# Long Run Effects of Fortifying Grain Products with Folic Acid*

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**Abstract**

In March 1996, the Food and Drug Administration (FDA) mandated the fortification of grain products with folic acid to prevent deficiency in folate, a critical micronutrient for fetal neurodevelopment. This paper examines the long run effects of this mandate on human capital outcomes. By comparing cohorts exposed and unexposed to the fortification across regions with different baseline folate deficiency levels, I find that in-utero exposure to the fortification raises the likelihood of post-secondary enrollment for young adults by 0.69 to 1.17 percentage points. It also reduces the likelihood of working full-time among 19-to-22-year-olds by 0.79 to 1.54 percentage points but has no impact on the labor supply of individuals over 22. Finally, my back-of-the-envelope calculation indicates that folic acid fortification provides long-term human capital benefits comparable to those of food stamps but at a significantly lower cost. (JEL I18, J22, J24, N32, N52, Q18)

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# 1 Introduction

Micronutrient deficiencies impair the physical and cognitive development of millions of children each year. However, evidence on their long-run effects has largely been limited to iodine and iron. This paper contributes new evidence by examining the long-run impact of the FDA's folic acid fortification mandate on human capital.

Food fortification is a cost-effective strategy to enhance micronutrient access, with significant implications for human capital. 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 deficiencies 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—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 pre-existing deficiencies. 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 pre-existing 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 link this variation, along with the timing of fortification, to birth outcomes from Vital Statistics Data and human capital outcomes from the American Community Surveys (ACS), employing a difference-in-difference framework to study long-term effects of in-utero exposure to folic acid fortification on educational outcomes.

To validate my research design, I compile multiple pieces of evidence showing: (1) folate content increased in a wide range of foods post-fortification; (2) dietary folate intake and blood

folate concentrations rose following fortification; (3) the prevalence of birth defects associated with folate deficiency declined after fortification, driven primarily by the decline in regions with higher pre-existing rates; and (4) the prevalence rate of folate-deficiency-associated birth defects are negatively correlated with key biomarkers for folate deficiency, supporting the use of prevalence rates folate-deficiency-associated birth defect as a proxy for spatial variation in pre-existing folate deficiency. I find that folic acid fortification has had a significant long-term impact on human capital investment. In-utero exposure to folic acid fortification increases the probability of post-secondary enrollment by 0.69 to 1.17 percentage points for young adults. This effect is driven by both the higher college enrollment among 19-to-22-year-olds and the increased graduate or professional school enrollment among those over 22. As a result, the likelihood of working full-time decreases by 0.79 to 1.54 percentage points for 19-to-22-year-olds. The labor supply remains unchanged for those over 22, likely because the effect on graduate/professional school enrollment is smaller and such programs are more likely to be part-time. Finally, by translating the increase in post-secondary enrollment into years of schooling, I find that folic acid fortification provides long-term human capital benefits comparable to those of food stamps but at a significantly lower cost.

This paper contributes to three strands of literature. First, it adds to the limited social science research on the socioeconomic effects of food fortification. While most existing studies focus on the long-term benefits of salt iodization on cognitive, health, and socioeconomic outcomes, such as improved cognitive ability and increased income (Feyrer, Politi and Weil, 2017; Serena, 2019; Adhvaryu et al., 2020; Huang, Liu and Zhou, 2020; Deng and Lindeboom, 2022*a*; Tafesse, 2022), little is known about the human capital effects of folic acid fortification. Unlike iodine and iron deficiencies (Niemesh, 2015), which primarily affect thyroid function and blood oxygen transport, folate deficiency directly affects nervous system development, potentially leading to more severe health consequences. Folic acid fortification, therefore, may have a greater influence on cognitive development and subsequent economic outcomes, such as educational attainment and income. Additionally, since folic acid fortification is less widely implemented than salt iodization and iron supplementation, particularly in developing countries[1], causal evidence from the U.S. can inform and motivate broader adoption of this policy. This paper also contributes to the scientific literature on the effects of folic acid fortification. While existing research primarily focuses on the short-term health benefits of folic acid supplementation (e.g., Wald et al., 2001; Quinlivan et al., 2002; Kancherla et al., 2022, etc.) or cost-benefit analyses of fortification (e.g., Grosse et al., 2005; Bentley et al., 2009; Llanos et al.,

[1]See the webpage of Global Fortification Data Exchange, https://fortificationdata.org/nutrient-intake-for-all-food-by-country/, for reference.

2007, etc.), there is a lack of causal evidence on its human capital effects. This study extends the scope of current research by examining the long-term educational outcomes associated with folic acid fortification.

Second, this paper contributes to the fetal origins literature by exploring the long-term effects of early-life nutritional access. Existing research demonstrates that early-life nutritional conditions have lasting effects, with negative shocks like 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) leading to poorer adult health and labor outcomes; while 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 (Hoynes, Page and Stevens, 2011; Rossin-Slater, 2013; Hoynes, Schanzenbach and Almond, 2016; Bailey et al., 2024), enhance cognitive development and socioeconomic outcomes. This study extends this body of work by examining the effects of folic acid fortification during early fetal development on school enrollment of young adults.

Finally, this paper sparks a discussion on the efficiency of current nutrition interventions. The U.S. federal government allocates hundreds of billions of dollars annually to improve food and nutrition security. A significant portion of this budget is directed toward food and nutrition assistance programs, which aim to subsidize nutritious foods for households in need. However, the challenge of altering consumer behavior raises concerns about the effectiveness of these programs (Smith and Gregory, 2023). Research by Allcott et al. (2019) reveals that only 10% of nutritional inequality can be attributed to access to healthier foods, with the remaining 90% driven by differences in demand. In contrast, reformulation—altering the nutrient composition of foods without requiring changes in consumer behavior—presents a potentially more effective strategy for improving the nutritional status of low-income households, particularly their intake of specific micronutrients. This paper studies a specific instance of reformulation: the folic acid fortification of grain products in the U.S. in the late 1990s and its impacts on health and human capital.

The paper is organized as follows: Section 2 provides the policy background; Section 3 proposes a conceptual framework linking fortification and long run educational outcomes; Section 4 describes the data and sample; Section 5 outlines the research design and discusses identifying assumptions; Sections 6 presents both descriptive and causal results; Section 7 an-

alyzes robustness and sensitivity of results; Section 8 discusses magnitude of estimates and policy cost-effectiveness; and, finally, Section 9 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[2] are typically fatal, with most affected infants dying before or shortly after birth. Mild NTDs, like spina bifida[3] allow survival into adulthood but carry a high risk of lifelong physical and mental disabilities (Yi et al., 2011). 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 anomalies, 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). Moreover, timely medical intervention is often hindered, as ultrasound screenings typically occur in the second trimester, when congenital anomalies become more detectable (Blumenfeld, Siegler and Bronshtein, 1993), and many pregnant women in the U.S. do not receive adequate prenatal care.

### 2.2 Sources of folate

Folate can be naturally obtained in foods such as beef liver, dark green leafy vegetables, beans, peas, nuts, fruits, and fruit juices. The poor stability of food folate under typical cooking conditions can substantially reduce the eventual amount of folate digested, which makes food folate less attractive as a means to enhance the folate status of pregnant women (McNulty and Pentieva, 2004). Despite proper cooking methods, it is still difficult to achieve the recommended level of folate intake for pregnant women from regular diets (Czeizel, 2000). According to the third National Health and Nutrition Examination Survey (NHANES III), mean daily folate consumption is 233.68 $\mu$g for women aged 15 to 49 from 1988 to 1994, which is far below 400 $\mu$g, the recommended folate intake for pregnant women from the United States Public Health

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

[3]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.

Services.

Besides food folate, people can also get folate from nutrition supplements such as over-the-counter folic acid tablets and multivitamin pills in pharmacies. Folic acid is synthetic form of folate. Folic acid supplements are often prescribed to pregnant women during their prenatal visits (Ray, Singh and Burrows, 2004). One problem with folic acid supplementation is poor awareness of and adherence to the supplementation recommendation (Toivonen et al., 2018). According to CDC guidance[4], folic acid supplementation should start at least one month prior to conception. However, approximately 50% of pregnancies are unintended in the U.S. (Finer and Zolna, 2016). From 1995 to 1998, only about 30% of women in the U.S. reported taking vitamin supplements containing folic acid every day and less than 10% of them knew folic acid should be taken before pregnancy (Petrini, Damus and Johnston, 1999). Moreover, low-income women may have more difficulties accessing and affording folate-rich foods and folic acid supplements (Czeizel, 2000). Therefore, policymakers need to come up with a more affordable, ideally passive means to ensure folic acid adequacy for pregnant women.

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

The U.S. has a long history of food fortification to improve public health, beginning with salt iodization in the 1920s, followed by vitamin D fortification of milk in the 1930s, and the enrichment of flour and bread with B vitamins and iron in the 1930s and 1940s. The most recent effort, folic acid fortification of grain products, began in the 1990s. The first wave of grain product fortification started in the 1940s after the identification of specific nutrient deficiency disorders in the U.S. In the early 1940s, the FDA established the first standard of identity for enriched flour, requiring the addition of iron and B vitamins, including niacin, thiamin, and riboflavin. By the 1950s, these standards extended to other cereal grain products, such as 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. It is widely regarded as one of the most successful public health initiatives in recent decades (Berry, Mulinare and Hamner, 2010).

Like earlier fortification efforts, this change was driven by accumulating scientific evidence on folic acid's potential to prevent neural tube defects (NTDs). In October 1990, as part of the Nutrition Labeling and Education Act, Congress directed the FDA to examine the link between folic acid and NTDs and to develop a plan for its addition to food products (Wright,

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

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, the FDA amended the standard of identity on March 5, 1996, to require the addition of 140 µg/100g of folic acid to enriched grain products by January 1, 1998 (Food and Drug Administration, 1996). However, fortification was largely completed by mid-1997 (Jacques et al., 1999), so the effective event date is considered to be March 5, 1996. For example, some chips contain folic acid because they include enriched wheat flour (Figure 1). Prior to the mandate, voluntary folic acid fortification was prohibited in standardized foods[5] and discouraged in other foods as part of a broader policy to avoid overfortification and nutrient imbalances in the population (Food and Drug Administration, 1996, 2015).


FIGURE 1: CHIPS WITH ENRICHED WHEAT FLOUR AS AN INGREDIENT

## 3 Conceptual Framework

The potential link between in-utero exposure to folic acid fortification and long-run human capital outcomes can be understood through a simple, standard labor supply model. Consider a young adult of age $g$ who allocates their time between leisure, work, and school within a time frame $T_g$ they can envision when making this decision. The individual derives utility from both leisure and consumption, while schooling increases future wages by building human capital. Their objective is to maximize utility by choosing how to allocate time across these activities. Let $L_g$, $W_g$, and $S_g$ represent the number of hours allocated to leisure, work, and school, respectively. The following constraint must hold: $T_g = L_g + W_g + S_g$. The young adult's utility function depends on leisure and consumption. Let $C$ be the individual's consumption, which is determined by her earnings from work. Assume the utility function i as simple as $U(L_g, C_g) = \eta_g \log(L_g) + (1 - \eta_g) \log(C_g)$ ,where $\eta_g \in (0, 1)$ represents the relative

[5]Standardized foods have a standard of identifty, such as enriched grain products.

importance of leisure versus consumption in the individual's preferences. Without loss of generality, I drop the subscript $g$, as this is what can be observed from the pooled cross-sectional data in later empirical analyses. I further assume that all of a young adult's income comes from working, and consumption ($C$) is equal to the wage rate ($w$) multiplied by the number of hours worked ($W$): $C = w \cdot W$. The individual's future wage depends on the time spent in school, which enhances their human capital. The wage is given by $w = w_0 + \theta S$ where $w_0$ is the base wage without any schooling, and $\theta$ is the return to education. The total time available is divided between leisure, work, and school: $T = L + W + S$. The individual's objective is to maximize utility by choosing $L$, $W$, and $S$ subject to the time constraint and the wage equation. Solve the utility maximization problem (see details in Section A) we can get optimal time allocated to school is:

$$S = \frac{(1-\eta)T}{1+2(1-\eta)} - \frac{2(1-\eta)w_0}{\theta\,(1+2(1-))}.$$

Let $\tilde{\eta} = \frac{(1-\eta)}{1+2(1-\eta)}$, we can further simply the expression of $S$ to be:

$$S = \tilde{\eta}\left(T - \frac{2w_0}{\theta}\right).$$

The optimal allocation of time to schooling increases with the return to education ($\theta$) and decreases with the base wage ($w_0$).

In-utero exposure to folic acid fortification is likely to increase young adults' time investment in education by improving cognitive ability and, consequently, increasing the return to education ($\theta$). Existing scientific literature provides evidence that folic acid supplementation improves cognitive ability. For example, animal studies have shown that maternal folate deficiency is associated with short-term memory impairment and anxiety-related behaviors in offspring. Similarly, human studies find that maternal folate deficiency correlates with poorer cognitive outcomes in children, including delayed motor development, lower verbal and visuospatial skills, reduced test scores, and an increased risk of neurodevelopmental disorders such as autism and ADHD (see Irvine et al. (2022) for a literature review).

Cognitive ability, on the other hand, may contribute to a higher return on education. Bowles, Gintis and Osborne (2001) find that introducing a measure of cognitive performance reduces the coefficients for years of education by an average of 18%. However, these measures cannot fully distinguish between cognitive ability prior to education and that acquired through education. Murnane, Willett and Levy (1995) finds that basic cognitive skills, as mea-

sured by high school test scores, had a significant impact on the wages of 24-year-olds, with the increase in the return to cognitive skills explaining the entire wage premium associated with post-secondary education for women. Heckman, Stixrud and Urzua (2006) use a structural approach to show that individuals with stronger latent cognitive skills tend to achieve higher earnings from additional schooling, as they are better able to translate educational achievements into higher productivity and wages, thus increasing their returns to education.

# 4 Data

The data for this analysis come from multiple sources. The treated group is defined as individuals born in states with high pre-fortification prevalence rate of birth defects tied to folate deficiency, calculated using restricted-access Vital Statistics Natality Data. I then link spatial variations in pre-existing prevalence of birth defects tied to folate deficiency to outcome variables from Vital Statistics Natality Data and the American Community Survey[6], based on state and time of birth.

## 4.1 Vital Statistics Natality Data

Vital Statistics Natality Data, derived from birth certificates, includes comprehensive information on all live births in the U.S. This data covers birth outcomes such as the month and year of birth, county of birth, birth weight, gestational age, and congenital anomalies, as well as maternal characteristics including age, race, Hispanic origin, educational attainment, and prenatal care adequacy (National Center for Health Statistics, 2003).

Natality data serves several purposes. First, I calculate pre-existing prevalence rates of birth defects tied to folate deficiency by dividing the number of CNS anomalies by the total number of births between January 1989 and June 1993. 1989 was selected as the starting point because it marks the first year states were required to report congenital anomalies on birth certificates, though five states (Louisiana, Nebraska, Oklahoma, New York, and New Mexico) began reporting these anomalies at later times than other states. To maximize states included, I include data up to mid-1993 to construct pre-existing CNS anomaly rates. Cohorts born after this period are used for cross-cohort comparisons to ensure at least four pre-periods for event study analysis. Birth certificate report five categories of CNS anomalies: spina bifida, anencephaly. hydrocephaly, microcephaly, and other CNS anomalies. Folate deficiency is the major

[6]Other surveys, such as the Early Childhood Longitudinal Study, Kindergarten Class of 2011 (ECLS-K 2011) and the National Longitudinal Surveys of Youth 1997 (NLSY97), are not used due to limitations in coverage and relevance. For example, ECLS-K 2011 includes only cohorts born after 2000, thus excluding those exposed to folic acid fortification. NLSY97 participants, who were ages 12 to 15 as of December 31, 1996, were not exposed to folic acid fortification.

cause of NTDs including spina bifda and anencephaly and can cause hydrocephaly directly or indirectly. Other NTDs are not reported separately on birth certificates. While the link between folate deficiency and microcephaly is less clear, microcephaly represents only a small proportion of total CNS anomalies. Therefore, in my primary analysis, I use CNS anomalies as a proxy for birth defects associated with folate deficiency. The resulting pre-existing prevalence rates of CNS anomalies (henceforth CNS anomaly rates) exhibit significant spatial variation (Figure 2).

