

Long Run Effects of Folic Acid Fortification*

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Abstract

The effectiveness of existing interventions aimed at improving public health through the provision of healthier foods is often limited by the challenge of changing consumer behavior. Food fortification offers a promising alternative by enhancing the nutritional content of widely consumed foods without requiring changes in consumer habits. This is particularly relevant in addressing micronutrient deficiencies, where fortification can serve as a scalable and low-cost solution. This paper studies the most recent food fortification effort in the U.S.—the folic acid fortification of enriched grain products in the late 1990s. This policy substantially increased folate intake and reduced the incidence of birth defects. By comparing cohorts exposed and unexposed to the fortification across regions with varying baseline folate deficiency levels, I find that (1) in the short run, fortification increased the share of births among disadvantaged mothers, likely due to improved fetal survival rates; and (2) in the long run, in-utero exposure to folic acid fortification resulted in greater investments in human capital, evidenced by an increased likelihood of enrolling in post-secondary education and a decline in the labor supply of young adults. My findings suggest that food fortification has significant potential to improve dietary and health outcomes and to generate broad, long-lasting benefits for children.

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1 Introduction

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 enriched grain products in the U.S. in the late 1990s and its impacts on health and 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 40 μ g/100g of folic acid (synthetic 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. This paper investigates the effects of folic acid fortification in utero on children's human capital outcomes.

I leverage geographical variation in pre-fortification birth defects tied to folate deficiency and the timing of folic acid fortification of enriched 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. My main results indicate that in-utero exposure to folic acid fortification led to higher likelihood of enrolling in post-secondary education in young adulthood.

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, 2022a; 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 countries¹, causal evidence from the U.S. can inform and motivate broader adoption of this policy.

This paper also 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, 2022b) and Ramadan fasting (Almond and Mazumder, 2011; Almond, Mazumder and Van Ewijk, 2015; Majid, 2015; Greve, Schultz-Nielsen and Tekin, 2017) leading to poorer

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

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 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., 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.

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 analyzes robustness and sensitivity of results; Section 8 discusses magnitude of estimates and policy cost-effectiveness; and, finally, Section 9 concludes.

2 Background

2.1 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 μg for women aged 15 to 49 from 1988 to 1994, which is far below 400 μg , the recommended folate intake for pregnant women from the United States Public Health 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 guidance², 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.

2.2 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 anencephaly³ are typically fatal, with most affected infants dying before or shortly after birth. Mild NTDs, like spina bifida⁴ 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.

²See <https://www.cdc.gov/ncbddd/folicacid/recommendations.html> (accessed on 05/20/2022) for reference.

³Infants with anencephaly are born without parts of the skull and brain.

⁴The 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.

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 enriched 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, 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 foods⁵ 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).

3 Conceptual Framework

In this section, I propose a simple model and draw on evidence from existing literature to conceptualize the potential connection between folic acid fortification and long-term educational outcomes. In Section 8, I also use this framework to interpret the regression coefficients and estimate the effect of in-utero exposure to folic acid fortification on cognitive ability.

⁵Standardized foods have a standard of identify, such as enriched grain products.



FIGURE 1: CHIPS WITH ENRICHED WHEAT FLOUR AS AN INGREDIENT

3.1 A simple framework of time allocation between leisure, work, and school

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. The utility function is:

$$U(L_g, C_g) = \eta_g \log(L_g) + (1 - \eta_g) \log(C_g),$$

where $\eta_g \in (0, 1)$ represents the relative importance of leisure versus consumption in the individual's preferences.

For simplicity, and 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 θ 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. Thus, the problem can be

written as:

$$\max_{L,W,S} U(L,C) = \eta \log(L) + (1 - \eta) \log(w \cdot W),$$

subject to: $w = w_0 + \theta S,$

$$T = L + W + S.$$

Solve the utility maximization problem (see details in Section A) we can get optimal time allocated to school is:

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

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 (θ) and decreases with the base wage (w_0).

3.2 Folic acid fortification, cognitive ability, and return to education

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 (θ). 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 measured 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 Survey⁶, 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).

Nativity 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 cause of NTDs including spina bifida 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 anal-

⁶Other surveys, such as the Early Childhood Longitudinal Study, Kindergarten Class of 2011 (ECLS-K 2011) and the National Longitudinal Survey 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.

ysis, 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).

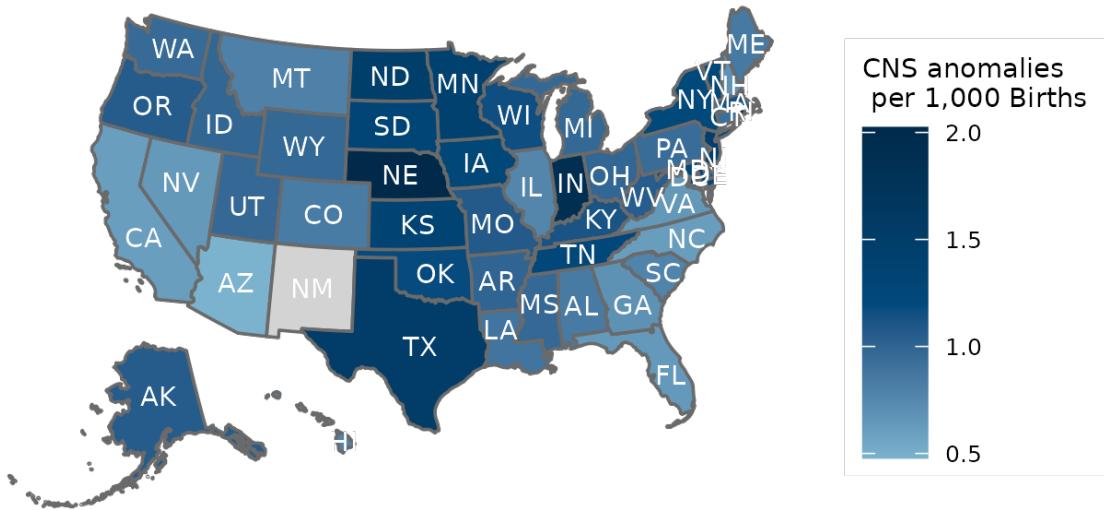


FIGURE 2: PRE-EXISTING CNS ANOMALY RATES BY STATE

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

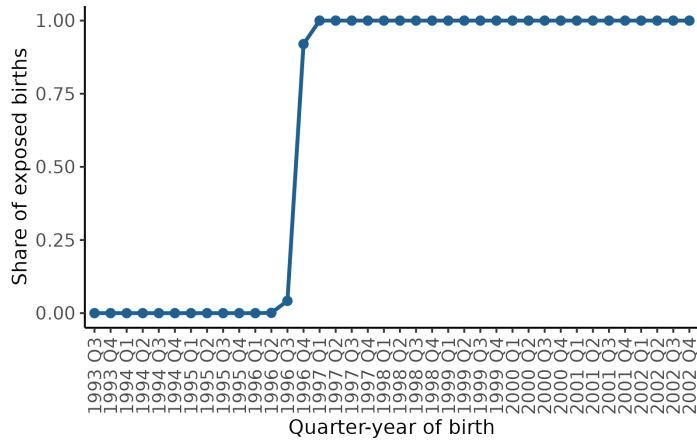


FIGURE 3: SHARE OF EXPOSED BIRTHS BY QUARTER-AND-YEAR-OF-BIRTH

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

4.2 National Assessment of Educational Progress

The test score data is from the National Assessment of Educational Progress (NAEP). The NAEP provides standardized test scores in mathematics and reading for 4th, 8th, and 12th graders, allowing for consistent comparisons across cohorts and states. I am currently still in the process of applying for the restricted-use version of NAEP.

4.3 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 pandemic⁷. 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,

⁷Response 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.

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 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 enriched 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, Thuiliez 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 , $\text{CNS anomaly rate}_s$ 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, μ_s is state-of-birth fixed effects to account for cohort-invariant unobserved heterogeneity, λ_t is quarter-and-year-of-birth fixed effects to control for cohort-specific shocks, C_{ist} is a set of control variables, and ε_{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:

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

I define year of effective exposure γ 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.

