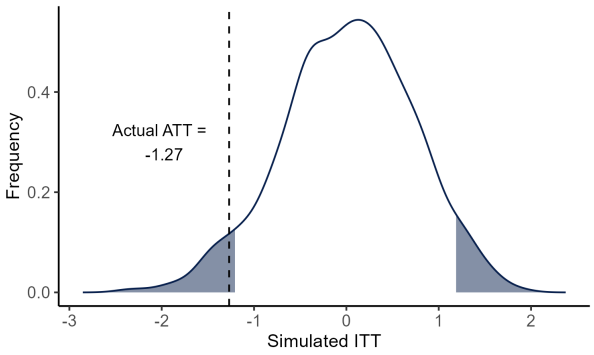
FIGURE B5: RANDOMIZATION TEST



(A) post-secondary school enrollment



(B) Working full time



(C) Annual earning

Notes: The figure plots estimates of overall effects. The treated group consists of births whose first trimester ended after the March 1996 authorization of folic acid fortification and who were born in states in the top quartile of baseline CNS anomaly rates. The unit of observation is the individual. Regressions are weighted by the IPUMS person weight; percentile calculations are weighted by the number of births. shaded areas represent $\leq$ 5th and $\geq$ 95th percentiles of our simulated null distribution.