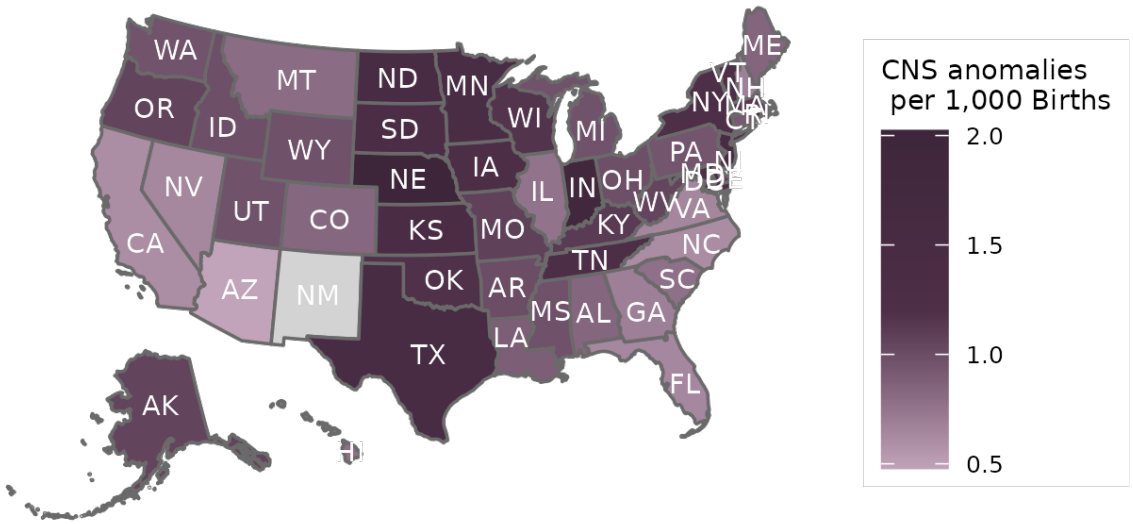



FIGURE 2: PRE-EXISTING CNS ANOMALY RATES BY STATE

Notes: Pre-existing CNS anomaly rates are aggregated from the birth-level Natality Data (restricted-use version) to state-of-birth level. The chosen period is from January 1989 to June 1993.

Second, I determine exposure timing based on weeks of gestation recorded on birth certificates. An infant is classified as exposed if their first trimester ends after March 1996, as neural tube closure occurs during this period and folic acid helps prevent CNS anomalies. I aggregate birth-level exposure dummy by quarter-and-year. As shown in Figure 3, the share of infants exposed to folic acid fortification during their first trimester increased sharply for births from the fourth quarter of 1996 onward. Therefore, individuals born in and after this period are defined as the exposed group. This pre-post variation, combined with the spatial variation in pre-existing CNS anomaly rates, forms the key variation driving my empirical strategy.

Finally, I assess the effects of folic acid fortification on maternal characteristics and birth outcomes from July 1993 to December 2002 to evaluate any compositional changes. I analyze whether fortification affects the proportion of disadvantaged mothers—those under 22 years

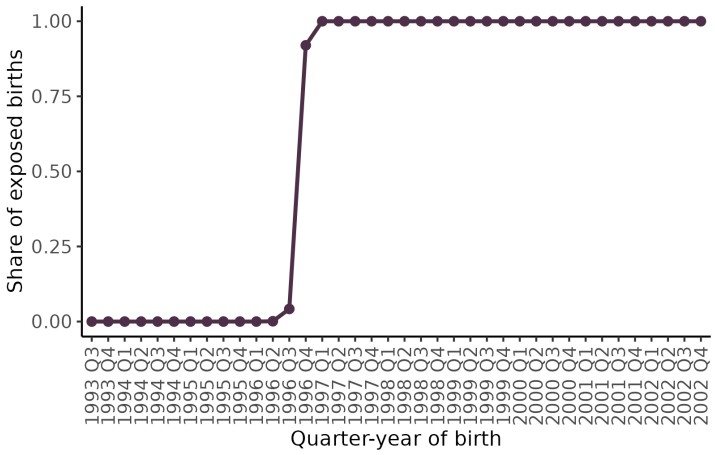



FIGURE 3: SHARE OF EXPOSED BIRTHS BY QUARTER-AND-YEAR-OF-BIRTH

Notes: An infant is considered exposed if her first trimester ends after March 1996, the month when folic acid fortification is authorized. Exposure is measure as birth level, and then aggregated to county-and-quarter-year cell.

old, without a college degree, lacking adequate prenatal care, or non-white or Hispanic.

### 4.2 American Community Surveys

I link state-level pre-existing CNS anomaly rates to young adult outcomes from the American Community Survey Public-Use Microdata Sample (ACS PUMS) for the periods 2017–2019 and 2021–2022, excluding ACS 2020 due to its high nonresponse rate caused by the pandemic[7]. I focus on young adults as they represent the oldest cohorts exposed to folic acid fortification since earliest cohorts exposed to fortification were born in the fourth quarter of 1996 and were in their 20s during the ACS periods used in this study. The final sample includes young adults of 19 to 29 years old.

The most relevant human capital outcomes for this group are high school completion and post-secondary education enrollment. Specifically, I examine the probability of young adults earning high school diploma or equivalent credential and their likelihood of enrolling in post-secondary education (including both college and graduate/professional schools). To ensure that post-secondary education enrollment is a meaningful measure of human capital investment, I disaggregate the data by age group: college enrollment for those aged 19–22, and graduate or professional school enrollment for those over 22.

In addition to the aforementioned education outcomes, I also examine the labor supply of young adults. A sign of greater human capital investment in young adulthood is reduced labor supply, as young adults are more likely to invest their time in education. The labor outcomes

[7]Response rates of ACS are 93.7% in 2017, 92% in 2018, 86% in 2019, 71.2% in 2020, 85.3% in 2021, and 84.4% in 2022.

of interest include usual hours worked per week, the probability of working full-time, and earnings, capturing both the quantity and price effects. Full-time workers are defined as those who typically work more than 40 hours per week.

The fact that the treatment variable is only available at the state level raises concerns about statistical power. To address this, in addition to the full sample, I also report results for nonmovers—those who reside in their state of birth at the time of the survey—since they are more likely to come from disadvantaged families that are less able to afford out-of-state tuition if they pursue post-secondary education. Children from disadvantaged families are also more likely to benefit from folic acid fortification, as their mothers may have been less able to afford nutrient-balanced diets before pregnancy. Table B1 shows that nonmovers are more likely to be people of color and Hispanic, and were more commonly born in the Midwest and South. Additionally, nonmovers are more comparable across cohorts, as they experience similar postnatal environments and are less likely to have extensive migration experiences. Nonmovers make up approximately 70% of all young adults in my sample, highlighting their substantial economic significance. Figure B1 demonstrates that in-utero exposure to folic acid fortification does not affect the likelihood of being a nonmover, suggesting that the results for nonmovers are unlikely to be influenced by compositional changes due to fortification.

## 5 Methods

An ideal empirical strategy would involve a randomized trial where pregnant women are randomly assigned to receive folic acid supplements, and their children are tracked into adulthood to compare outcomes. However, this approach is not feasible at scale. Instead, I utilize the timing of the 1996 folic acid fortification of grain products and spatial variation in pre-existing CNS anomaly rates to assess the effect of folic acid supplementation on human capital.

My approach is similar to studies examining the benefits of disease interventions based on pre-existing regional disease prevalence. For example, pre-existing hookworm infection rates have been used to measure the effect of hookworm eradication campaigns (Bleakley, 2007), malaria rates to evaluate malaria eradication efforts (Bleakley, 2010; Kuecken, Thuilliez and Valfort, 2021), measles rates for measles vaccination (Atwood, 2022), pneumonia rates for Sulfa antibiotic introduction (Lazuka, 2020), and goiter rates for salt iodization (Feyrer, Politi and Weil, 2017; Adhvaryu et al., 2020).

### 5.1 Empirical models

I employ a cohort difference-in-difference framework with continuous treatment to assess the effects of folic acid fortification. My preferred model specification includes multiple fixed effects and individual-level controls for a more precise estimation. The empirical model is:

$$Y_{ist} = \beta \text{CNS anomaly rate}_s \times Post_{it} + \mu_s + \lambda_t + C_{ist} + \varepsilon_{ist}, \quad (1)$$

where $Y_{ist}$ represents the outcome for individual $i$ who born in state $s$ and quarter-and-year $t$, CNS anomaly rate$_s$ is a measure of pre-existing CNS anomaly rates at state-of-birth level, the dummy variable $Post_{it}$ indicates whether the cohort is exposed, $\mu_s$ is state-of-birth fixed effects to account for cohort-invariant unobserved heterogeneity, $\lambda_t$ is quarter-and-year-of-birth fixed effects to control for cohort-specific shocks, $C_{ist}$ is a set of control variables, and $\varepsilon_{ist}$ is an error term. In $C_{ist}$ I control for (i) individual characteristics including gender, race dummies, and Hispanic origin, (ii) confounding policies including Medicaid eligibility of pregnant women estimated by Hoynes and Luttmer (2011) to control for expansion of Medicaid and State Children's Health Insurance Programs from 1997, exposure to mental health parity laws, dummies for first major waiver of Aid to Families with Dependent Children (AFDC) program and for the actual implementation of Temporary Assistance to Needy Families (TANF) block grant to control for confounding effects of welfare reform in 1996, (iii) a Bartik-style measure of state-by-year unemployment rate from Ganong and Liebman (2018) to control for local economic conditions at birth, (vi) survey-year fixed effects to control for unobservables specific to the year of interview, and, following Hoynes, Page and Stevens (2011) and Hoynes, Schanzenbach and Almond (2016), state-level baseline characteristics interacted with linear time trend (quarter-and-year-of-birth) to control for possible differences in trends across states.

### 5.2 Identifying assumptions

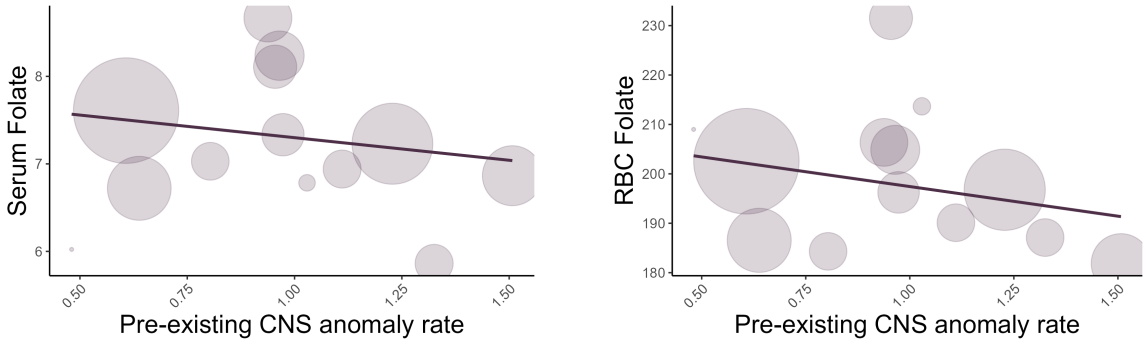
The validity of this research design hinges on several assumptions. First, pre-existing CNS anomaly rates should be uncorrelated with other factors influencing the outcomes. To partially test this, I regress baseline CNS anomaly rates on pre-intervention characteristics aggregated at the state level or finer commuting-zone-by-state level. The results, shown in Table 1, indicate that only 3 out of 13 characteristics are statistically significant, with 60%-70% of the variation remaining unexplained, suggesting substantial quasi-randomness in the variation. Nonetheless, to control for possible differences in cross-sectional trends that might be spuriously correlated with fortification exposure, I include all the pre-invention characteristics interacted with

linear time trends in my main regressions. To further ease this concern, I present event study results for all of my main outcomes to see whether different regions are trending differently prior to fortification. For event study design, I follow the following empirical model:

$$Y_{ist} = \sum_{\gamma=1992, \gamma \neq 1995}^{2002} \beta_\gamma \text{CNS anomaly rate}_s \times \mathbf{1}\{t \in \gamma\} + \mu_s + \lambda_t + C_{ist} + \varepsilon_{ist}, \tag{2}$$

I define year of effective exposure $\gamma$ based on the timing of the first trimester, aligning the year of effective exposure with the year of birth if the birth occurred in the fourth quarter, or the prior year otherwise. All other symbols remain consistent with those in Equation 1.

Second, pre-existing CNS anomaly rates should reflect the levels of local maternal folate deficiency. While large-scale data on maternal folate deficiency is not available, I find a strong negative correlation between pre-existing CNS anomaly rates and two biomarkers of folate deficiency from NHANES III (Figures 4a-4b). Serum folate concentration serves as a biomarker for acute deficiency, while RBC folate concentration indicates chronic deficiency. Additionally, regions with higher pre-existing CNS anomaly rates experienced greater declines in these rates post-fortification (Figure 8b), supporting the validity of this assumption.




(A) Serum folate

(B) RBC folate

FIGURE 4: CORRELATION BETWEEN PRE-EXISTING CNS ANOMALY RATE AND BIOMARKERS OF FOLATE DEFICIENCY AT STATE LEVEL

Notes: Data source is public-use NHANES III (1988-1994). Geographical identifiers that are not suppressed include 35 counties from 13 states. y-coordinate of bubble centroid is average serum or RBC folate concentration at state level. Bubble size represents the sum of individual sample weight from that state. Fitted line is predicted values from the regression of individual serum or RBC folate concentration on state-level CNS anomaly rate as in Table (Columns (5) and (6), 2).

For CNS anomaly rate$_s$ in Equation 1, I report results using both continuous CNS anomaly rates and binary indicators for residents in high-exposure regions. Continuous CNS anomaly rates allow for the retention of more variation, but there are two key concerns: (i) The parallel trend assumption is stronger in models with continuous exposure, and event study results cannot distinguish between standard and stronger parallel trends in event studies (Callaway,

TABLE 1: CORRELATION BETWEEN PRE-EXISTING CNS ANOMALY RATE AND BASELINE CHARACTERISTICS

| | Pre-existing CNS anomaly rates (per 1,000 births) | | | |
|---|---|---|---|---|
| | CZ-by-state mean (SD) | State level regression | CZ-by-state Level regression | |
| | (1) | (2) | (3) | (4) |
| *Demographic features* | | | | |
| Share of black (%), 1988 | 7.42 | -0.0214** | -0.0150*** | -0.0098** |
| | (12.10) | (0.0095) | (0.0028) | (0.0041) |
| Share of female (%), 1988 | 50.95 | 0.1241 | 0.0327 | -0.0538 |
| | (1.52) | (0.1231) | (0.0423) | (0.0413) |
| Share of under 5 (%), 1988 | 7.47 | 0.2364* | 0.1435** | 0.0853 |
| | (1.28) | (0.1326) | (0.0550) | (0.1023) |
| Share of over 65 (%), 1988 | 14.31 | -0.0266 | -0.0561** | 0.0061 |
| | (4.06) | (0.0568) | (0.0267) | (0.0306) |
| Birth rate (%), 1988 | 13.97 | 0.0015 | -0.0147 | 0.0100 |
| | (5.20) | (0.0259) | (0.0139) | (0.0129) |
| Death rate (%), 1988 | 9.93 | 0.1745 | 0.2168*** | 0.1281** |
| | (2.39) | (0.1826) | (0.0531) | (0.0496) |
| Log population, 1988 | 11.23 | -0.0151 | -0.1325*** | -0.0784** |
| | (1.57) | (0.0709) | (0.0299) | (0.0308) |
| *Economic conditions* | | | | |
| Transfer income p.p. (1,000$), 1988 | 2.09 | -0.3374 | -0.2855** | -0.4117 |
| | (0.38) | (0.3801) | (0.1716) | (0.3096) |
| Income p.p. (1,000$), 1985 | 8.69 | 0.1093 | 0.0822** | 0.0438 |
| | (1.83) | (0.0687) | (0.0383) | (0.0348) |
| Federal funds p.p. (1,000$), 1986 | 3.03 | -0.1507 | -0.0548** | -0.0213 |
| | (1.43) | (0.0903) | (0.0333) | (0.0242) |
| Unemployment rate (%), 1986 | 8.53 | 0.0037 | 0.0207 | 0.0174 |
| | (3.61) | (0.0388) | (0.0153) | (0.0118) |
| *Agriculture* | | | | |
| Value of produces sold per farm (million $), 1987 | 0.69 | -3.366* | -0.6388 | 0.1378 |
| | (0.81) | (1.712) | (0.6030) | (0.3530) |
| Average farm size (1,000 acres), 1987 | 0.89 | -0.0172 | -0.0433* | -0.0010 |
| | (1.96) | (0.0745) | (0.0216) | (0.0188) |
| State FE | | | | ✓ |
| Observations | | 49 | 857 | 857 |
| $R^2$ | | 0.5505 | 0.1798 | 0.3567 |
| Adjusted $R^2$ | | 0.3836 | 0.1671 | 0.3074 |

Notes: The table presents coefficients and standard errors clustered at state level (in parenthesis). Regressions are weighted by population of 1988. Both CNS anomaly rate and baseline characteristics are aggregated to state or CZ-by-state level. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. 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.