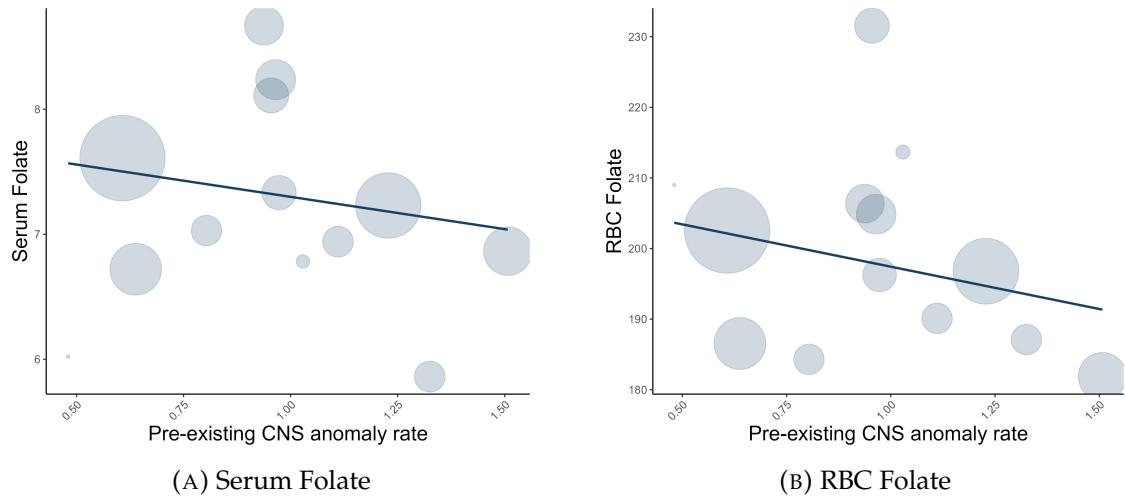


FIGURE 4: CORRELATION BETWEEN PRE-EXISTING CNS ANOMALY RATE AND BIOMARKERS OF FOLATE DEFICIENCY AT STATE LEVEL

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), B2).

For CNS anomaly rate_s in Equation 1, I report results using both continuous CNS

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

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, 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 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 before and 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 before and 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 $400 \mu\text{g}$ 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 before and 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

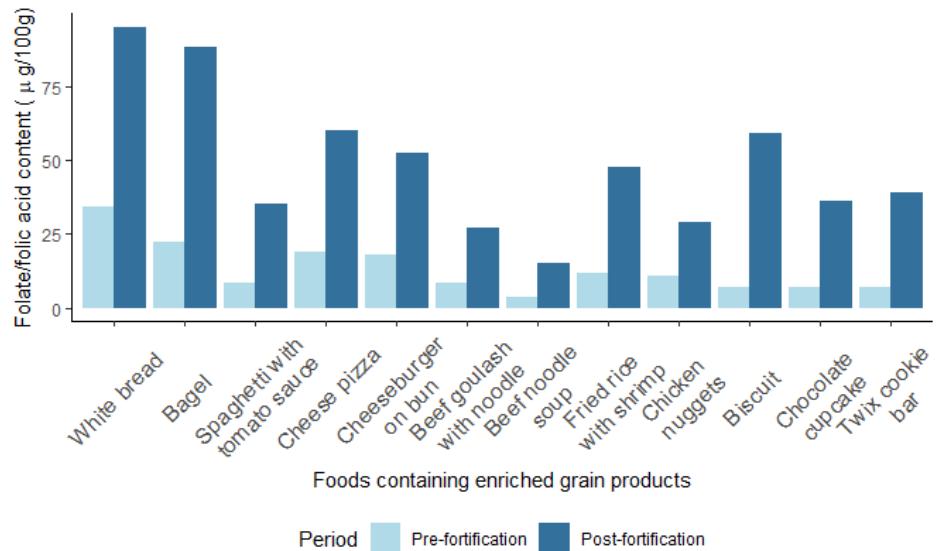


FIGURE 5: CHANGES IN FOLATE CONTENTS IN SELECTED FOODS DUE TO FORTIFICATION

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.

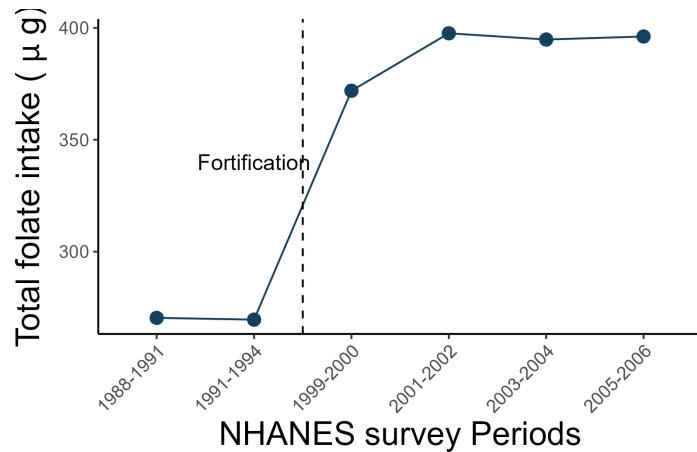


FIGURE 6: DIETARY FOLATE CONCENTRATIONS BEFORE AND AFTER FORTIFICATION

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

improvement in folate absorption. Blood folate measurement remain unchanged from 1998 to 2006 (Pfeiffer et al., 2012).

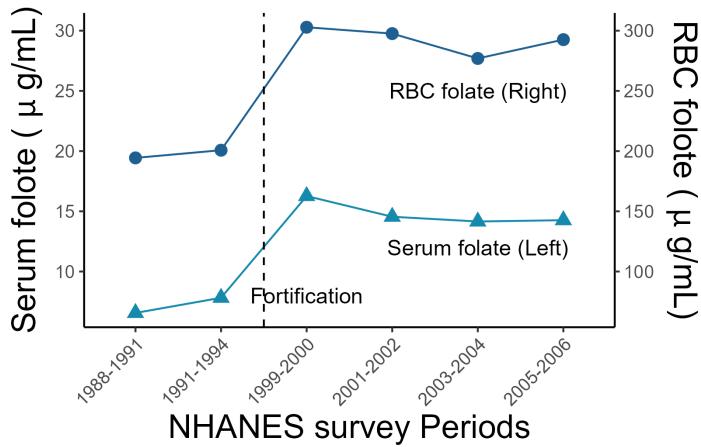


FIGURE 7: BLOOD FOLATE CONCENTRATIONS BEFORE AND AFTER FORTIFICATIONN

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.

6.4 Congenital anomalies before and 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 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.

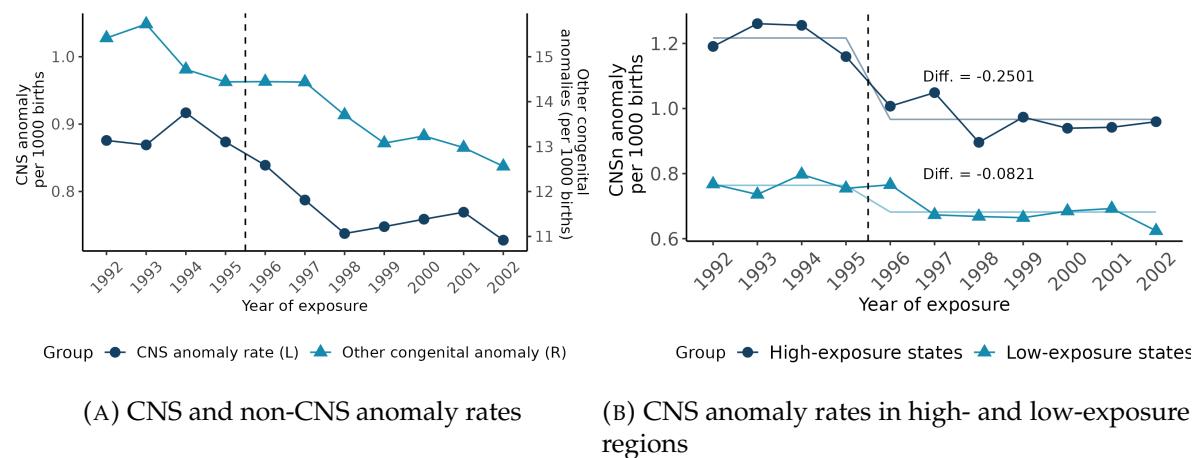


FIGURE 8: TRENDS IN CONGENITAL ANOMALY RATES

Notes: The unit of CNS Anomaly rate is cases per 1,000 births. High exposure is defined with top 30% pre-existing CNS anomaly rate.