Goodman-Bacon and Sant'Anna, 2024); (ii) Continuous exposure assumes a linear relationship between pre-existing CNS anomaly rates and local maternal folate deficiency, which is possibly untrue in reality. In contrast, models using binary exposure measures possibly have less variation but do not require the stricter parallel trend assumption. They rely on a more realistic assumption: regions with higher pre-existing CNS anomaly rates are likely to have a

TABLE 2: CORRELATION BETWEEN PRE-EXISTING CNS ANOMALY RATE AND FOLATE MEASURES

| | Serum foalte (1) | RBC folate (2) |
|---|---|---|
| CNS anomaly rate | -0.5164* | -11.99** |
| | (0.2687) | (4.709) |
| $R^2$ | 0.0007 | 0.0014 |
| Observations | 10,842 | 10,913 |

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 (26 CZ-by-state units) from 13 states.

correspondingly higher extent of folate deficiency.

# 6 Results

I begin by presenting descriptive evidence on folate content in foods, dietary folate intake, blood folate concentrations, and congenital anomalies before and after fortification. Following this, I employ a cohort difference-in-differences framework to provide causal evidence on birth outcomes, test scores, and school enrollment in young adulthood.

## 6.1 Folate content in foods increases after folic acid fortification

First, I observe that folate content in foods increased after fortification. The Continuing Survey of Food Intakes by Individuals (CSFII), conducted by the USDA, offers valuable insights into the food consumption and nutritional intake of Americans. Using data from the CSFII 1994-1996 and 1998 surveys, I can observe folate content in sampled foods both before and after fortification, based on USDA's calculation from recipes. The CSFII reports reasons for changes in food composition, including enrichment or fortification, reformulation, agricultural or processing modifications, and the Nutrition Labeling and Education Act. As illustrated in Figure 5, fortification significantly increased folic acid content across a wide range of foods. Overall, folic acid levels rose in over 350 basic food items due to fortification (Anderson et al., 2001).

## 6.2 Dietary folate intake increases after folic acid fortification

Second, I observe a significant increase in dietary folate intake after fortification. Data from the National Health and Nutrition Examination Surveys (NHANES) reveal that dietary folate intake rose by nearly 50%, approaching the recommended daily level of 400 $\mu g$ in the post-fortification period (Figure 6). Notably, these intake figures exclude folic acid obtained from nutritional supplements and medications (Ahluwalia et al., 2016).

## 6.3 Blood folate increased after folic acid fortification

Third, in line with the increase in dietary folate intake, blood folate concentrations also rose significantly following fortification. Using data from the same NHANES dataset as dietary folate intake, Figure 7 illustrates trends in serum and red blood cell (RBC) folate concentrations—both key biomarkers of folate deficiency. The results show that serum folate levels more than doubled, while RBC folate levels increased by nearly 50%, indicating a sustained improvement in folate absorption. Blood folate measurement remain unchanged from 1998 to 2006 (Pfeiffer et al., 2012).

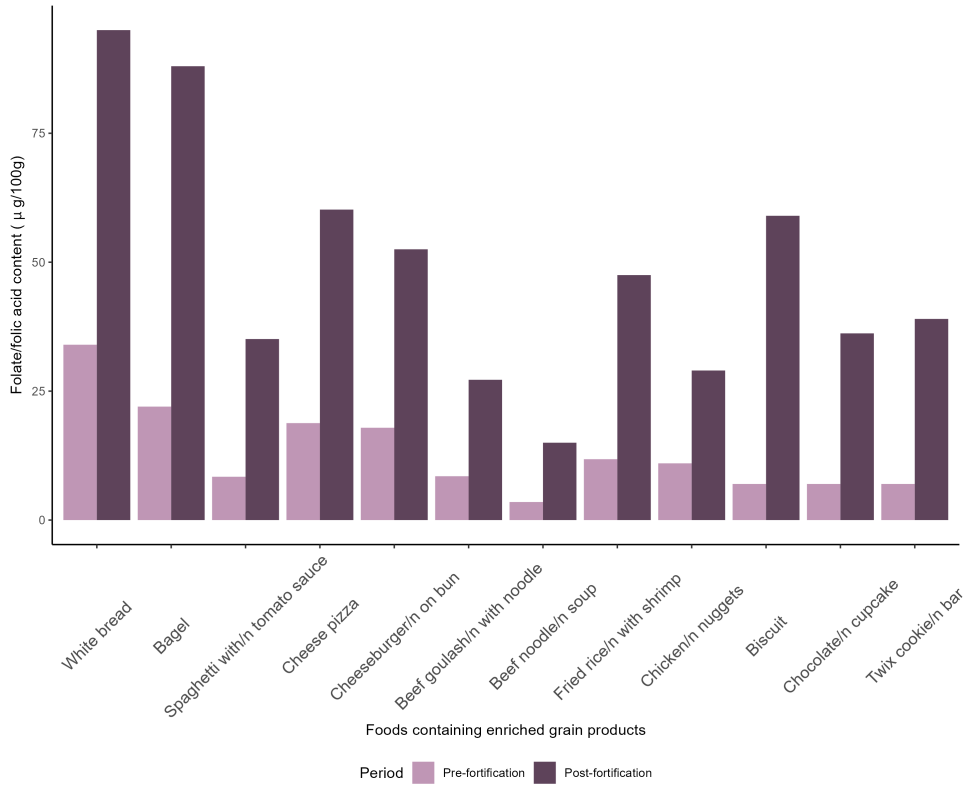



FIGURE 5: CHANGES IN FOLATE CONTENTS IN SELECTED FOODS DUE TO FORTIFICATION

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.

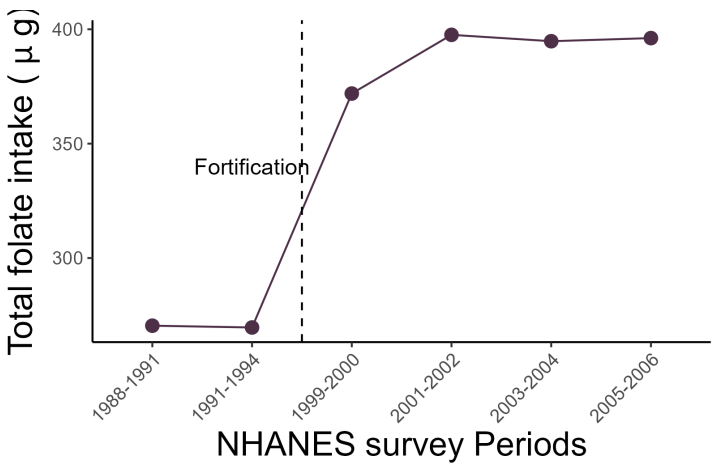



FIGURE 6: DIETARY FOLATE CONCENTRATIONS BEFORE AND AFTER FORTIFICATIO

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.

## 6.4 Congenital anomalies decline after folic acid fortification

Fourth, as folate intake and absorption increased, there was a corresponding decline in the incidence of central nervous system (CNS) anomalies. After a stable period from 1992 to 1996, CNS

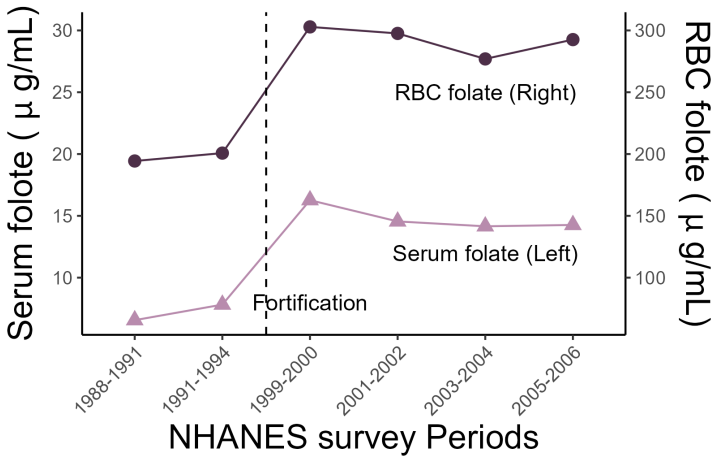
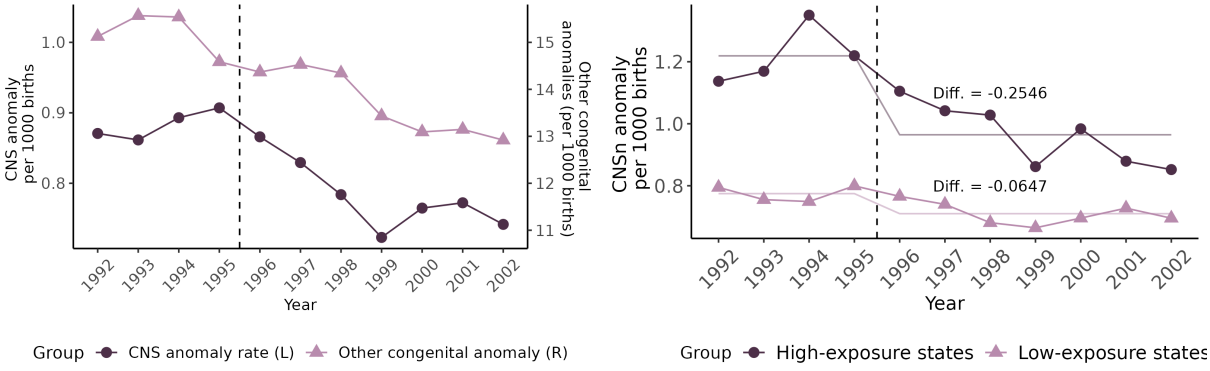



FIGURE 7: BLOOD FOLATE CONCENTRATIONS BEFORE AND AFTER FORTIFICATIONN

Notes: Data on dietary 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.

anomaly rates significantly declined following fortification. Concerns that this decline might be attributed to broader healthcare improvements are mitigated by the stability of other CNS anomaly rates during the same period, as shown in Figure 8a. Moreover, Figure 8b shows that CNS anomaly rates declined in both high- and low-exposure regions, with a more pronounced decline in the high-exposure regions.




(A) CNS and non-CNS anomaly rates

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

FIGURE 8: TRENDS IN CONGENITAL ANOMALY RATES

Notes: The unit of CNS Anomaly rate is cases per 1,000 births. High exposure is defined with top 25% pre-existing CNS anomaly rate.

### 6.5 More infants born to disadvantaged mothers is born after folic acid fortification

Fifth, I find folic acid fortification increases share of births given by disadvantaged mothers. The model used to estimate effects on mortality selection is a modified version of Equation 1:

$$Y_{cj\tau} = \beta \text{CNS anomaly rate}_j \times Post_{c\tau} + \zeta_c + \psi_\tau + C_{cj\tau} + \varepsilon_{cj\tau}. \tag{3}$$

In this specification, $Y_{cj\tau}$ represents the mean outcomes of interest for births in county $i$ and quarter-year of birth $\tau$. The variable CNS anomaly rate$_j$ denotes the state-level pre-existing CNS anomaly rate, and $Post_{c\tau}$ indicates whether a cohort was exposed to fortification. The term $\zeta_c$ represents county-of-birth fixed effects, which control for unobserved, cohort-invariant factors, while $\psi_\tau$ captures quarter-and-year-of-birth fixed effects to account for cohort-specific shocks. The control variables $C_{cj\tau}$ include: (i) state-level policies such as Medicaid eligibility for pregnant women, mental health parity law rollouts, and indicators for the first major AFDC waiver and TANF implementation; (ii) a Bartik-style measure of the county-by-year unemployment rate; and (iii) baseline county characteristics interacted with quarter-and-year-of-birth to capture time-varying local factors. The error term is $\varepsilon_{cj\tau}$.

Table 3 shows that, in high-exposure regions defined by the 75th percentile of pre-existing CNS anomaly rates, folic acid fortification increases shares of births given by mothers who are not older than 22 years old by 0.41 percentage points, mothers with less than college education by 0.58 percentage points, unmarried mothers by 0.86 percentage points, and mothers without adequate prenatal care by 1.39 percentage points, compared to low-exposure regions defined by the 25th percentile of pre-existing CNS anomaly rates. Figure 9 shows that these estimates are not driven by pre-fortification trends between high- and low-exposure regions, with the exception of unmarried mothers. The results are consistent when using a binary exposure model (see Table B2 and Figure B2).

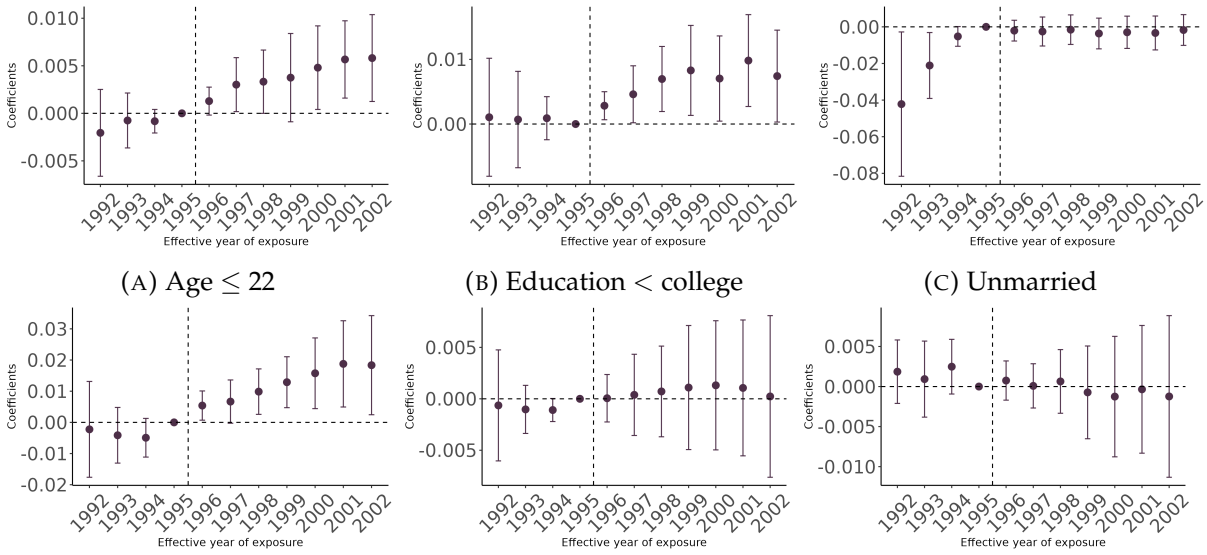
One possible explanation for the increased share of births among disadvantaged mothers is the improved survival rate of their fetuses. However, we lack comprehensive data on all fetuses, as the fetal death files from Vital Statistics Data primarily include a small subset of fetuses, most of which are older than 20 weeks.

The results on birth shares suggest that effects of folic acid fortification on subsequent outcomes may be attenuated by the rising proportion of births given by disadvantaged mothers. These newborns are more likely to face challenges in both the short and long term, po-

TABLE 3: EFFECTS OF FOLIC ACID FORTIFICATION ON MATERNAL CHARACTERISTICS

| | Share of mothers with following characteristics | | | | | |
|---|---|---|---|---|---|---|
| | Age $\leq$ 22 | Education $<$ college | Unmarried | Inadequate prenatal care | Non-white | Hispanic |
| | (1) | (2) | (3) | (4) | (5) | (6) |
| CNS anomaly rate $\times$ Post | 0.0041*** | 0.0058* | 0.0086*** | 0.0139** | 0.0014 | -0.0012 |
| | (0.0013) | ( 0.0030) | (0.0025) | (0.0058) | (0.0025) | (0.0032) |
| Observations | 111,683 | 111,678 | 111,683 | 111,683 | 111,683 | 111,678 |
| $R^2$ | 0.9095 | 0.9394 | 0.9092 | 0.7837 | 0.9847 | 0.9906 |
| Dep. var. mean | 0.2697 | 0.5462 | 0.3198 | 0.2341 | 0.1976 | 0.1801 |

Notes: Regressions and dependent variable mean are weighted by number of births in each cell. In parentheses are standard errors clustered at state-of-birth level. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. Both coefficients and standard errors are rescaled by the difference between 25th percentile and 75th percentile state-level CNS anomaly rates (0.57). I control for all baseline county-level characteristics interacted with linear time trend in all regressions.




(A) Age $\leq$ 22 (B) Education $<$ college (C) Unmarried

(D) Inadequate prenatal care (E) Non-white (F) Hispanic

FIGURE 9: DYNAMIC EFFECTS OF FOLIC ACID FORTIFICATION ON MATERNAL CHARACTERISTICS, CONTINUOUS EXPOSURE

Notes: Regressions are weighted by number of births in each cell. Standard errors are clustered at state-of-birth level. Both coefficients and standard errors are rescaled by the difference between 25th percentile and 75th percentile state-level CNS anomaly rates (0.57). All regressions include quarter-and-year-of-birth FE and county-of-maternal-residence FE. I control for all baseline county-level characteristics interacted with linear time trend in all regressions.

tentially lowering the average subsequent outcomes. As a result, the impact of fortification on future outcomes may appear negative, null, or positive, depending on the balance between the improvements in outcomes for those who would have been born regardless of fortification and the "diluting" effects from the additional disadvantaged births that occurred due to fortification.