6.5 Effects of folic acid fortification on birth outcomes

Fifth, I find folic acid fortification increases share of births given by disadvantaged mothers. Specially, 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 (Table 2). 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 B3 and Figure B2).

TABLE 2: EFFECTS OF FOLIC ACID FORTIFICATION ON MATERNAL CHARACTERISTICS

| | Share of mothers with following characteristics | | | | | |
|-------------------------|---|---------------------|-----------------------|--------------------------|--------------------|---------------------|
| | Age ≤ 22 | Education < college | Unmarried | Inadequate prenatal care | Non-white | Hispanic |
| | (1) | (2) | (3) | (4) | (5) | (6) |
| CNS anomaly rate × Post | 0.0041*** (0.0013) | 0.0058* (0.0030) | 0.0086*** (0.0025) | 0.0139** (0.0058) | 0.0014 (0.0025) | -0.0012 (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.

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, potentially 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.

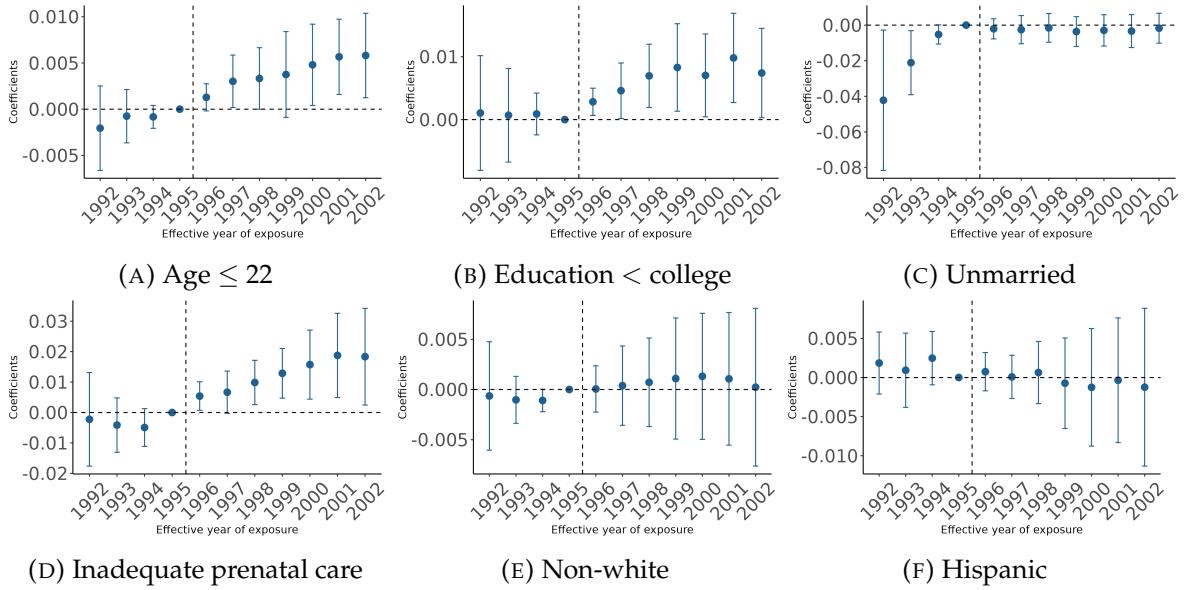


FIGURE 9: DYNAMIC EFFECTS OF FOLIC ACID FORTIFICATION ON MATERNAL CHARACTERISTICS, CONTINUOUS EXPOSURE

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.

6.6 Effects of folic acid fortification on test scores (in progress)

Sixth, I plan to investigate the effects of in-utero exposure to folic acid fortification on children's or young adults' test scores, utilizing data from the National Assessment of Educational Progress (NAEP). The NAEP provides standardized test scores in mathematics and reading for 4th, 8th, and 12th graders, allowing for consistent comparisons across cohorts and states. This analysis will require access to restricted-use version of NAEP, and I am currently in the process of applying for the necessary data access.

To estimate the impact of early-life fortification exposure, I use the state of schooling as a proxy for the state of birth, adjusting for mobility patterns, given the relatively low migration rates at younger ages. Adding results on test scores in primary and secondary schools provides a more holistic understanding of how improved nutritional status in utero affects human capital development at different life stages.

6.7 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.

6.7.1 Educational outcomes

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 30%), 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.

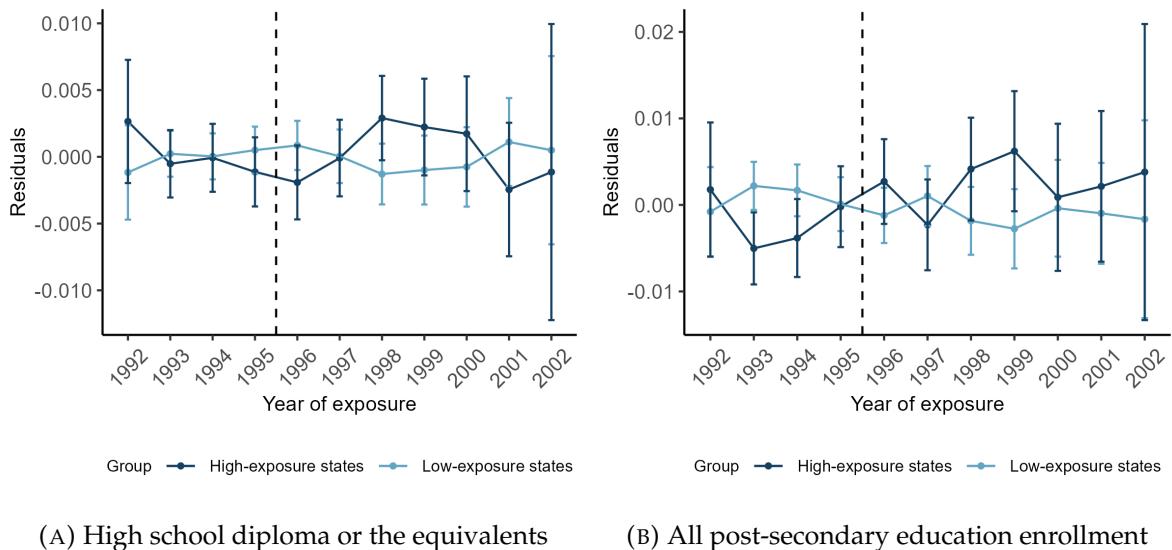


FIGURE 10: COHORT TRENDS IN RESIDUAL EDUCATIONAL OUTCOMES OF YOUNG ADULTS

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 30% pre-existing CNS anomaly rate; low-exposure is defined otherwise. The average and standard errors are weighted by individual sample weight.

Table 3 presents the difference-in-difference estimates of effects of in-utero exposure to folic acid fortification on young adults' educational outcomes. Columns (1) and (5) indicate

that in-utero exposure to folic acid fortification does not affect probability of young adults earning high school diploma or equivalent credentials, which 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.

Columns (2) and (6) show that in-utero exposure to folic acid fortification increases the likelihood of 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.

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, Columns (3), (4), (5), and (6) show 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.

Figure 11 presents the dynamic effects of in-utero exposure to folic acid fortification on young adults' educational outcomes. 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. In terms of post-secondary education enrollment (Figure 11b) and college enrollment among 19- to 22-year-olds (Figure 11c), the cohort-specific coefficients shift upward immediately following folic acid fortification, with this pattern being more pronounced among non-movers. Figure 11d shows that 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 are noisier due to the

TABLE 3: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON EDUCATIONAL OUTCOMES OF YOUNG ADULTS

| | Full sample | | | | Nonmovers | | | |
|-------------------------|--|--------------------------------------|---------------------|---|--|--------------------------------------|----------------------|---|
| | High school diploma or secondary education equivalents | All post- secondary enrollment | College 22 | Grad. or prof. school enrollment, age > 22 | High school diploma or secondary education equivalents | All post- secondary enrollment | College 22 | Grad. or prof. school enrollment, age > 22 |
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| CNS anomaly rate × Post | -0.0001 (0.0015) | 0.0069* (0.0040) | 0.0088* (0.0045) | 0.0039*** (0.0013) | -0.0012 (0.0014) | 0.0098*** (0.0035) | 0.0127** (0.0048) | 0.0040** (0.0020) |
| Observations | 1,440,521 | 1,440,521 | 807,669 | 632,852 | 1,027,413 | 1,027,413 | 581,820 | 445,593 |
| R ² | 0.0123 | 0.1363 | 0.0623 | 0.0110 | 0.0134 | 0.1341 | 0.0598 | 0.0116 |
| Dep. var. mean | 0.9307 | 0.3627 | 0.4927 | 0.0608 | 0.9253 | 0.3580 | 0.4821 | 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.

smaller sample size for each birth cohort.

In Table 4, I use a similar specification as in Table 3, but replace the continuous pre-existing CNS anomaly rates with a binary indicator for high-exposure regions. I define high-exposure regions as states in the top 40%, 30%, and 20% of the birth-weighted distribution of pre-existing CNS anomaly rates. The results in Table 4 are consistent with previous findings but are easier to interpret. Depending on the cutoffs used to categorize high- and low-exposure regions, young adults exposed to folic acid fortification in utero and born in high-exposure states show no significant change in the probability of earning a high school diploma or equivalent credentials. However, they experience 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 the probability of enrolling in college for those aged 19–22, and a 0.48 to 0.51 percentage point increase in the probability of enrolling in graduate or professional schools for those over 22. Among non-movers, I observe similar patterns but generally larger increases in probability of post-secondary education enrollment: nonmover 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 the probability of enrolling in college for those aged 19–22, and a 0.40 to 0.62 percentage point increase in the probability of enrolling in graduate or professional schools for those over 22.

Figure 12 presents the dynamic effect estimates using binary treatment variables for the full sample. The results are, once again, consistent with previous findings. Under these

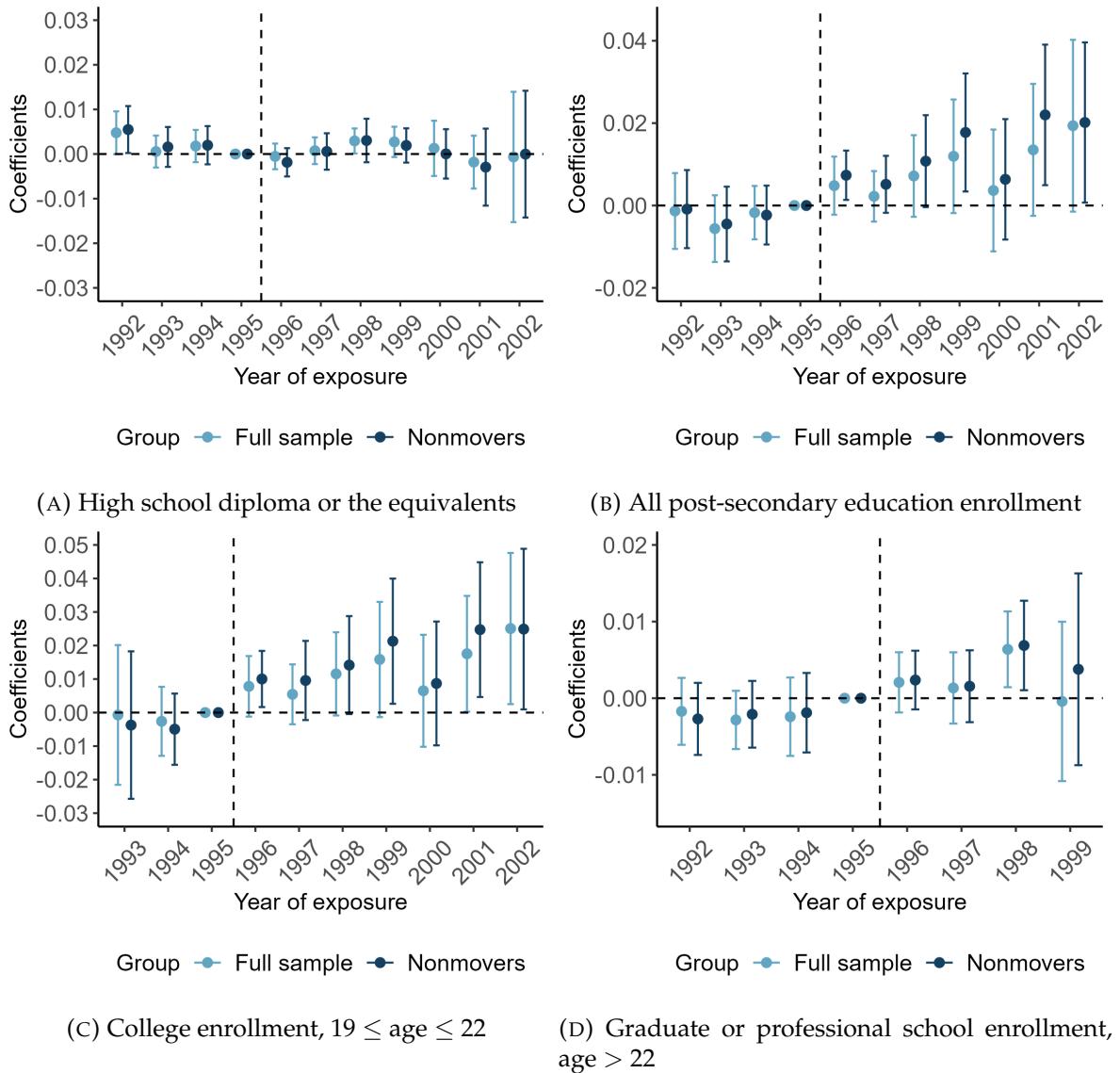


FIGURE 11: DYNAMIC EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON EDUCATIONAL OUTCOMES OF YOUNG ADULTS

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.

specifications, I also do not observe any significant pre-fortification differential trends in outcomes, further supporting the validity of my earlier conclusions. The dynamic effects for non-movers similarly align with the earlier results, as shown in Figure B4.