### 6.6 Effects of folic acid fortification on young adults' outcomes

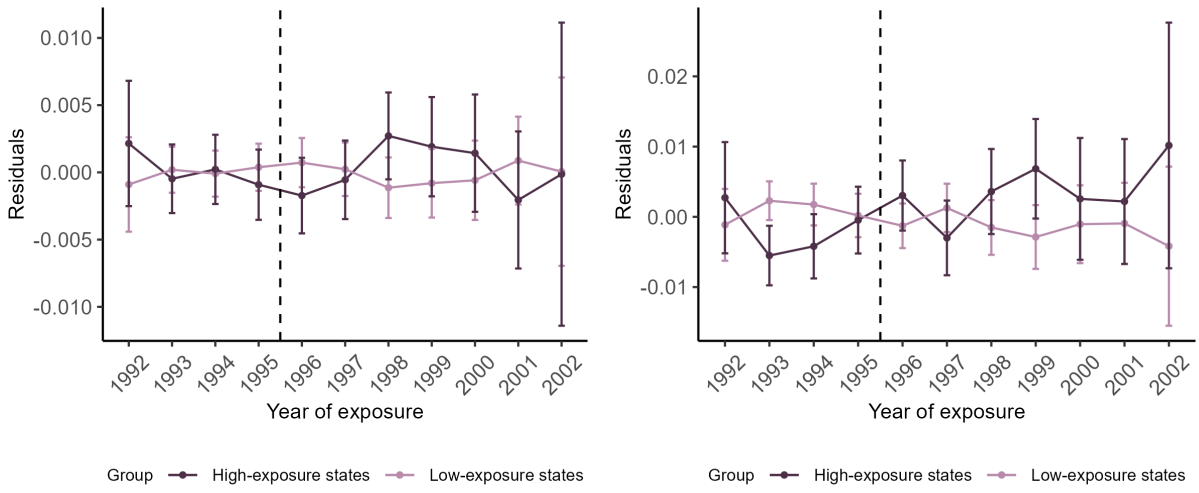
Finally, I find that in-utero exposure to folic acid fortification increases school enrollment and reduces labor supply at young adulthood. In this section, I first provide evidence of the effects of in-utero exposure on the probability of young adults earning a high school diploma or equivalent credentials, as well as their likelihood of enrolling in post-secondary education. I then discuss how the labor supply of young adults reflects this shift.

#### 6.6.1 In-utero exposure to folic acid fortification increases post-secondary education enrollment among young adults

Figure 10 presents a regression-adjusted graphical overview of my results. I plot the average residuals for each birth cohort and for high- and low-exposure groups, adjusting for all regressors listed in Equation 1, except for the key treatment interaction term, CNS anomaly rates $\times$ *Postit*. Figure 10a shows that, even after accounting for potential noise and confounders, the likelihood of young adults earning a high school diploma or equivalent credentials remains relatively unchanged regardless of in-utero exposure to folic acid fortification. Figure 10b shows that the regression-adjusted probability of enrolling in post-secondary education increases in high-exposure states (those with pre-existing CNS anomaly rates in the top 25%), particularly after 1998 when the fortification mandate was fully implemented. In low-exposure states, the trends appear relatively flat. An alternative visualization (see Figure B3) plots the dependent variables for each birth cohort in high- and low-exposure regions without regression adjustment. While the trends are similar to Figure 10, they are less apparent, as the effects of fortification are relatively small compared to the mean values of the dependent variables.

Table 4 indicates that in-utero exposure to folic acid fortification does not affect probability of young adults earning high school diploma or equivalent credentials. This is likely due to high baseline rates in this outcomes: over 90% of young adults have high school diploma or equivalent credentials, making little room for this outcomes to improve despite the potential increase in cognitive ability due to in-utero exposure to folic acid fortification.

Table 5 show that in-utero exposure to folic acid fortification increases the likelihood of




(A) High school diploma or the equivalents (B) All post-secondary education enrollment

FIGURE 10: COHORT TRENDS IN RESIDUAL EDUCATIONAL OUTCOMES OF YOUNG ADULTS

Notes: These graphs present cohort average educational outcomes of young adults for high- and low-exposure regions. High exposure is defined as states with top 25% pre-existing CNS anomaly rate; low-exposure is defined otherwise. The average and standard errors are weighted by individual sample weight.

TABLE 4: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON THE LIKELIHOOD OF YOUNG ADULTS EARNING HIGH SCHOOL DIPLOMA/EQUIVALENT CREDENTIALS

| | Continuous (1) | Top 30 (2) | Top 25 (3) | Top 20 (4) |
|---|---|---|---|---|
| | | Panel A: full sample | | |
| CNS anomaly rate × Post | -0.0001 | | | |
| | (0.0015) | | | |
| High CNS anomaly × Post | | 0.0012 | 0.0007 | 0.0012 |
| | | (0.0013) | (0.0013) | (0.0014) |
| Observations | 1,440,521 | 1,440,521 | 1,440,521 | 1,440,521 |
| $R^2$ | 0.0123 | 0.0123 | 0.0123 | 0.0123 |
| Dep. var. mean | 0.9307 | 0.9307 | 0.9307 | 0.9307 |
| | | Panel B: nonmovers | | |
| CNS anomaly rate × Post | -0.0012 | | | |
| | (0.0014) | | | |
| High CNS anomaly × Post | | 0.0005 | 0.0001 | 0.0002 |
| | | (0.0015) | (0.0015) | (0.0016) |
| Observations | 1,027,413 | 1,027,413 | 1,027,413 | 1,027,413 |
| $R^2$ | 0.0134 | 0.0134 | 0.0134 | 0.0134 |
| Dep. var. mean | 0.9253 | 0.9253 | 0.9253 | 0.9253 |

Notes: Standard errors are clustered on state of birth. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors of continuous exposure specification are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, survey-year fixed effects, gender, race, Hispanic origin, Medicaid eligibility, exposure to mental health parity laws, welfare reforms, local unemployment rates, and county-level baseline characteristics interacted with time trends.

young adults enrolling in post-secondary education. The effect is more pronounced among non-movers, aligning with the expectation that fortification has a greater impact on disadvantaged populations. Specifically, in-utero exposure increases the probability of post-secondary enrollment by 0.69 percentage points for young adults born in states with relatively high pre-existing CNS anomaly rates (at the 75th percentile) compared to those born in states with lower rates (at the 25th percentile). For non-movers, the effect is even larger, with an increase of 0.98 percentage points, and more precise.

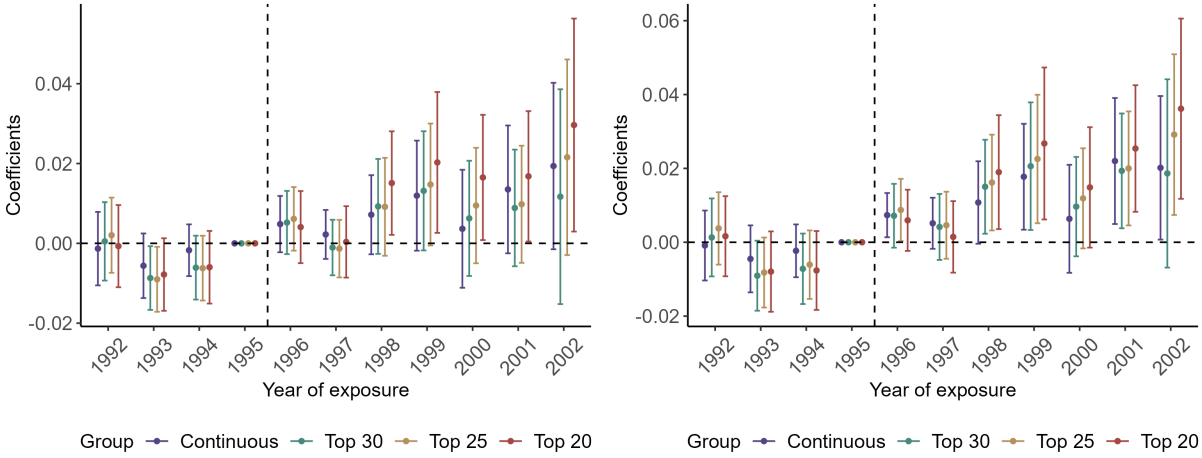
TABLE 5: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON THE LIKELIHOOD OF YOUNG ADULTS ENROLLING IN POST-SECONDARY EDUCATION

| | Continuous (1) | Top 30 (2) | Top 25 (3) | Top 20 (4) |
|---|---|---|---|---|
| | | Panel A: full sample | | |
| CNS anomaly rate × Post | 0.0069* | | | |
| | (0.0040) | | | |
| High CNS anomaly × Post | | 0.0095** | 0.0103** | 0.0117** |
| | | (0.0039) | (0.0041) | (0.0050) |
| Observations | 1,440,521 | 1,440,521 | 1,440,521 | 1,440,521 |
| $R^2$ | 0.1363 | 0.1363 | 0.1363 | 0.1363 |
| Dep. var. mean | 0.3627 | 0.3627 | 0.3627 | 0.3627 |
| | | Panel B: nonmovers | | |
| CNS anomaly rate × Post | 0.0097*** | | | |
| | (0.0035) | | | |
| High CNS anomaly × Post | | 0.0143*** | 0.0150*** | 0.0144*** |
| | | (0.0038) | (0.0039) | (0.0048) |
| Observations | 1,027,413 | 1,027,413 | 1,027,413 | 1,027,413 |
| $R^2$ | 0.1341 | 0.1341 | 0.1341 | 0.1341 |
| Dep. var. mean | 0.3580 | 0.3580 | 0.3580 | 0.3580 |

Notes: Standard errors are clustered on state of birth. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors of continuous exposure specification are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, survey-year fixed effects, gender, race, Hispanic origin, Medicaid eligibility, exposure to mental health parity laws, welfare reforms, local unemployment rates, and county-level baseline characteristics interacted with time trends.

Figure 11 presents the dynamic effects of in-utero exposure to folic acid fortification on post-secondary education enrollment. These dynamic effects align with the difference-in-difference estimates. For all educational outcomes, the pre-fortification cohort-specific coefficients are close to zero, indicating that the effects of folic acid fortification are not influenced by pre-fortification differential trends in outcomes. Figure B4 shows that the cohort-specific coefficients for college enrollment among the 19-to-22-year-olds shift upward immediately following folic acid fortification, with this pattern being more pronounced among non-movers, while the effects on graduate/professional school enrollment for those over 22 are primarily

driven by the cohort born in 1998. Noticeably, estimates of the dynamic effects on graduate/professional school enrollment for those over 22 are noisier due to the smaller sample size for each birth cohort.




(A) Full sample (B) Nonmovers

FIGURE 11: DYNAMIC EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON POST-SECONDARY EDUCATION ENROLLMENT

Notes: Standard errors are clustered on state of birth. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. Controls and other fixed effects include state-by-year share of Medicaid-eligible pregnant women, ACDF and TANF waiver dummies, state mental health parity law implementation dummy, race fixed effect, survey-year fixed effects, Hispanic origin, gender, Bartik-style change in state unemployment rate at birth, and county-level pre-intervention characteristics interacted with linear time trend.

We cannot interpret this effect as increased human capital investment if enrollment rises but is also delayed, as delayed enrollment is not distinguishable in the aforementioned coefficients. However, Table 6 shows that the effect is driven by increases in both college enrollment among 19- to 22-year-olds and graduate/professional school enrollment among those over 22. Specifically, for young adults born in states with relatively high pre-existing CNS anomaly rates, compared to those born in states with lower rates, in-utero exposure increases the probability of college enrollment by 0.88 percentage points for those aged 19–22, and the likelihood of graduate or professional school enrollment by 0.39 percentage points for those over 22. This suggests that the rise in post-secondary education occurs at the appropriate ages. For nonmovers, the effect on college enrollment for those aged 19-22 is also substantially larger, with an increase of 1.27 percentage points, while the effect on graduate/professional school enrollment for those over 22 is of similar magnitude, with an increase of 0.4 percentage points.

I replace the continuous pre-existing CNS anomaly rates with a binary indicator for high-exposure regions and re-estimate the analyses. High-exposure regions are defined as states in the top 30%, 25%, and 20% of the birth-weighted distribution of pre-existing CNS anomaly

TABLE 6: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON THE LIKELIHOOD OF YOUNG ADULTS ENROLLING IN POST-SECONDARY EDUCATION, BY AGE GROUP

| | College enrollment, $19 \leq$ age $\leq 22$ | | | | Graduate or professional school enrollment, age $> 22$ | | | |
|---|---|---|---|---|---|---|---|---|
| | Continuous (1) | Top 30 (2) | Top 25 (3) | Top 20 (4) | Continuous (5) | Top 30 (6) | Top 25 (7) | Top 20 (8) |
| **Panel A: full sample** | | | | | | | | |
| CNS anomaly rate × Post | 0.0088* | | | | 0.0039*** | | | |
| | (0.0045) | | | | (0.0013) | | | |
| High CNS anomaly × Post | | 0.0148*** | 0.0156*** | 0.0111** | | 0.0048*** | 0.0052*** | 0.0049** |
| | | (0.0049) | (0.0050) | (0.0049) | | (0.0016) | (0.0016) | (0.0020) |
| Observations | 807,669 | 807,669 | 807,669 | 807,669 | 632,852 | 632,852 | 632,852 | 632,852 |
| $R^2$ | 0.0623 | 0.0623 | 0.0623 | 0.0623 | 0.0110 | 0.0110 | 0.0110 | 0.0110 |
| Dep. var. mean | 0.4927 | 0.4927 | 0.4927 | 0.4927 | 0.0608 | 0.0608 | 0.0608 | 0.0608 |
| **Panel B: nonmovers** | | | | | | | | |
| CNS anomaly rate × Post | 0.0127** | | | | 0.0040** | | | |
| | (0.0048) | | | | (0.0019) | | | |
| High CNS anomaly × Post | | 0.0217*** | 0.0227*** | 0.0161*** | | 0.0055** | 0.0062** | 0.0040* |
| | | (0.0055) | (0.0056) | (0.0052) | | (0.0024) | (0.0024) | (0.0024) |
| Observations | 581,820 | 581,820 | 581,820 | 581,820 | 445,593 | 445,593 | 445,593 | 445,593 |
| $R^2$ | 0.0598 | 0.0598 | 0.0599 | 0.0598 | 0.0116 | 0.0116 | 0.0116 | 0.0116 |
| Dep. var. mean | 0.4821 | 0.4821 | 0.4821 | 0.4821 | 0.0525 | 0.0525 | 0.0525 | 0.0525 |

Notes: Standard errors are clustered on state of birth. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors of continuous exposure specification are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, survey-year fixed effects, gender, race, Hispanic origin, Medicaid eligibility, exposure to mental health parity laws, welfare reforms, local unemployment rates, and county-level baseline characteristics interacted with time trends.

rates. The results using binary exposure indicators align with those from the continuous measure but offer clearer interpretation. Depending on the thresholds for high- and low-exposure regions, young adults exposed to folic acid fortification in utero in high-exposure states show no significant change in high school graduation rates. However, they demonstrate a 0.77 to 1.17 percentage point increase in the probability of enrolling in post-secondary education, a 1.11 to 1.48 percentage point increase in college enrollment among those aged 19–22, and a 0.48 to 0.51 percentage point increase in enrollment in graduate or professional programs for individuals over 22.

For non-movers, a similar pattern emerges, with generally larger increases in post-secondary enrollment. Specifically, non-mover young adults experience a 1.04 to 1.44 percentage point increase in the probability of enrolling in post-secondary education, a 1.61 to 2.17 percentage point increase in college enrollment for those aged 19–22, and a 0.40 to 0.62 percentage point increase in the likelihood of enrolling in graduate or professional programs for those over 22.

The dynamic effect estimates using binary exposure indicators are also consistent with with those from the continuous measure. Under these specifications, I also do not observe any significant pre-fortification differential trends in outcomes, further supporting the validity of my earlier conclusions.

### 6.6.2 In-utero exposure to folic acid fortification decreases labor supply among the 19-to-22-year-olds but not those over 22

Having established evidence that in-utero exposure to folic acid fortification has positively influenced human capital investment in young adulthood, I now turn to its impact on labor supply. For young adults, higher labor supply does not necessarily indicate better human capital outcomes. In fact, if they choose to invest more in education, they must allocate less time to work. Consistent with this, I find a decline in labor supply among the 19- to 22-year-olds, which supports the earlier findings on increased post-secondary education enrollment.