TABLE 4: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON EDUCATIONAL OUTCOMES OF YOUNG ADULTS, HIGH- VERSUS LOW-EXPOSURE REGIONS

| | Full sample | | | | Nonmovers | | | |
|----------------|---|-------------------------------|-----------------------|--------------------------|---|-------------------------------|-----------------------|--------------------------|
| | HS or the equiva- lents (1) | All post- secondary (2) | College (3) | Grad. or Prof. (4) | HS or the equiva- lents (5) | All post- secondary (6) | College (7) | Grad. or Prof. (8) |
| Top 40 × Post | 0.0016 (0.0013) | 0.0077** (0.0034) | 0.0136*** (0.0047) | 0.0051*** (0.0017) | 0.0010 (0.0017) | 0.0104*** (0.0034) | 0.0168*** (0.0056) | 0.0062*** (0.0022) |
| Observations | 1,440,521 | 1,440,521 | 807,669 | 632,852 | 1,027,413 | 1,027,413 | 581,820 | 445,593 |
| R ² | 0.0123 | 0.1363 | 0.0623 | 0.0110 | 0.0134 | 0.1341 | 0.0598 | 0.0116 |
| Dep. var. mean | 0.9307 | 0.3627 | 0.4927 | 0.0608 | 0.9253 | 0.3580 | 0.4821 | 0.0525 |
| Top 30 × Post | 0.0012 (0.0013) | 0.0095** (0.0039) | 0.0148*** (0.0049) | 0.0048*** (0.0016) | 0.0005 (0.0015) | 0.0143*** (0.0038) | 0.0217*** (0.0055) | 0.0055** (0.0024) |
| Observations | 1,440,521 | 1,440,521 | 807,669 | 632,852 | 1,027,413 | 1,027,413 | 581,820 | 445,593 |
| R ² | 0.0123 | 0.1363 | 0.0623 | 0.0110 | 0.0134 | 0.1341 | 0.0599 | 0.0116 |
| Dep. var. mean | 0.9307 | 0.3627 | 0.4927 | 0.0608 | 0.9253 | 0.3580 | 0.4821 | 0.0525 |
| Top 20 × Post | 0.0012 (0.0014) | 0.0117** (0.0050) | 0.0111** (0.0049) | 0.0049** (0.0020) | 0.0002 (0.0016) | 0.0144*** (0.0048) | 0.0161*** (0.0052) | 0.0040* (0.0024) |
| Observations | 1,440,521 | 1,440,521 | 807,669 | 632,852 | 1,027,413 | 1,027,413 | 581,820 | 445,593 |
| R ² | 0.0123 | 0.1363 | 0.0623 | 0.0110 | 0.0134 | 0.1341 | 0.0598 | 0.0116 |
| Dep. var. mean | 0.9307 | 0.3627 | 0.4927 | 0.0608 | 0.9253 | 0.3580 | 0.4821 | 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. 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.

6.7.2 Labor supply

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 5 shows that, 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 usual hours worked per week by 0.0712 to 0.3981 hours, a reduction in the probability of working full-time by 0.79 to 1.54 percentage points, and a decrease in

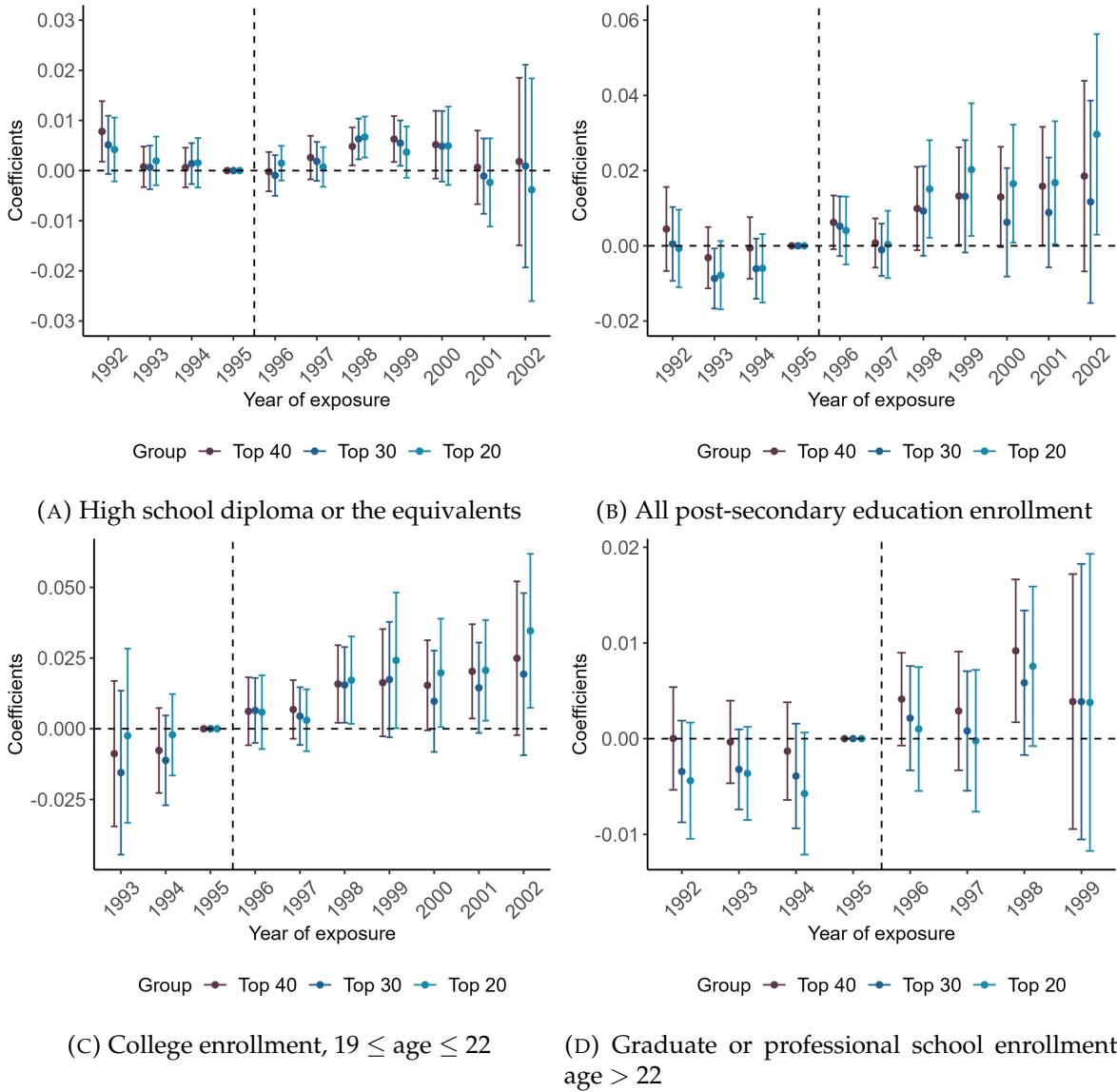


FIGURE 12: DYNAMIC EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON EDUCATIONAL OUTCOMES OF YOUNG ADULTS, HIGH- VERSUS LOW-EXPOSURE REGIONS, FULL SAMPLE

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.

annual earnings by 68.2 to 359.7 dollars. These declines are larger and more precise when stricter definitions of high-exposure regions are applied. Similar to the effects on school enrollment, the effects on labor supply are more pronounced among nonmovers: they experience a decline in usual hours worked per week by 0.2560 to 0.6798 hours, a reduction in the

probability of working full-time by 1.24 to 2.36 percentage points, and a decrease in annual earnings by 227.7 to 601.9 dollars.