Table 7 shows that in-utero exposure to folic acid fortification reduces likelihood of working full-time among the 19-to-22-year-olds. Full-time workers are those whose usual hours worked per week are larger than 40 hours. Specifically, depending on the definition of high-exposure regions, young adults aged 19–22 who were exposed to folic acid fortification and born in high-exposure areas experience a decline in the probability of working full-time by 0.79 to 1.54 percentage points. Similar to the effects on school enrollment, the effects on labor supply are more pronounced among nonmovers: they experience a decline in the probability of working full-time by 1.25 to 2.36 percentage points.

Table 7 also shows that young adults over 22 do not reduce their labor supply as the 19-to-22-year-olds do. This is consistent with the smaller increase in graduate/professional school enrollment observed in earlier results, as well as the fact that a larger share of graduate/professional programs are part-time.

Figure 12 presents the dynamic effect estimates for probability of the 19-to-22-year-olds using both continuous and binary treatment variables for the full sample and non-movers. The results are consistent with the difference-in-difference estimates shown in Table 7. Overall, the cohort-specific coefficients are close to zero before fortification and shift downward after fortification, especially for non-movers and when stricter definitions of high-exposure regions are applied.

Table B3 and Figure B5 present results for usual hours worked per week, which are top-coded at 99 hours. The estimates align with findings on the probability of full-time work.

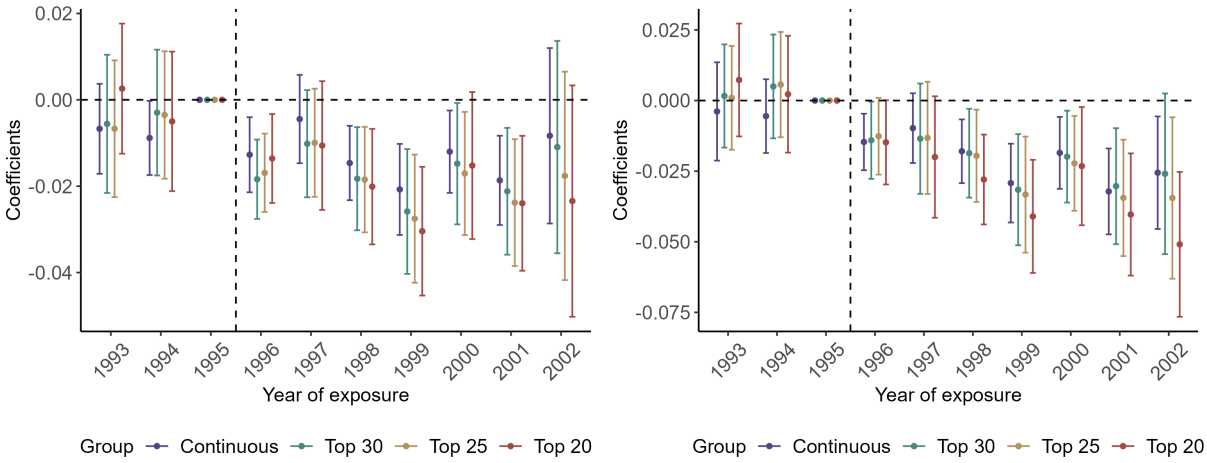
TABLE 7: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON THE LIKELIHOOD OF YOUNG ADULTS WORKING FULL-TIME, BY AGE GROUP

| | $19 \leq$ Age $\leq 22$ | | | | Age $> 22$ | | | |
|---|---|---|---|---|---|---|---|---|
| | Continuous | Top 30 | Top 25 | Top 20 | Continuous | Top 30 | Top 25 | Top 20 |
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| | | | | Panel A: full sample | | | | |
| CNS anomaly rate × Post | -0.0079* | | | | -0.0023 | | | |
| | (0.0041) | | | | (0.0032) | | | |
| High CNS anomaly × Post | | -0.0154*** | -0.0150*** | -0.0149*** | | -0.0042 | -0.0031 | 0.0002 |
| | | (0.0040) | (0.0040) | (0.0046) | | (0.0038) | (0.0039) | (0.0047) |
| Observations | 617,265 | 617,265 | 617,265 | 617,265 | 533,035 | 533,035 | 533,035 | 533,035 |
| $R^2$ | 0.0618 | 0.0618 | 0.0618 | 0.0618 | 0.0411 | 0.0411 | 0.0411 | 0.0411 |
| Dep. var. mean | 0.3995 | 0.3995 | 0.3995 | 0.3995 | 0.6816 | 0.6816 | 0.6816 | 0.6816 |
| | | | | Panel B: nonmovers | | | | |
| CNS anomaly rate × Post | -0.0125** | | | | 0.0001 | | | |
| | (0.0049) | | | | (0.0031) | | | |
| High CNS anomaly × Post | | -0.0195*** | -0.0198*** | -0.0236*** | | -0.0012 | -0.0006 | 0.0027 |
| | | (0.0057) | (0.0059) | (0.0064) | | (0.0040) | (0.0040) | (0.0048) |
| | | (0.0055) | (0.0056) | (0.0052) | | (0.0024) | (0.0024) | (0.0024) |
| Observations | 439,032 | 439,032 | 439,032 | 439,032 | 370,806 | 370,806 | 370,806 | 370,806 |
| $R^2$ | 0.0628 | 0.0628 | 0.0628 | 0.0628 | 0.0436 | 0.0436 | 0.0436 | 0.0436 |
| Dep. var. mean | 0.3935 | 0.3935 | 0.3935 | 0.3935 | 0.6702 | 0.6702 | 0.6702 | 0.6702 |

Notes: Standard errors are clustered on state of birth. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors of continuous exposure specification are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, survey-year fixed effects, gender, race, Hispanic origin, Medicaid eligibility, exposure to mental health parity laws, welfare reforms, local unemployment rates, and county-level baseline characteristics interacted with time trends.

Specifically, depending on how high-exposure regions are defined, in-utero exposure to folic acid fortification reduces usual weekly working hours for 19-to-22-year-olds by 0.07 to 0.40 hours, with a larger reduction observed among non-movers. Working hours for individuals over 22 show no clear effect.

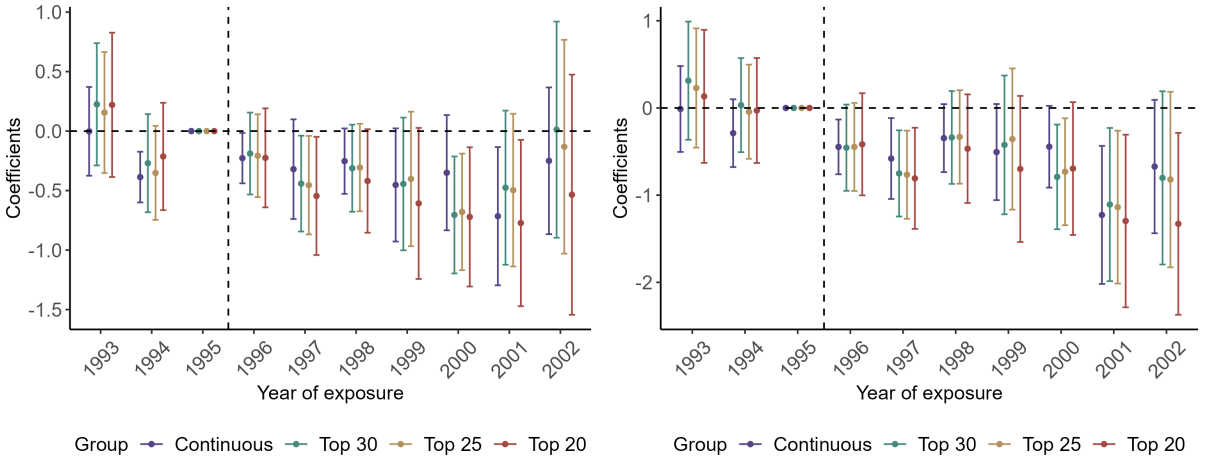
Next, I focus on the annual earnings of young adult workers, defined as individuals reporting positive usual hours worked per week. Table 8 shows results that are consistent with those for full-time employment likelihood. Among young adult workers, those exposed to folic acid fortification in utero and born in high-exposure areas experience a decline in annual earnings of \$152.8 to \$379.4. As with the effects on school enrollment and full-time employment likelihood, the impact on annual earnings is more pronounced among non-movers, who see a decline of \$357.2 to \$595.6. The dynamic effect results in Figure 13 indicate that these estimates are unlikely to be driven by pre-fortification trends.




(A) Full sample (B) Nonmovers

FIGURE 12: DYNAMIC EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON LIKELIHOOD OF THE 19-TO-22YEAR-OLDS WORKING FULL-TIME

Notes: Standard errors are clustered on state of birth. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. Controls and other fixed effects include state-by-year share of Medicaid-eligible pregnant women, ACDF and TANF waiver dummies, state mental health parity law implementation dummy, race fixed effect, survey-year fixed effects, Hispanic origin, gender, Bartik-style change in state unemployment rate at birth, and county-level pre-intervention characteristics interacted with linear time trend.




(A) Full sample (B) Nonmovers

FIGURE 13: DYNAMIC EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON ANNUAL EARNING ($1,000) OF 19-TO-22YEAR-OLD WORKERS

Notes: Standard errors are clustered on state of birth. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. Controls and other fixed effects include state-by-year share of Medicaid-eligible pregnant women, ACDF and TANF waiver dummies, state mental health parity law implementation dummy, race fixed effect, survey-year fixed effects, Hispanic origin, gender, Bartik-style change in state unemployment rate at birth, and county-level pre-intervention characteristics interacted with linear time trend.

### 6.6.3 Effects are larger for female, white, and non-Hispanic individuals

This section discusses heterogeneous effects of in-utero exposure to folic acid fortification on young adults' outcomes by gender, race, and ethnicity. I divide full sample and nonmovers

TABLE 8: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON ANNUAL EARNING ($1,000) OF YOUNG ADULT WORKERS, BY AGE GROUP

| | $19 \leq$ Age $\leq 22$ | | | | Age $> 22$ | | | |
|---|---|---|---|---|---|---|---|---|
| | Continuous (1) | Top 30 (2) | Top 25 (3) | Top 20 (4) | Continuous (5) | Top 30 (6) | Top 25 (7) | Top 20 (8) |
| | | | | Panel A: full sample | | | | |
| CNS anomaly rate × Post | -0.1528 | | | | -0.2388 | | | |
| | (0.1065) | | | | (0.2320) | | | |
| High CNS anomaly × Post | | -0.2887* | -0.2576* | -0.3794** | | -0.1805 | -0.0858 | -0.1199 |
| | | (0.1513) | (0.1512) | (0.1810) | | (0.2745) | (0.2839) | (0.3166) |
| Observations | 617,265 | 617,265 | 617,265 | 617,265 | 533,035 | 533,035 | 533,035 | 533,035 |
| $R^2$ | 0.0607 | 0.0607 | 0.0607 | 0.0607 | 0.1000 | 0.1000 | 0.1000 | 0.1000 |
| Dep. var. mean | 14.4654 | 14.4654 | 14.4654 | 14.4654 | 33.5725 | 33.5725 | 33.5725 | 33.5725 |
| | | | | Panel B: nonmovers | | | | |
| CNS anomaly rate × Post | -0.3572** | | | | 0.2128 | | | |
| | (0.1364) | | | | (0.2830) | | | |
| High CNS anomaly × Post | | -0.5956*** | -0.5538*** | -0.5829** | | 0.1790 | 0.2491 | 0.4755 |
| | | (0.1783) | (0.1781) | (0.2207) | | (0.3001) | (0.3064) | (0.3749) |
| Observations | 439,032 | 439,032 | 439,032 | 439,032 | 370,806 | 370,806 | 370,806 | 370,806 |
| $R^2$ | 0.0622 | 0.0622 | 0.0622 | 0.0622 | 0.1026 | 0.1026 | 0.1026 | 0.1026 |
| Dep. var. mean | 14.4783 | 14.4783 | 14.4783 | 14.4783 | 32.0269 | 32.0269 | 32.0269 | 32.0269 |

Notes: Standard errors are clustered on state of birth. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors of continuous exposure specification are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, survey-year fixed effects, gender, race, Hispanic origin, Medicaid eligibility, exposure to mental health parity laws, welfare reforms, local unemployment rates, and county-level baseline characteristics interacted with time trends.

into female and male, white and non-white, and Hispanic and non-Hispanic and present results using both continuous pre-existing CNS anomaly rate and binary indicator for high-exposure states (defined as states in the top 25% of the birth-weighted distribution of pre-existing CNS anomaly rates).

Similar to Adhvaryu et al. (2020) who find that in-utero exposure to salt iodization has a greater effect on women's incomes compared to men's, as in Table B4, I find a larger and clearer effects of folic acid fortification on women.

Table B5 shows that in-utero exposure to folic acid fortification has a greater impact on post-secondary education enrollment among non-white individuals. However, Table B5 indicates that non-white individuals exposed to fortification and born in high-exposure states do not experience a larger decline in labor supply compared to their white counterparts. This is likely because the increase in post-secondary education enrollment among non-white individuals is driven by those who would otherwise not be working (the idlers).

I also find that the previously observed effects are primarily driven by the non-Hispanic population. For the Hispanic population, the effects of folic acid fortification on both educational and labor outcomes are either small or statistically insignificant. One possible explanation is that U.S.-born Hispanic individuals, who are more likely to have immigrant parents, may be influenced to consume more ethnic foods that are not fortified with folic acid.

# 7 Robustness

This section examines the robustness of the earlier findings, with a focus on key outcomes: post-secondary enrollment among all young adults and the likelihood of full-time employment for individuals aged 19 to 22. I present results using both a continuous measure of pre-existing CNS anomaly rates and a binary indicator for high-exposure states. The binary indicator addresses the possibility that the continuous measure may not fully capture actual levels of pre-existing folate deficiency.

## 7.1 Additional controls

First, I test the robustness of my results by adding additional control variables. In Table B6, I include census region-of-birth dummies interacted with a linear cohort trend to check if earlier estimates were influenced by region-specific trends. In Table B7, I add state-of-residence fixed effects to account for potential postnatal migration effects. Additionally, in Table B8, I control for the mean values of dependent variables from 1989 to 1992, interacted with a post-fortification dummy, to account for mean reversion. The inclusion of these controls does not significantly alter the results.

## 7.2 Placebo test

Second, I create a sample of young adults born between 1983 and 1992 and within the same age range. I then re-run the regressions for these cohorts to verify that the earlier estimates are not driven by long-term cohort trends. As shown in Table B9, the effects of in-utero exposure to folic acid fortification either become statistically noisy or are absorbed by other control variables due to collinearity, further supporting the validity of my original results.

## 7.3 Randomization test

To further assess the robustness of my results against random noise, I randomly assign pre-existing CNS anomaly rates to different states 1,000 times and re-run the regressions with these randomized exposures. The distribution of these simulated effects is plotted alongside the ac-

tual effects in Figure B6. The randomization test results are consistent with my earlier findings: the actual effects fall within the tails of the distribution of simulated effects for the statistically significant estimates. This suggests that my earlier findings are unlikely to be the result of random noise.

# 8 Discussion

## 8.1 Interpretation of coefficients

Based on the simple conceptual framework described in Section 3, I argue that the regression results on post-secondary education enrollment ($\beta$) can be interpreted as the marginal increase in the probability of post-secondary education caused by in-utero exposure to folic acid fortification. This increase is likely due to improvements in cognitive ability, which in turn enhance the return to post-secondary education ($\theta$). The coefficients from the regressions using continuous exposure can be expressed as follows:

$$\beta = \frac{\partial P\left(S = \tilde{\eta}\left(T - \frac{2w_0}{\theta}\right) > \bar{S}\right)}{\partial \theta} \cdot \frac{\partial \theta}{\partial \phi} \cdot \frac{\partial \phi}{\partial \kappa}, \tag{4}$$

where $P(S > \bar{S})$ denotes the probability that $S > \bar{S}$ where $\bar{S}$ is average time required to gain certain educational attainment of interest, $\phi$ represents cognitive ability, and $\kappa$ represents the intensity of in-utero exposure to folic acid fortification. The coefficients from the regressions using binary exposure can be viewed as a special case of $\beta$, where $\partial\kappa$ corresponds to the mean difference in the intensities of in-utero exposure to folic acid fortification between high- and low-exposure regions.

This decomposed expression of $\beta$ in Equation 4 allows me to estimate the effect of in-utero exposure to folic acid fortification on cognitive ability, $\frac{\partial \phi}{\partial \kappa}$, as I can draw on existing literature to obtain estimates for the other two multipliers. I use $\frac{\partial \theta}{\partial \phi} = 0.18$ from Bowles, Gintis and Osborne (2001), who find that, on average, introducing cognitive ability measures reduces the coefficients of schooling on earnings by 18%. For $\frac{\partial P\left(S=\tilde{\eta}\left(T-\frac{2w_0}{\theta}\right)>\bar{S}\right)}{\partial \theta}$, I adopt earnings elasticities of post-secondary education enrollment estimated by Wiswall and Zafar (2015), which range from 0.0358 to 0.0618 for 1% increase in expected earning depending on the major. The implied effect of in-utero exposure to folic acid fortification on cognitive ability is thus estimated to be between 0.79 and 1.37 percentage points, which is similar in magnitudes to findings in scientific literature. For example, Villamor et al. (2012) find that a daily increase of $600\mu g$ in folate

intake during the first trimester of pregnancy is associated with a 1.6 percentage point increase in children's cognitive test scores.