TABLE 5: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON LABOR OUTCOMES OF YOUNG ADULTS AGED 19-22

| | Full sample | | | Nonmovers | | |
|-------------------------|------------------------------|--------------------------|---------------------------------|------------------------------|--------------------------|---------------------------------|
| | Hours worked per week (1) | Working full-time (2) | Annual earning (\$1,000) (3) | Hours worked per week (4) | Working full-time (5) | Annual earning (\$1,000) (6) |
| CNS anomaly rate × Post | -0.0712 (0.1247) | -0.0079* (0.0041) | -0.1034 (0.0961) | -0.2560 (0.1640) | -0.0125** (0.0049) | -0.3221** (0.1322) |
| Observations | 807,669 | 617,265 | 807,669 | 581,820 | 439,032 | 581,820 |
| R ² | 0.0551 | 0.0618 | 0.0566 | 0.0610 | 0.0628 | 0.0591 |
| Dep. var. mean | 23.5413 | 0.3995 | 11.0806 | 23.1146 | 0.3935 | 10.9597 |
| Top 40 × Post | -0.1537 (0.1443) | -0.0109** (0.0045) | -0.0682 (0.1206) | -0.3276* (0.1871) | -0.0124** (0.0055) | -0.2277 (0.1555) |
| Observations | 807,669 | 617,265 | 807,669 | 581,820 | 439,032 | 581,820 |
| R ² | 0.0551 | 0.0618 | 0.0566 | 0.0610 | 0.0628 | 0.0591 |
| Dep. var. mean | 23.5413 | 0.3995 | 11.0806 | 23.1146 | 0.3935 | 10.9597 |
| Top 30 × Post | -0.3019** (0.1238) | -0.0154*** (0.0040) | -0.2440* (0.1427) | -0.5064*** (0.1882) | -0.0195*** (0.0057) | -0.5112*** (0.1804) |
| Observations | 807,669 | 617,265 | 807,669 | 581,820 | 439,032 | 581,820 |
| R ² | 0.0551 | 0.0618 | 0.0566 | 0.0610 | 0.0628 | 0.0591 |
| Dep. var. mean | 23.5413 | 0.3995 | 11.0806 | 23.1146 | 0.3935 | 10.9597 |
| Top 20 × Post | -0.3981*** (0.1280) | -0.0149*** (0.0046) | -0.3597** (0.1642) | -0.6798*** (0.1978) | -0.0236*** (0.0064) | -0.6019*** (0.2136) |
| Observations | 807,669 | 617,265 | 807,669 | 581,820 | 439,032 | 581,820 |
| R ² | 0.0551 | 0.0618 | 0.0566 | 0.0610 | 0.0628 | 0.0591 |
| Dep. var. mean | 23.5413 | 0.3995 | 11.0806 | 23.1146 | 0.3935 | 10.9597 |

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 13 presents the dynamic effect estimates 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 5. 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.

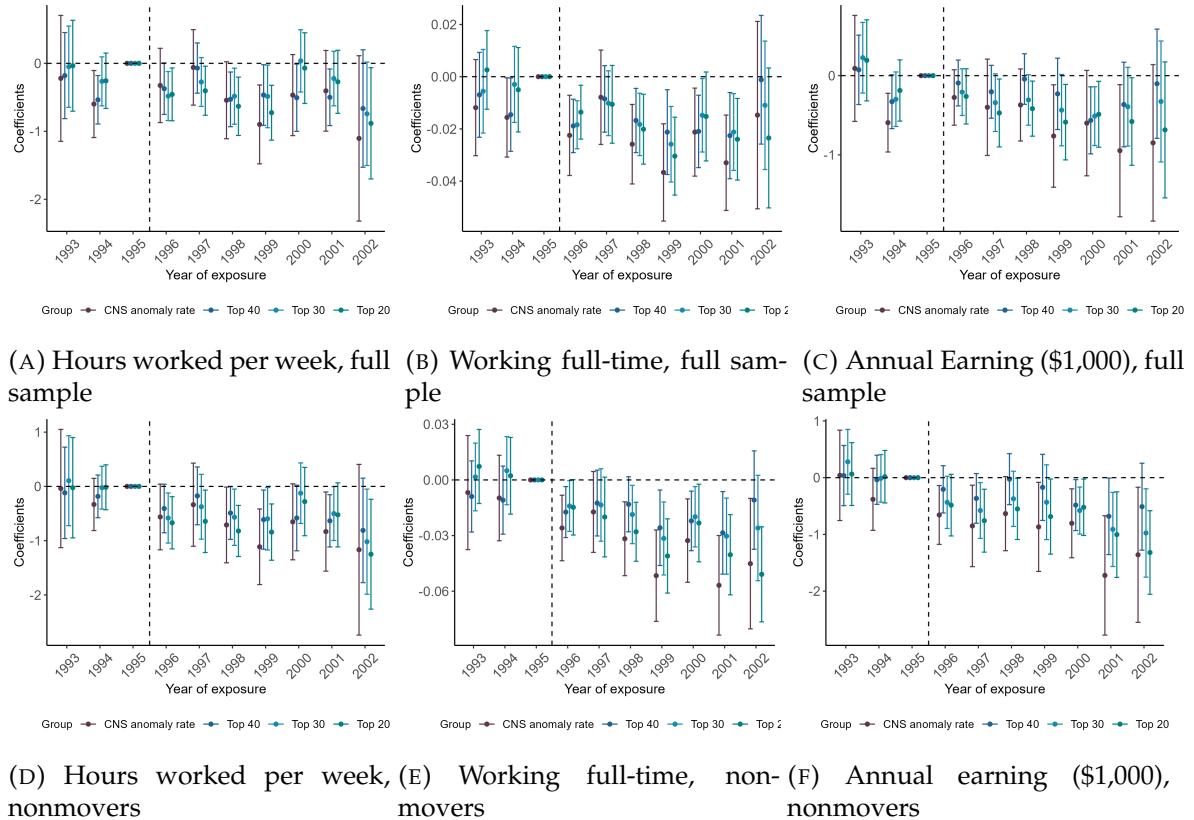


FIGURE 13: DYNAMIC EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON LABOR OUTCOMES OF YOUNG ADULTS BETWEEN 19-22 YEARS OLD

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, ACFD 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 6 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. The dynamic effect estimates are also in line with the difference-in-difference estimates, as shown in Figure B5.

6.7.3 Heterogeneity by gender, race, and ethnicity

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 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-

TABLE 6: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON LABOR OUTCOMES OF YOUNG ADULTS OVER 22

| | Full sample | | | Nonmovers | | |
|-------------------------|------------------------------|---------------------|----------------------|------------------------------|---------------------|---------------------|
| | Hours worked per week (1) | Full-time (2) | Earning (3) | Hours worked per week (4) | Full-time (5) | Earning (6) |
| CNS anomaly rate × Post | 0.0253 (0.1283) | -0.0023 (0.0032) | -0.2474 (0.2502) | 0.1006 (0.1412) | 1e-04 (0.0031) | 0.1157 (0.3022) |
| Observations | 632,852 | 533,035 | 632,852 | 445,593 | 370,806 | 445,593 |
| R ² | 0.0326 | 0.0411 | 0.0868 | 0.0351 | 0.0436 | 0.0873 |
| Dep. var. mean | 32.1876 | 0.6816 | 28.5998 | 31.4838 | 0.6702 | 26.9809 |
| Top 40 × Post | -0.0292 (0.1301) | -0.0063 (0.0039) | -0.4106* (0.2344) | -0.0131 (0.1373) | -0.0058 (0.0041) | -0.0660 (0.2730) |
| Observations | 632,852 | 533,035 | 632,852 | 445,593 | 370,806 | 445,593 |
| R ² | 0.0326 | 0.0411 | 0.0868 | 0.0351 | 0.0436 | 0.0873 |
| Dep. var. mean | 32.1876 | 0.6816 | 28.5998 | 31.4838 | 0.6702 | 26.9809 |
| Top 30 × Post | 0.0004 (0.1283) | -0.0042 (0.0038) | -0.1824 (0.2632) | 0.0846 (0.1377) | -0.0012 (0.0040) | 0.0622 (0.2993) |
| Observations | 632,852 | 533,035 | 632,852 | 445,593 | 370,806 | 445,593 |
| R ² | 0.0326 | 0.0411 | 0.0868 | 0.0351 | 0.0436 | 0.0873 |
| Dep. var. mean | 32.1876 | 0.6816 | 28.5998 | 31.4838 | 0.6702 | 26.9809 |
| Top 20 × Post | 0.0659 (0.1581) | 0.0002 (0.0047) | -0.1328 (0.3046) | 0.1125 (0.1593) | 0.0027 (0.0048) | 0.3109 (0.3601) |
| Observations | 632,852 | 533,035 | 632,852 | 445,593 | 370,806 | 445,593 |
| R ² | 0.0326 | 0.0411 | 0.0868 | 0.0351 | 0.0436 | 0.0873 |
| Dep. var. mean | 32.1876 | 0.6816 | 28.5998 | 31.4838 | 0.6702 | 26.9809 |

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.

exposure states (defined as states in the top 30% of the birth-weighted distribution of pre-existing CNS anomaly rates).