### 8.2 Magnitudes

In this section, I compare the long-run effects of folic acid fortification on educational outcomes with those of other nutrition enhancement programs, such as salt iodization, iron fortification of bread, and food assistance programs. Since the exposed cohorts are still relatively young, making "years of schooling" less meaningful as a measure of human capital, I translate the earlier results on post-secondary education enrollment into years of schooling for a more consistent comparison.

Conservatively, suppose the college graduation rate is 60%, that unfinished college students drop out at the end of their first year, and that the average length of a graduate program is 2 years. Given these assumptions, the positive impact of fortification on school enrollment translates into: $(40\% \times 1 + 60\% \times 4) \times 0.0088 + 2 \times 0.0039 = 0.0324$ years of schooling. The standard error is calculated as: $\sqrt{(2.8 \times 0.0045)^2 + (2 \times 0.0013)^2} = 0.0129$. As in Figure 14, the magnitude of the effect of in-utero exposure to folic acid fortification is smaller than that of both salt iodization and the food stamp program, though it is quite similar to the latter.

Despite delivering similar long-term human capital benefits, food fortification, including folic acid fortification, is significantly less costly than programs like food stamps, now known as the Supplemental Nutrition Assistance Program (SNAP). For instance, the total annual cost of folic acid fortification is estimated at $3 million, accounting for both the cost of the fortificant and the annualized expense of updating nutrition labels (Grosse et al., 2005). Salt iodization costs an estimated $2–$5 cents per person annually (World Health Organization, 2014), translating to a total annual cost of $6–$15 million, assuming an average U.S. population of 300 million in the 2000s. The annual cost of iron fortification is estimated at $21–$56 million (Baltussen, Knai and Sharan, 2004). By contrast, the average annual cost of food stamps in the 2000s was $29 billion[8]. Calculating comparable cost-effectiveness statistics for these programs is challenging. On the cost side, intervention expenses fluctuate yearly, and we have limited understanding of how general equilibrium effects—such as changes in the prices of substitutes and complements—might influence these costs over time. On the benefit side, comprehensive analysis requires accounting for all potential benefits, including medical cost savings from reduced morbidity and mortality, as well as long-term health and labor market gains for in-

[8]Source: USDA Food and Nutrition Service. https://fns-prod.azureedge.us/sites/default/files/resource-files/snap-annualsummary-11.pdf (Accessed November 29, 2024).

dividuals exposed to these interventions. While accurately estimating costs and benefits is complex, the stark difference in cost magnitudes suggests that both folic acid fortification and salt iodization achieve long-term human capital benefits comparable to those of food stamps but at a much lower cost.

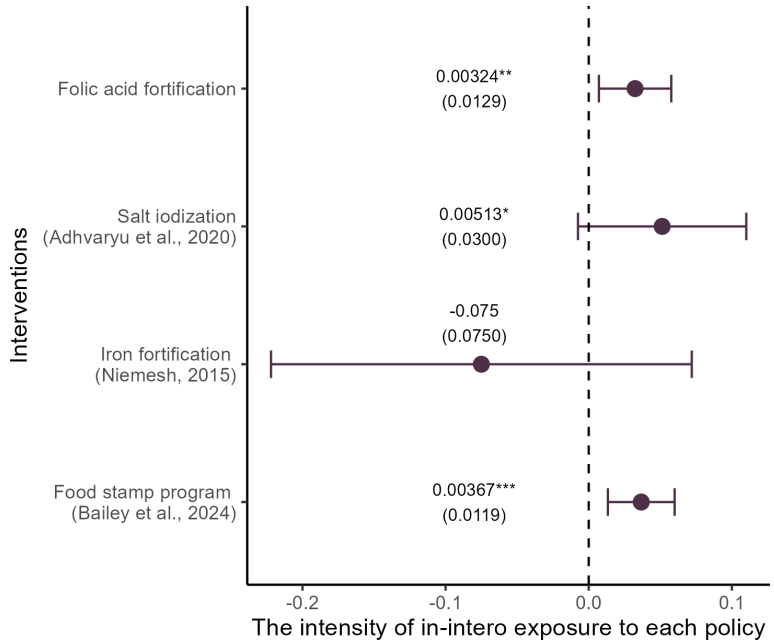



FIGURE 14: COMPARING EFFECTS OF IN-UTERO EXPOSURE TO DIFFERENT NUTRITIONAL INTERVENTIONS ON YEARS OF SCHOOLING

Notes: The effect of folic acid fortification on years of schooling is derived from its impact on post-secondary education enrollment, while all other effects are regression coefficients obtained from the corresponding studies. The estimate from Adhvaryu et al. (2020) measures the effect of prenatal exposure to salt iodization on years of schooling, using goiter rates as a proxy for iodine deficiency. Similarly, the estimate from Niemesh (2015) assesses the impact of prenatal exposure to iron fortification of bread on years of schooling, with estimated iron consumption representing iron deficiency. Both estimates are rescaled based on the interquartile range (25th to 75th percentile) of goiter rates or iron consumption, respectively. The estimate from Bailey et al. (2024) examines the effect of being born in areas with access to food stamps on years of schooling.

# 9 Conclusion

Food fortification bypasses the challenge of changing consumer behaviors, offering a potentially more effective solution to dietary problems than existing food and nutritional interventions. This paper investigates the folic acid fortification of grain products, authorized in March 1996, the most recent food fortification policy in the U.S., which was aimed at reducing the risk of folate deficiency. I leverage geographic variation in pre-existing folate deficiency and the timing of fortification to identify its effects.

First, I present evidence showing that folic acid fortification increased the folate content of various foods, raised folate intake, improved blood folate levels, and reduced birth defects.

By linking geographic variation in pre-existing folate deficiency to survey data based on place and time of birth, I then analyze how fortification influenced children's long-term outcomes. In the short term, fortification led to an increase in births among disadvantaged mothers, likely due to improved fetal survival rates. In the long run, in-utero exposure to folic acid fortification resulted in greater investments in human capital, reflected by an increased likelihood of enrolling in post-secondary education and a decline in young adults' labor supply. These effects were more pronounced for non-movers, who were more likely to come from disadvantaged families where the mother may not have afforded a nutritionally balanced diet. The results on birth outcomes suggest that the actual effects of folic acid fortification may be even larger due to mortality selection.

Using a simple model of time allocation between leisure, work, and school, I estimate the implied effect of in-utero exposure to folic acid fortification on cognitive ability to be between 0.79 and 1.37 percentage points (moving from the 25th to the 75th percentile of pre-existing CNS anomaly rates). This range is consistent with findings from the scientific literature. When comparing the magnitude of my estimates with those of other nutritional interventions, such as salt iodization and the food stamp program, I find that the effect of folic acid fortification is slightly smaller than salt iodization but comparable to the food stamp program.

This paper has some limitations. First, existing data do not allow me to distinguish marginal survivors—those who would not have been born without fortification—from others (always survivors). Since folic acid fortification affects the composition of births that survive to young adulthood, it is unclear whether the imprecise results on birth and long-term outcomes are driven by heterogeneity in effects or worse outcomes among marginal survivors. One potential explanation for the imprecision is that if fortification both improves outcomes for always survivors and increases the number of marginal survivors, the average outcomes in exposed cohorts may appear noisier, as the improved outcomes of always survivors are diluted by the potentially lower outcomes of marginal survivors. Second, I assume that folic acid fortification primarily affects pregnancies during the first trimester, as supported by the scientific literature. If fortification affects children at other life stages, which is less clear from medical research, its effects may be harder to isolate, especially given unknown migration patterns and exposure durations.

Consistent with the broader fetal origins literature, the positive long-term effects of folic acid fortification suggest that early-life interventions can yield substantial, long-lasting benefits for children. Moreover, given the evidence from the U.S., it is reasonable to believe that

folic acid fortification could have even greater benefits in developing countries, where access to folate-rich foods or folic acid supplements is more limited. The low unit cost of fortification, combined with the fact that it requires no changes in consumer behavior, makes it an especially attractive public health intervention for developing nations.

For future research, it would be valuable to extend this study to other life stages as more data become available. Currently, the oldest exposed cohorts are still in their 20s, but revisiting the effects of folic acid fortification in a few years would allow for an analysis of its impact on a wider range of human capital outcomes, such as years of education, full-time worker income, and family formation. Another promising avenue for future research is examining the effects of folic acid fortification in a developing country setting, where the impacts are expected to be more pronounced due to the higher prevalence of folate deficiency.

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# Appendix

## A Technical details

### A.1 Conceptual framework: solve for optimal time allocated to schooling

The utility maximization problem of a young adult deciding life time allocation between leisure, work, and school can be written as:

$$\begin{aligned}
&\max_{L,W,S} U(L,C) = \alpha \log(L) + (1-\alpha)\log(w \cdot W),\\
&\text{subject to: } w = w_0 + \beta S,\\
&\qquad\qquad T = L + W + S.
\end{aligned}$$

Substitute the wage function $w = w_0 + \beta S$ into the utility function and solve the optimization problem:

$$U(L,W,S) = \alpha \log(L) + (1-\alpha)\log((w_0 + \beta S) \cdot W).$$

The Lagrangian function is:

$$\mathcal{L}(L,W,S,\lambda) = \alpha \log(L) + (1-\alpha)\log((w_0 + \beta S) \cdot W) + \lambda(T - L - W - S).$$

The first-order conditions are:

$$\begin{aligned}
\frac{\partial \mathcal{L}}{\partial L} &= \frac{\alpha}{L} - \lambda = 0,\\
\frac{\partial \mathcal{L}}{\partial W} &= \frac{(1-\alpha)}{W} - \lambda = 0,\\
\frac{\partial \mathcal{L}}{\partial S} &= \frac{(1-\alpha)\beta}{w_0 + \beta S} - \lambda = 0,\\
\frac{\partial \mathcal{L}}{\partial \lambda} &= T - L - W - S = 0.
\end{aligned}$$

From the first two first-order conditions, equate the marginal utility of leisure and work: $\frac{\alpha}{L} = \frac{(1-\alpha)}{W}$. Solving for $W$: $W = \frac{(1-\alpha)}{\alpha} L$. From the third condition, equate the marginal utility of schooling to the marginal utility of work: $\frac{(1-\alpha)\beta}{w_0+\beta S} = \lambda$, we obtain:

$$\begin{aligned}
S &= \frac{(1-\alpha)L}{\alpha} - \frac{w_0}{\beta},\\
L &= \frac{\alpha\left(T + \frac{w_0}{\beta}\right)}{1 + 2(1-\alpha)}.
\end{aligned}$$

Substitute $L$ into the expression for $S$ and combine terms we have:

$$S = \frac{(1-\alpha)T}{1+2(1-\alpha)} - \frac{2(1-\alpha)w_0}{\beta\left(1+2(1-\alpha)\right)}.$$

## B Tables and figures

TABLE B1: CHARACTERISTICS OF NONMOVERS AND MOVERS, UNEXPOSED COHORTS

| Characteristics | Nonmovers | Movers | Mean difference |
|---|---|---|---|
| Age | 24.1033 | 24.3946 | -0.2913*** |
| Female | 0.4865 | 0.4922 | -0.0058*** |
| Non-white | 0.3255 | 0.2853 | 0.0402*** |
| Hispanic | 0.1986 | 0.1441 | 0.0546*** |
| Northeast | 0.1158 | 0.1407 | -0.0250*** |
| Midwest | 0.2128 | 0.2075 | 0.0053*** |
| South | 0.3519 | 0.3229 | 0.0290*** |
| West | 0.2342 | 0.2377 | -0.0036*** |

Notes: This table reports mean of characteristics of nonmovers and movers. Sample weights are used. ***, **, and * indicate that t-test are significant at the 1%, 5%, and 10% levels.

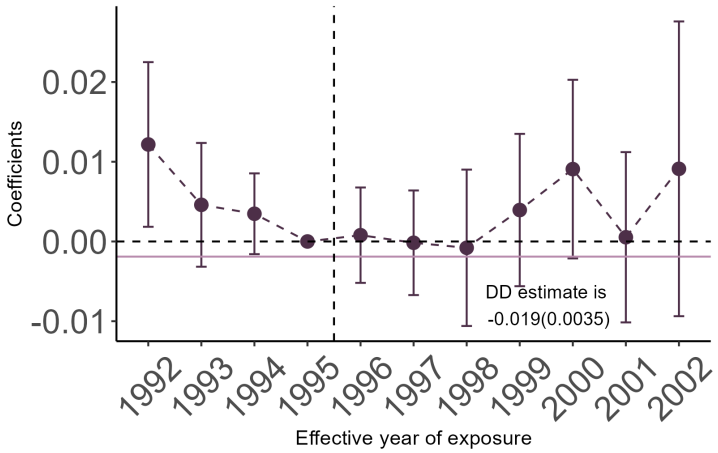


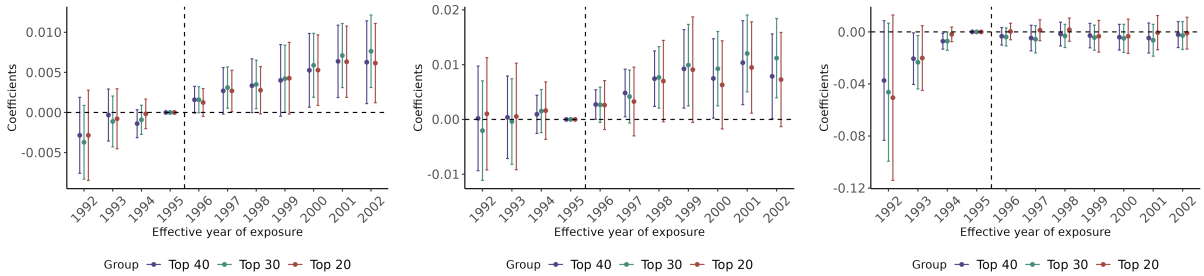

FIGURE B1: IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION DOES NOT AFFECT PROBABILITY OF BEING A NONMOVER

Notes: Dependent variable is whether individual is a nonmover. The corresponding DD estimate is -0.0019 (0.0031), both small and insignificant. Model specification is the same as Equation 1.

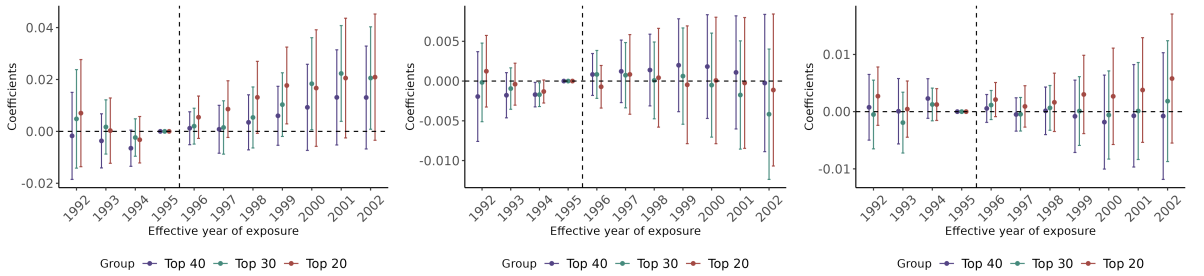
TABLE B2: EFFECTS OF FOLIC ACID FORTIFICATION ON MATERNAL CHARACTERISTICS, HIGH- VERSUS LOW-EXPOSURE REGIONS

| | Share of mothers with following characteristics | | | | | |
|---|---|---|---|---|---|---|
| | Age $\leq$ 22 | Education $<$ college | Unmarried | Inadequate prenatal care | Non-white | Hispanic |
| | (1) | (2) | (3) | (4) | (5) | (6) |
| Top 40 $\times$ Post | 0.0046*** | 0.0064** | 0.0078** | 0.0087 | 0.0025 | -0.0012 |
| | (0.0014) | (0.0031) | (0.0032) | (0.0066) | (0.0026) | (0.0035) |
| Observations | 111,683 | 111,678 | 111,683 | 111,683 | 111,683 | 111,678 |
| $R^2$ | 0.9095 | 0.9394 | 0.9090 | 0.7826 | 0.9847 | 0.9906 |
| Dependent Variable Mean | 0.2697 | 0.5462 | 0.3198 | 0.2341 | 0.1976 | 0.1801 |
| Top 30 $\times$ Post | 0.0052*** | 0.0074** | 0.0085** | 0.0098 | 0.0007 | 0.0005 |
| | (0.0014) | (0.0033) | (0.0032) | (0.0077) | (0.0026) | (0.0031) |
| Observations | 111,683 | 111,678 | 111,683 | 111,683 | 111,683 | 111,678 |
| $R^2$ | 0.9095 | 0.9394 | 0.9091 | 0.7826 | 0.9847 | 0.9906 |
| Dependent Variable Mean | 0.2697 | 0.5462 | 0.3198 | 0.2341 | 0.1976 | 0.1801 |
| Top 20 $\times$ Post | 0.0042*** | 0.0054 | 0.0104*** | 0.0137 | 0.0004 | 0.0017 |
| | (0.0016) | (0.0047) | (0.0032) | (0.0100) | (0.0032) | (0.0037) |
| Observations | 111,683 | 111,678 | 111,683 | 111,683 | 111,683 | 111,678 |
| $R^2$ | 0.90943 | 0.93932 | 0.90910 | 0.78289 | 0.98469 | 0.99061 |
| Dep. var. mean | 0.2697 | 0.5462 | 0.3198 | 0.2341 | 0.1976 | 0.1801 |

Notes: Observations are weighted by number of births in each cell. Mean, median, upper 40th quantile and upper 30th quantile are also weighted by number of birth of cells. In parentheses are standard errors clustered at state-of-birth level. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. All regressions include quarter-and-year-of-birth FE and county-of-maternal-residence FE. I control for all baseline county-level characteristics interacted with linear time trend in all regressions.