I do not have prior expectations regarding gender differences in the effects of in-utero exposure to folic acid fortification. Adhvaryu et al. (2020) finds that in-utero exposure to salt iodization has a greater effect on women's incomes compared to men's, possibly because

women have lower baseline incomes and therefore more potential for improvement. However, women consistently have higher baseline rates of post-secondary education enrollment across age groups. Given this, I do not expect to observe larger effects for women based on the same rationale. Tables B4 and B5 show that, in some cases, the estimates are larger for females, while in others, they are larger for males.

Table B6 shows that in-utero exposure to folic acid fortification has a greater impact on post-secondary education enrollment among non-white individuals. However, Table B7 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).

Tables B8 and B9 suggest 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 discusses the robustness of the earlier results. For convenience, I focus on the key findings related to post-secondary education enrollment for young adults, college enrollment for individuals aged 19 to 22, graduate/professional school enrollment for those over 22, and labor supply 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, defined as those in the top 30% of the birth-weighted distribution of pre-existing CNS anomaly rates. The latter results address the possibility that the continuous exposure measure may not fully capture actual pre-existing folate deficiency levels.

7.1 Additional controls

First, I test the robustness of my results by adding additional control variables. In Table B10, 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 B11, I add state-of-residence fixed effects to account for potential postnatal migration effects. Additionally, in Table B12, 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 B13, 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 actual 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, while this is less the case for the statistically insignificant 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 (β) 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 (θ). The coefficients from the regressions using continuous exposure can be expressed as follows:

$$\beta = \frac{\partial P(S = \tilde{\eta} \left(T - \frac{2w_0}{\theta} \right) > 0)}{\partial \theta} \cdot \frac{\partial \theta}{\partial \phi} \cdot \frac{\partial \phi}{\partial \kappa}, \quad (3)$$

where $P(S > 0)$ denotes the probability that $S > 0$, ϕ represents cognitive ability, and κ 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 β , 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 β in Equation 3 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 $\frac{\partial P(S=\bar{\eta}(T-\frac{2w_0}{\theta})>0)}{\partial \theta}$, 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: $\sqrt{(2.8 \times 0.0045)^2 + (2 \times 0.0013)^2} = 0.0129$. 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.

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 enriched 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

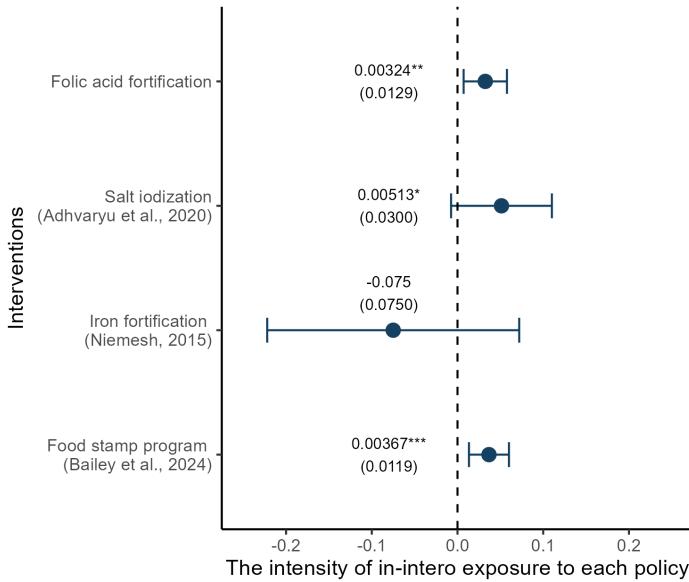


FIGURE 14: COMPARING EFFECTS OF IN-UTERO EXPOSURE TO DIFFERENT NUTRITIONAL INTERVENTIONS ON YEARS OF SCHOOLING

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.

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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Appendices

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:

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

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:

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

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.

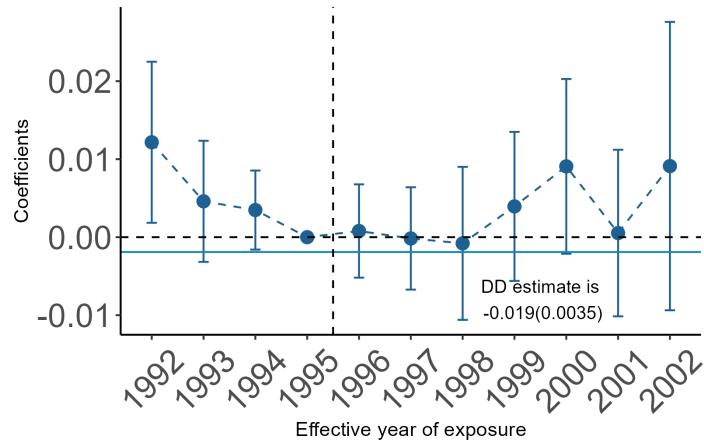


FIGURE B1: IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION DOES NOT AFFECT PROBABILITY OF BEING A NOMOVER

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

| | Dietary folate (1) | Serum folate (2) | RBC folate (3) | Dietary folate (4) | Serum folate (5) | RBC folate (6) |
|--|-----------------------|------------------------|----------------------|-----------------------|----------------------|---------------------|
| CNS anomaly rate, CZ-by-state level | -0.3616 (3.658) | -0.8190*** (0.2881) | -14.43*** (4.884) | | | |
| CNS anomaly rate, state level | | | | 1.269 (3.703) | -0.5164* (0.2687) | -11.99** (4.709) |
| R ² | 2.30×10 ⁻⁶ | 0.0018 | 0.0023 | 2.62×10 ⁻⁵ | 0.0007 | 0.0014 |
| Observations | 12,928 | 10,842 | 10,913 | 12,928 | 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.

TABLE B3: EFFECTS OF FOLIC ACID FORTIFICATION ON MATERNAL CHARACTERISTICS, HIGH- VERSUS LOW-EXPOSURE REGIONS

| | Share of mothers with following characteristics | | | | | |
|-------------------------|---|----------------------------|-----------------------|---------------------------------|--------------------|---------------------|
| | Age ≤ 22 (1) | Education < college (2) | Unmarried (3) | Inadequate prenatal care (4) | Non-white (5) | Hispanic (6) |
| Top 40 × Post | 0.0046*** (0.0014) | 0.0064** (0.0031) | 0.0078** (0.0032) | 0.0087 (0.0066) | 0.0025 (0.0026) | -0.0012 (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 × Post | 0.0052*** (0.0014) | 0.0074** (0.0033) | 0.0085** (0.0032) | 0.0098 (0.0077) | 0.0007 (0.0026) | 0.0005 (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 × Post | 0.0042*** (0.0016) | 0.0054 (0.0047) | 0.0104*** (0.0032) | 0.0137 (0.0100) | 0.0004 (0.0032) | 0.0017 (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.