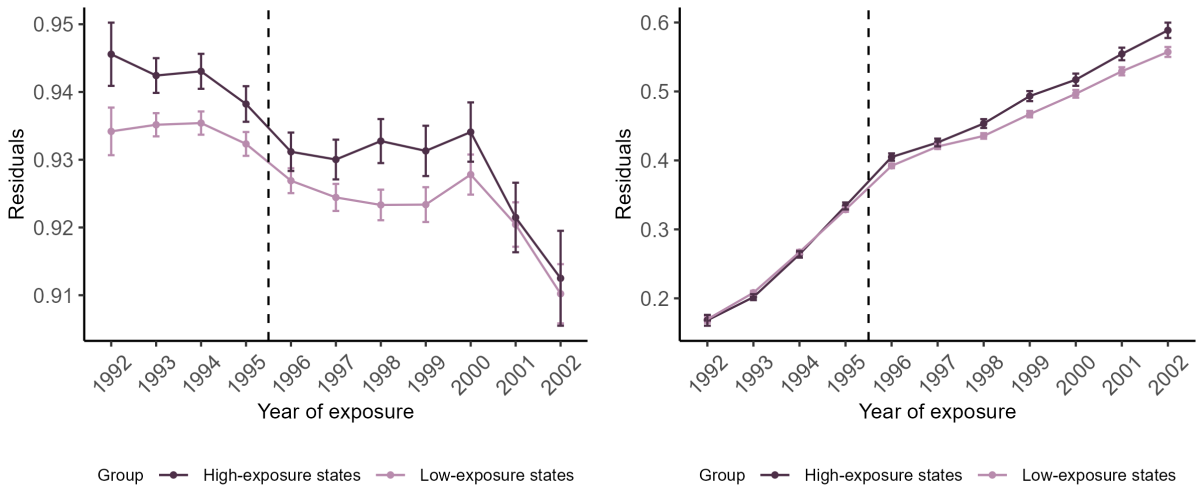

(A) Age $\leq$ 22 (B) Education $<$ college (C) Unmarried




(D) Inadequate prenatal care (E) Non-white (F) Hispanic

FIGURE B2: DYNAMIC EFFECTS OF FOLIC ACID FORTIFICATION ON MATERNAL CHARACTERISTICS, BINARY EXPOSURE

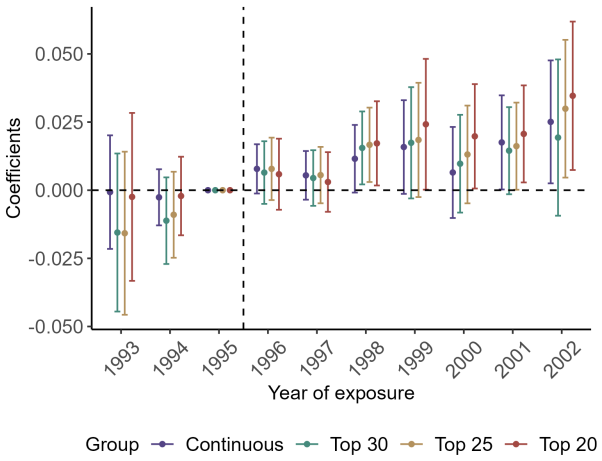
Notes: For each bin, from left to right, the points with error bar represent estimates when continuous exposure are replaced by dummies for above mean, above median, top 40, and top 30 pre-existing CNS anomaly rates. Regressions are weighted by number of births of cells. Standard errors are clustered at CZ level. I control for all baseline county-level characteristics interacted with linear time trend in all regressions. I define advantaged mothers as someones who are older than 22, are married, have attended college, are non-Hispanic white, and have received adequate prenatal care.



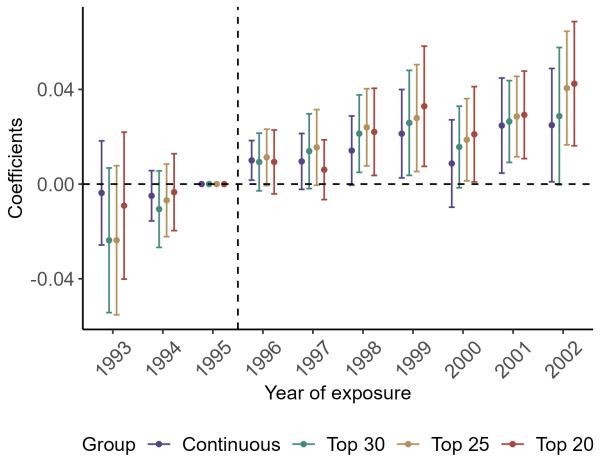

(A) High school diploma or the equivalents (B) All post-secondary education enrollment

FIGURE B3: COHORT TRENDS IN RESIDUAL EDUCATIONAL OUTCOMES OF YOUNG ADULTS

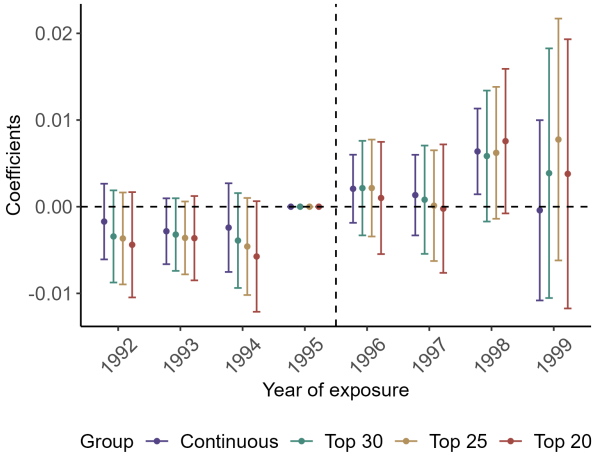
Notes: These graphs present cohort average educational outcomes of young adults for high- and low-exposure regions. High exposure is defined as states with top 30% pre-existing CNS anomaly rate; low-exposure is defined otherwise. The average and standard errors are weighted by individual sample weight.



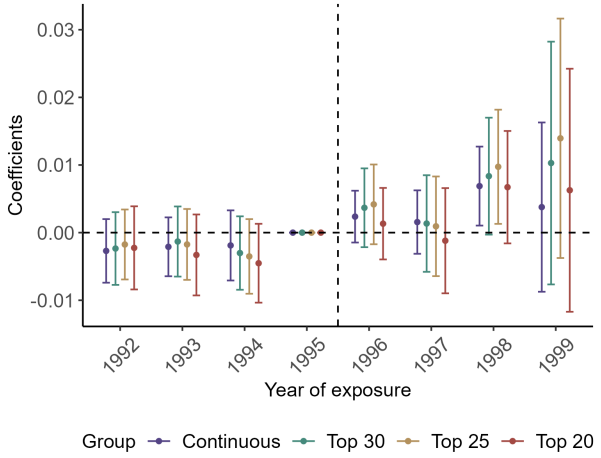

(A) College enrollment, $19 \leq$ age $\leq 22$, full sample




(B) College enrollment, $19 \leq$ age $\leq 22$, nonmovers




(C) Graduate or professional school enrollment, age $> 22$, full sample




(D) Graduate or professional school enrollment, age $> 22$, nonmovers

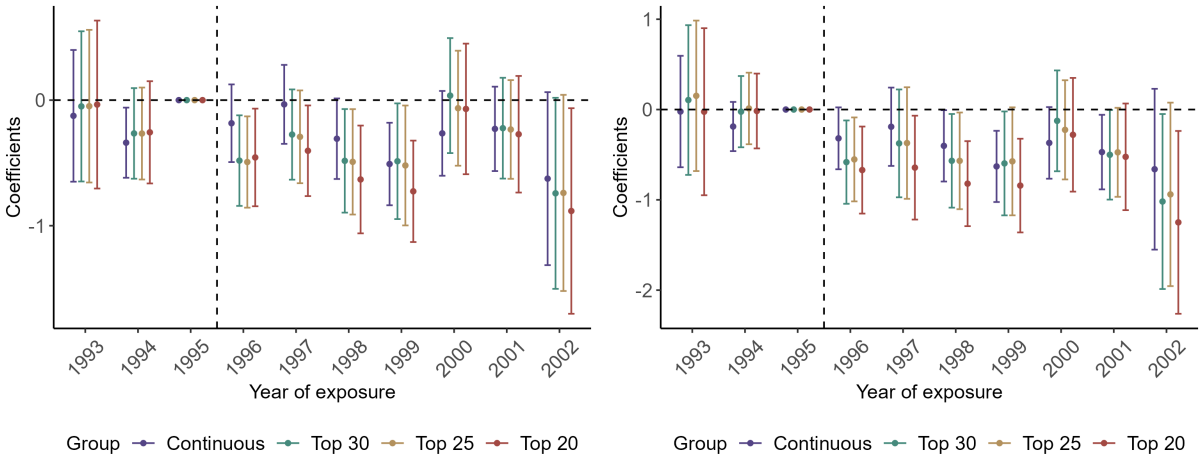
FIGURE B4: DYNAMIC EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON POST-SECONDARY EDUCATION ENROLLMENT, BY AGE GROUP

Notes: Standard errors are clustered on state of birth. All regressions and dependent variable means are weighted by ACS sample weight. Percentiles are weighted by number of births. Controls and other fixed effects include state-by-year share of Medicaid-eligible pregnant women, ACDF and TANF waiver dummies, state mental health parity law implementation dummy, race fixed effect, state-of-residence-and-survey-year fixed effect, Hispanic origin, gender, Bartik-style change in state unemployment rate at birth, and county-level pre-intervention characteristics interacted with linear time trend.

TABLE B3: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON USUAL HOURS WORKED PER WEEK OF YOUNG ADULTS, BY AGE GROUP

| | $19 \leq$ Age $\leq 22$ | | | | Age $> 22$ | | | |
|---|---|---|---|---|---|---|---|---|
| | Continuous (1) | Top 30 (2) | Top 25 (3) | Top 20 (4) | Continuous (5) | Top 30 (6) | Top 25 (7) | Top 20 (8) |
| | | | | Panel A: full sample | | | | |
| CNS anomaly rate × Post | -0.0712 | | | | 0.0253 | | | |
| | (0.1247) | | | | (0.1283) | | | |
| High CNS anomaly × Post | | -0.3019** | -0.3207** | -0.3981*** | | 0.0004 | -0.0005 | 0.0659 |
| | | (0.1238) | (0.1273) | (0.1280) | | (0.1283) | (0.1268) | (0.1581) |
| Observations | 807,669 | 807,669 | 807,669 | 807,669 | 632,852 | 632,852 | 632,852 | 632,852 |
| $R^2$ | 0.0551 | 0.0551 | 0.0551 | 0.0551 | 0.0326 | 0.0326 | 0.0326 | 0.0326 |
| Dep. var. mean | 23.5413 | 23.5413 | 23.5413 | 23.5413 | 32.1876 | 32.1876 | 32.1876 | 32.1876 |
| | | | | Panel B: nonmovers | | | | |
| CNS anomaly rate × Post | -0.2560 | | | | 0.1006 | | | |
| | (0.1640) | | | | (0.1412) | | | |
| High CNS anomaly × Post | | -0.5064*** | -0.5140*** | -0.6798*** | | 0.0846 | 0.0658 | 0.1125 |
| | | (0.1882) | (0.1951) | (0.1978) | | (0.1377) | (0.1370) | (0.1593) |
| Observations | 581,820 | 581,820 | 581,820 | 581,820 | 445,593 | 445,593 | 445,593 | 445,593 |
| $R^2$ | 0.0610 | 0.0610 | 0.0610 | 0.0610 | 0.0351 | 0.0351 | 0.0351 | 0.0351 |
| Dep. var. mean | 23.1146 | 23.1146 | 23.1146 | 23.1146 | 31.4838 | 31.4838 | 31.4838 | 31.4838 |

Notes: Standard errors are clustered on state of birth. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors of continuous exposure specification are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, survey-year fixed effects, gender, race, Hispanic origin, Medicaid eligibility, exposure to mental health parity laws, welfare reforms, local unemployment rates, and county-level baseline characteristics interacted with time trends.




(A) Full sample

(B) Nonmovers

FIGURE B5: DYNAMIC EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON USUAL HOURS WORKED PER WEEK OF 19-TO-22YEAR-OLDS

Notes: Standard errors are clustered on state of birth. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. Controls and other fixed effects include state-by-year share of Medicaid-eligible pregnant women, ACDF and TANF waiver dummies, state mental health parity law implementation dummy, race fixed effect, survey-year fixed effects, Hispanic origin, gender, Bartik-style change in state unemployment rate at birth, and county-level pre-intervention characteristics interacted with linear time trend.

TABLE B4: HETEROGENEITY OF LONG RUN EFFECTS OF FOLIC ACID FORTIFICATION, BY GENDER

| | Post-secondary education enrollment | | | | Working full-time, $19 \leq$ Age $\leq 22$ | | | |
|---|---|---|---|---|---|---|---|---|
| | Full sample | | Nonmovers | | Full sample | | Nonmovers | |
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| | | | | Panel A: female | | | | |
| CNS anomaly rate × Post | 0.0084** | | 0.0135*** | | -0.0107** | | -0.0175*** | |
| | (0.0039) | | (0.0035) | | (0.0041) | | (0.0062) | |
| Top 25 × Post | | 0.0106** | | 0.0178*** | | -0.0210*** | | -0.0279*** |
| | | (0.0041) | | (0.0043) | | (0.0060) | | (0.0100) |
| Observations | 697,675 | 697,675 | 495,378 | 495,378 | 301,752 | 301,752 | 215,074 | 215,074 |
| $R^2$ | 0.1459 | 0.1460 | 0.1408 | 0.1408 | 0.0378 | 0.0379 | 0.0402 | 0.0403 |
| Dep. var. mean | 0.4032 | 0.4032 | 0.4001 | 0.4001 | 0.3164 | 0.3164 | 0.3141 | 0.3141 |
| | | | | Panel B: male | | | | |
| CNS anomaly rate × Post | 0.0055 | | 0.0062 | | -0.0054 | | -0.0078 | |
| | (0.0048) | | (0.0047) | | (0.0062) | | (0.0074) | |
| Top 25 × Post | | 0.0102** | | 0.0123** | | -0.0091 | | -0.0118 |
| | | (0.0050) | | (0.0052) | | (0.0068) | | (0.0088) |
| Observations | 742,846 | 742,846 | 532,035 | 532,035 | 315,513 | 315,513 | 223,958 | 223,958 |
| $R^2$ | 0.1172 | 0.1172 | 0.1169 | 0.1169 | 0.0365 | 0.0365 | 0.0412 | 0.0412 |
| Dep. var. mean | 0.3241 | 0.3241 | 0.3180 | 0.3180 | 0.4787 | 0.4787 | 0.4697 | 0.4697 |

Notes: Standard errors are clustered on state of birth. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors of continuous exposure specification are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, survey-year fixed effects, gender, race, Hispanic origin, Medicaid eligibility, exposure to mental health parity laws, welfare reforms, local unemployment rates, and county-level baseline characteristics interacted with time trends.

TABLE B5: HETEROGENEITY OF LONG RUN EFFECTS OF FOLIC ACID FORTIFICATION, BY RACE AND HISPANIC ORIGIN

| | Post-secondary education enrollment | | | | Working full-time, $19 \leq$ Age $\leq 22$ | | | |
|---|---|---|---|---|---|---|---|---|
| | Full sample | | Nonmovers | | Full sample | | Nonmovers | |
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| | | | | Panel A: white | | | | |
| CNS anomaly rate × Post | 0.0045 | | 0.0066 | | -0.0106** | | -0.0171*** | |
| | (0.0049) | | (0.0046) | | (0.0049) | | (0.0057) | |
| Top 25 × Post | | 0.0098* | | 0.0132*** | | -0.0177*** | | -0.0238*** |
| | | (0.0050) | | (0.0049) | | (0.0041) | | (0.0049) |
| Observations | 1,013,479 | 1,013,479 | 710,631 | 710,631 | 452,605 | 452,605 | 319,023 | 319,023 |
| $R^2$ | 0.1456 | 0.1456 | 0.1423 | 0.1424 | 0.0674 | 0.0674 | 0.0690 | 0.0690 |
| Dep. var. mean | 0.3739 | 0.3739 | 0.3699 | 0.3699 | 0.3970 | 0.3970 | 0.3916 | 0.3916 |
| | | | | Panel B: non-white | | | | |
| CNS anomaly rate × Post | 0.0108** | | 0.0152*** | | -0.0020 | | 0.0000 | |
| | (0.0046) | | (0.0048) | | (0.0065) | | (0.0088) | |
| Top 25 × Post | | 0.0114** | | 0.0184*** | | -0.0066 | | -0.0041 |
| | | (0.0051) | | (0.0064) | | (0.0077) | | (0.0114) |
| Observations | 427,042 | 427,042 | 316,782 | 316,782 | 164,660 | 164,660 | 120,009 | 120,009 |
| $R^2$ | 0.1132 | 0.1132 | 0.1136 | 0.1136 | 0.0524 | 0.0524 | 0.0540 | 0.0540 |
| Dep. var. mean | 0.3399 | 0.3399 | 0.3352 | 0.3352 | 0.4051 | 0.4051 | 0.3977 | 0.3977 |
| | | | | Panel C: Hispanic | | | | |
| CNS anomaly rate × Post | 0.0041 | | 0.0055 | | -0.0022 | | -0.0047 | |
| | (0.0040) | | (0.0051) | | (0.0088) | | (0.0117) | |
| Top 25 × Post | | 0.0102* | | 0.0107 | | -0.0119 | | -0.0176 |
| | | (0.0057) | | (0.0069) | | (0.0105) | | (0.0145) |
| Observations | 250,217 | 250,217 | 195,317 | 195,317 | 104,174 | 104,174 | 80,452 | 80,452 |
| $R^2$ | 0.0987 | 0.0987 | 0.1024 | 0.1024 | 0.0617 | 0.0617 | 0.0636 | 0.0636 |
| Dep. var. mean | 0.3398 | 0.3398 | 0.3477 | 0.3477 | 0.4353 | 0.4353 | 0.4252 | 0.4252 |
| | | | | Panel D: non-Hispanic | | | | |
| CNS anomaly rate × Post | 0.0086 | | 0.0115** | | -0.0095* | | -0.0150** | |
| | (0.0056) | | (0.0046) | | (0.0050) | | (0.0058) | |
| Top 25 × Post | | 0.0125** | | 0.0173*** | | -0.0165*** | | -0.0217*** |
| | | (0.0051) | | (0.0045) | | (0.0047) | | (0.0065) |
| Observations | 1,190,304 | 1,190,304 | 832,096 | 832,096 | 513,091 | 513,091 | 358,580 | 358,580 |
| $R^2$ | 0.1466 | 0.1466 | 0.1445 | 0.1445 | 0.0615 | 0.0615 | 0.0629 | 0.0630 |
| Dep. var. mean | 0.3681 | 0.3681 | 0.3607 | 0.3607 | 0.3911 | 0.3911 | 0.3853 | 0.3853 |

Notes: Standard errors are clustered on state of birth. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors of continuous exposure specification are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, survey-year fixed effects, gender, race, Hispanic origin, Medicaid eligibility, exposure to mental health parity laws, welfare reforms, local unemployment rates, and county-level baseline characteristics interacted with time trends.

TABLE B6: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION, CONTROLLING FOR CENSUS DIVISION SPECIFIC TREND

| | Post-secondary education enrollment | | | | Working full-time, $19 \leq$ Age $\leq 22$ | | | |
|---|---|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| | | | | Panel A: full sample | | | | |
| CNS anomaly rate × Post | 0.0013 | | | | -0.0068 | | | |
| | (0.0040) | | | | (0.0050) | | | |
| High CNS anomaly × Post | | 0.0046 | 0.0056 | 0.0060 | | -0.0128*** | -0.0117** | -0.0106** |
| | | (0.0051) | (0.0051) | (0.0051) | | (0.0045) | (0.0046) | (0.0045) |
| Observations | 1,314,116 | 1,314,116 | 1,314,116 | 1,314,116 | 564,360 | 564,360 | 564,360 | 564,360 |
| $R^2$ | 0.1333 | 0.1333 | 0.1333 | 0.1333 | 0.0614 | 0.0614 | 0.0614 | 0.0614 |
| Dep. var. mean | 0.3598 | 0.3598 | 0.3598 | 0.3598 | 0.4030 | 0.4030 | 0.4030 | 0.4030 |
| | | | | Panel B: nonmovers | | | | |
| CNS anomaly rate × Post | 0.0057 | | | | -0.0153** | | | |
| | (0.0041) | | | | (0.0069) | | | |
| High CNS anomaly × Post | | 0.0102* | 0.0106* | 0.0113 * | | -0.0214*** | -0.0215*** | -0.0211*** |
| | | (0.0059) | (0.0057) | (0.0058) | | (0.0061) | (0.0061) | (0.0062) |
| Observations | 938,092 | 938,092 | 938,092 | 938,092 | 401,793 | 401,793 | 401,793 | 401,793 |
| $R^2$ | 0.1315 | 0.1315 | 0.1315 | 0.1315 | 0.0626 | 0.0626 | 0.0626 | 0.0626 |
| Dep. var. mean | 0.3551 | 0.3551 | 0.3551 | 0.3551 | 0.3974 | 0.3974 | 0.3974 | 0.3974 |

Notes: Standard errors are clustered on state of birth. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors of continuous exposure specification are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, survey-year fixed effects, gender, race, Hispanic origin, Medicaid eligibility, exposure to mental health parity laws, welfare reforms, local unemployment rates, and county-level baseline characteristics interacted with time trends.

TABLE B7: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON EDUCATIONAL OUTCOMES OF YOUNG ADULTS, CONTROLLING FOR STATE OF RESIDENCE FIXED EFFECTS

| | Post-secondary education enrollment | | | | Working full-time, $19 \leq$ Age $\leq 22$ | | | |
|---|---|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| | | | | Panel A: full sample | | | | |
| CNS anomaly rate × Post | 0.0070* | | | | -0.0082* | | | |
| | (0.0040) | | | | (0.0041) | | | |
| High CNS anomaly × Post | | 0.0096** | 0.0103** | 0.0116** | | -0.0155*** | -0.0152*** | -0.0153*** |
| | | (0.0038) | (0.0040) | (0.0048) | | (0.0040) | (0.0040) | (0.0045) |
| Observations | 1,440,521 | 1,440,521 | 1,440,521 | 1,440,521 | 617,265 | 617,265 | 617,265 | 617,265 |
| $R^2$ | 0.1385 | 0.1385 | 0.1385 | 0.1385 | 0.0645 | 0.0645 | 0.0645 | 0.0645 |
| Dep. var. mean | 0.3627 | 0.3627 | 0.3627 | 0.3627 | 0.3995 | 0.3995 | 0.3995 | 0.3995 |
| | | | | Panel B: nonmovers | | | | |
| CNS anomaly rate × Post | 0.0097*** | | | | -0.0125** | | | |
| | (0.0035) | | | | (0.0049) | | | |
| High CNS anomaly × Post | | 0.0143*** | 0.0150*** | 0.0144*** | | -0.0195*** | -0.0198*** | -0.0236*** |
| | | (0.0038) | (0.0039) | (0.0048) | | (0.0057) | (0.0059) | (0.0064) |
| Observations | 938,092 | 938,092 | 938,092 | 938,092 | 401,793 | 401,793 | 401,793 | 401,793 |
| $R^2$ | 0.1315 | 0.1315 | 0.1315 | 0.1315 | 0.0626 | 0.0626 | 0.0626 | 0.0626 |
| Dep. var. mean | 0.3551 | 0.3551 | 0.3551 | 0.3551 | 0.3974 | 0.3974 | 0.3974 | 0.3974 |

Notes: Standard errors are clustered on state of birth. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors of continuous exposure specification are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, survey-year fixed effects, gender, race, Hispanic origin, Medicaid eligibility, exposure to mental health parity laws, welfare reforms, local unemployment rates, and county-level baseline characteristics interacted with time trends.

TABLE B8: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON EDUCATIONAL OUTCOMES OF YOUNG ADULTS, ACCOUNTING FOR MEAN REVERSION

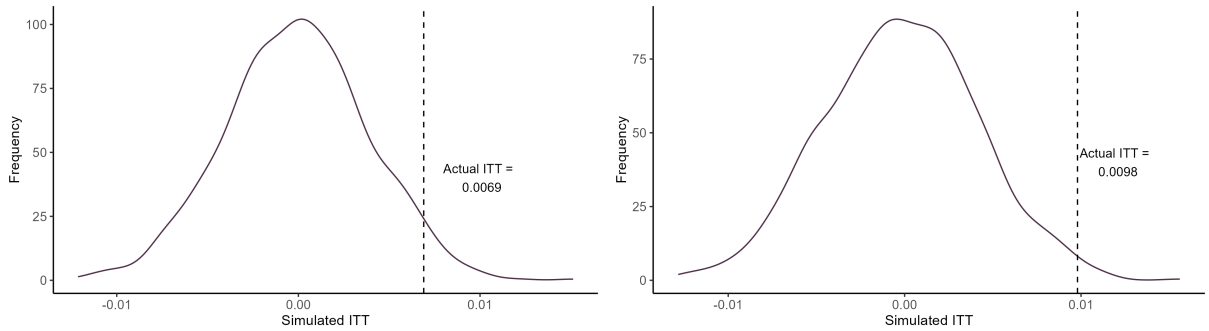
| | Post-secondary education enrollment | | | | Working full-time, $19 \leq$ Age $\leq 22$ | | | |
|---|---|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| | | | | Panel A: full sample | | | | |
| CNS anomaly rate × Post | 0.0070* | | | | -0.0070 | | | |
| | (0.0038) | | | | (0.0044) | | | |
| High CNS anomaly × Post | | 0.0094** | 0.0100** | 0.0115** | | -0.0143*** | -0.0140*** | -0.0138*** |
| | | (0.0038) | (0.0039) | (0.0048) | | (0.0042) | (0.0043) | (0.0049) |
| Observations | 1,440,521 | 1,440,521 | 1,440,521 | 1,440,521 | 617,265 | 617,265 | 617,265 | 617,265 |
| $R^2$ | 0.1385 | 0.1385 | 0.1385 | 0.1385 | 0.0645 | 0.0645 | 0.0645 | 0.0645 |
| Dep. var. mean | 0.3627 | 0.3627 | 0.3627 | 0.3627 | 0.3995 | 0.3995 | 0.3995 | 0.3995 |
| | | | | Panel B: nonmovers | | | | |
| CNS anomaly rate × Post | 0.0104*** | | | | -0.0103* | | | |
| | (0.0034) | | | | (0.0054) | | | |
| High CNS anomaly × Post | | 0.0148*** | 0.0153*** | 0.0150*** | | -0.0171** | -0.0176*** | -0.0212*** |
| | | (0.0037) | (0.0038) | (0.0048) | | (0.0064) | (0.0064) | (0.0067) |
| Observations | 1,027,413 | 1,027,413 | 1,027,413 | 1,027,413 | 439,032 | 439,032 | 439,032 | 439,032 |
| $R^2$ | 0.1341 | 0.1341 | 0.1341 | 0.1341 | 0.0628 | 0.0628 | 0.0628 | 0.0628 |
| Dep. var. mean | 0.3580 | 0.3580 | 0.3580 | 0.3580 | 0.3935 | 0.3935 | 0.3935 | 0.3935 |

Notes: Standard errors are clustered on state of birth. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors of continuous exposure specification are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, survey-year fixed effects, gender, race, Hispanic origin, Medicaid eligibility, exposure to mental health parity laws, welfare reforms, local unemployment rates, and county-level baseline characteristics interacted with time trends.

TABLE B9: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON EDUCATIONAL OUTCOMES OF YOUNG ADULTS, PLACEBO TEST USING COHORTS BORN IN 1983-1992

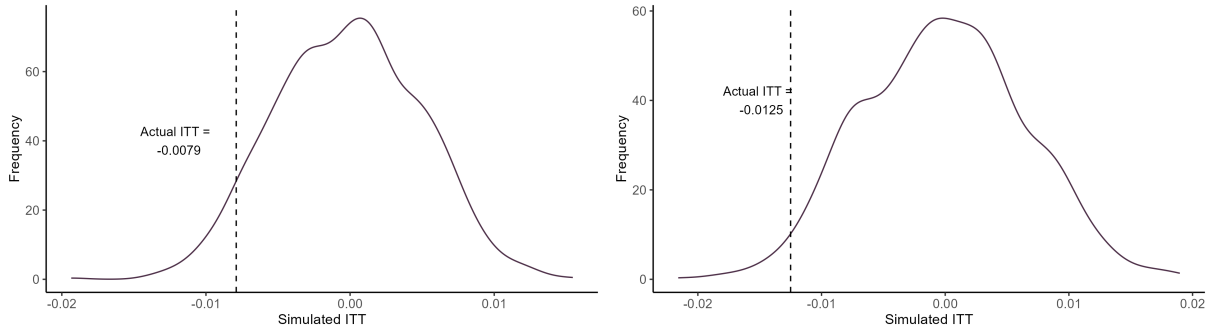
| | Post-secondary education enrollment | | | | Working full-time, 19 ≤ Age ≤ 22 | | | |
|---|---|---|---|---|---|---|---|---|
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| | | | | Panel A: full sample | | | | |
| CNS anomaly rate × Post | -0.0025 | | | | – | | | |
| | (0.0037) | | | | – | | | |
| High CNS anomaly × Post | | -0.0042 | -0.0049 | -0.0012 | | – | – | – |
| | | (0.0043) | (0.0043) | (0.0047) | | – | – | – |
| Observations | 1,553,624 | 1,553,624 | 1,553,624 | 1,553,624 | 267,338 | 267,338 | 267,338 | 267,338 |
| $R^2$ | 0.1013 | 0.1013 | 0.1013 | 0.1013 | 0.0547 | 0.0547 | 0.0547 | 0.0547 |
| Dep. var. mean | 0.2559 | 0.2559 | 0.2559 | 0.2559 | 0.3719 | 0.3719 | 0.3719 | 0.3719 |
| | | | | Panel B: nonmovers | | | | |
| CNS anomaly rate × Post | -0.0017 | | | | – | | | |
| | (0.0040) | | | | – | | | |
| High CNS anomaly × Post | | -0.0039 | -0.0051 | 0.0003 | | – | – | – |
| | | (0.0050) | (0.0050) | (0.0052) | | – | – | – |
| Observations | 1,096,269 | 1,096,269 | 1,096,269 | 1,096,269 | 191,687 | 191,687 | 191,687 | 191,687 |
| $R^2$ | 0.1067 | 0.1067 | 0.1067 | 0.1067 | 0.0561 | 0.0561 | 0.0561 | 0.0561 |
| Dep. var. mean | 0.2508 | 0.2508 | 0.2508 | 0.2508 | 0.3610 | 0.3610 | 0.3610 | 0.3610 |

Notes: Standard errors are clustered on state of birth. ***, **, and * indicate that the estimates are significant at the 1%, 5%, and 10% levels. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors of continuous exposure specification are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, survey-year fixed effects, gender, race, Hispanic origin, Medicaid eligibility, exposure to mental health parity laws, welfare reforms, local unemployment rates, and county-level baseline characteristics interacted with time trends.




(A) All post-secondary education enrollment, full sample

(B) All post-secondary education enrollment, nonmovers




(C) Working full-time, $19 \leq$ age $\leq 22$, full sample

(D) Working full-time, $19 \leq$ age $\leq 22$, nonmovers

FIGURE B6: RANDOMIZATION TEST

Notes: Standard errors are clustered on state of birth. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (5.6 cases per 10,000 births). Percentiles are weighted by number of births. Controls and other fixed effects include state-by-year share of Medicaid-eligible pregnant women, ACDF and TANF waiver dummies, state mental health parity law implementation dummy, race fixed effect, survey-year fixed effects, Hispanic origin, gender, Bartik-style change in state unemployment rate at birth, and state-level pre-intervention characteristics interacted with linear time trend.