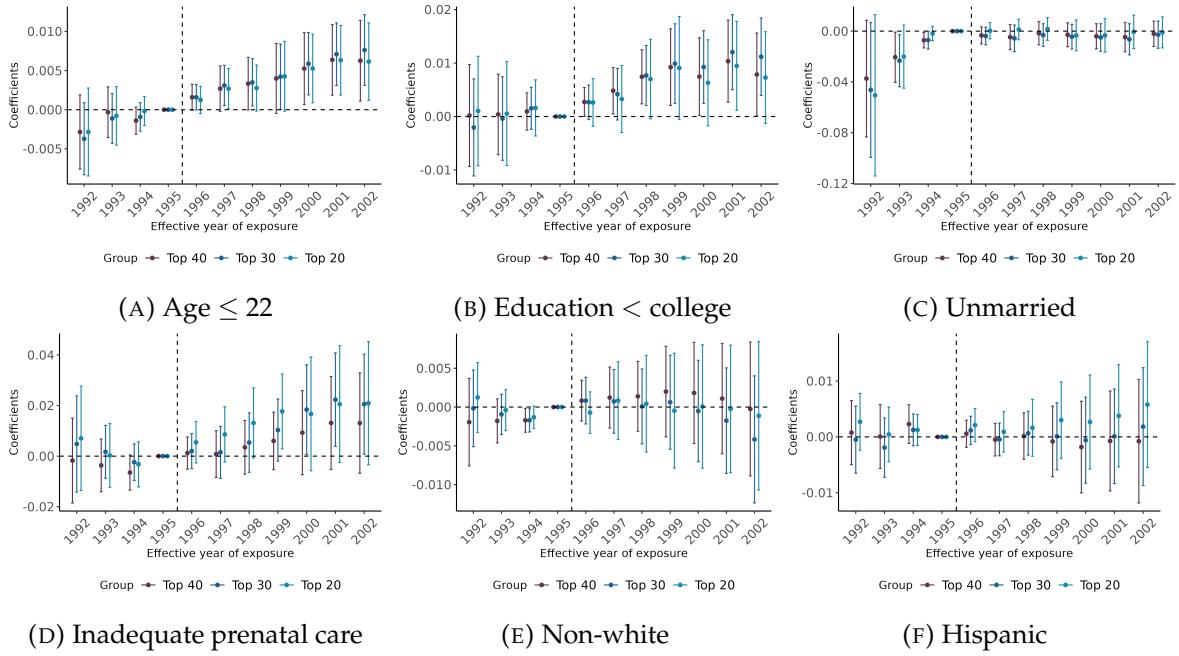


FIGURE B2: DYNAMIC EFFECTS OF FOLIC ACID FORTIFICATION ON MATERNAL CHARACTERISTICS, BINARY EXPOSURE

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.

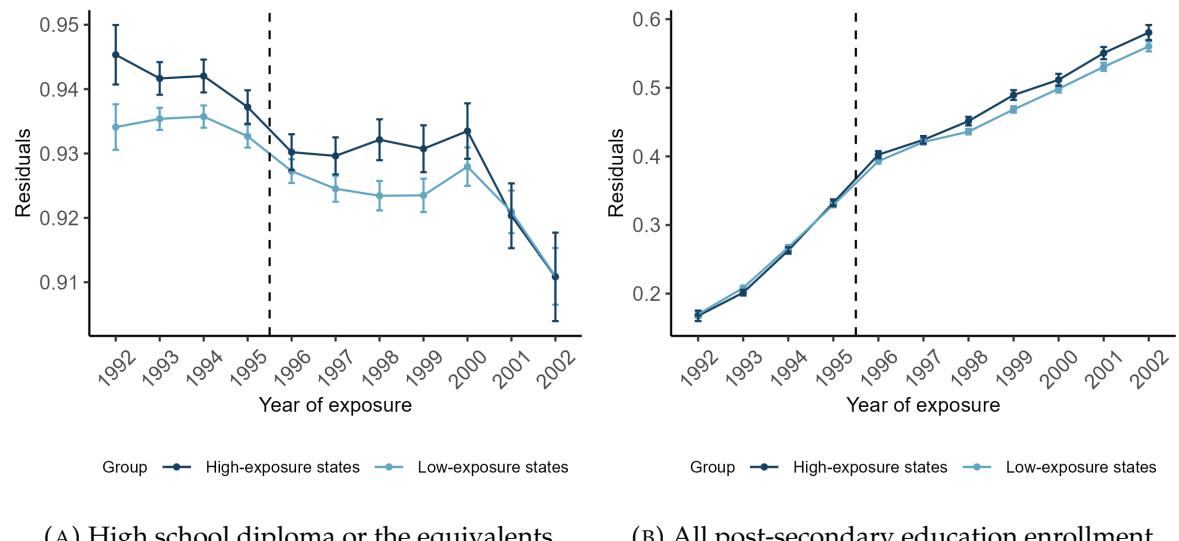


FIGURE B3: COHORT TRENDS IN RESIDUAL EDUCATIONAL OUTCOMES OF YOUNG ADULTS

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.

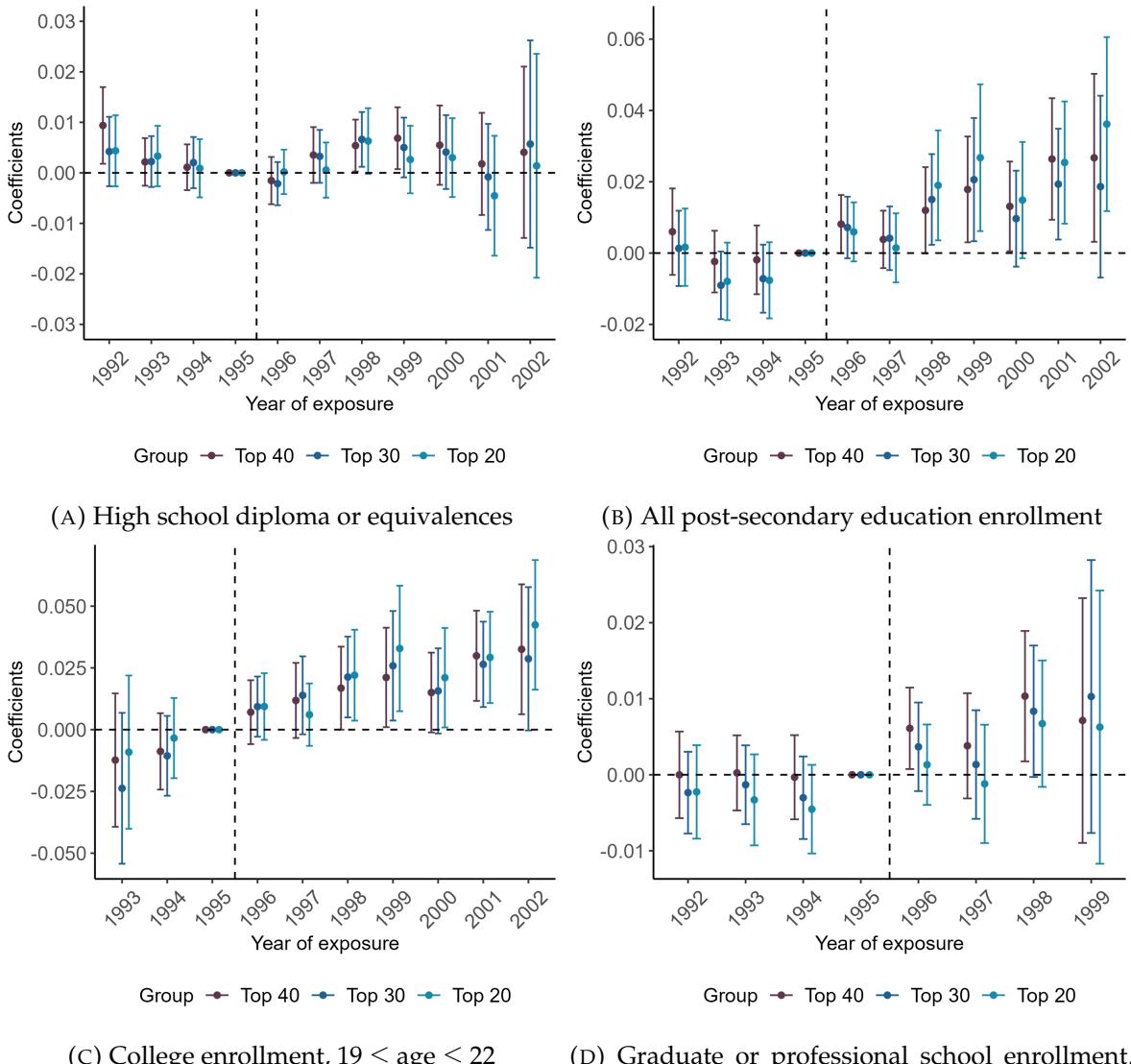


FIGURE B4: DYNAMIC EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON EDUCATIONAL OUTCOMES OF YOUNG ADULTS, HIGH- VERSUS LOW-EXPOSURE REGIONS, NONMOVERS

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.

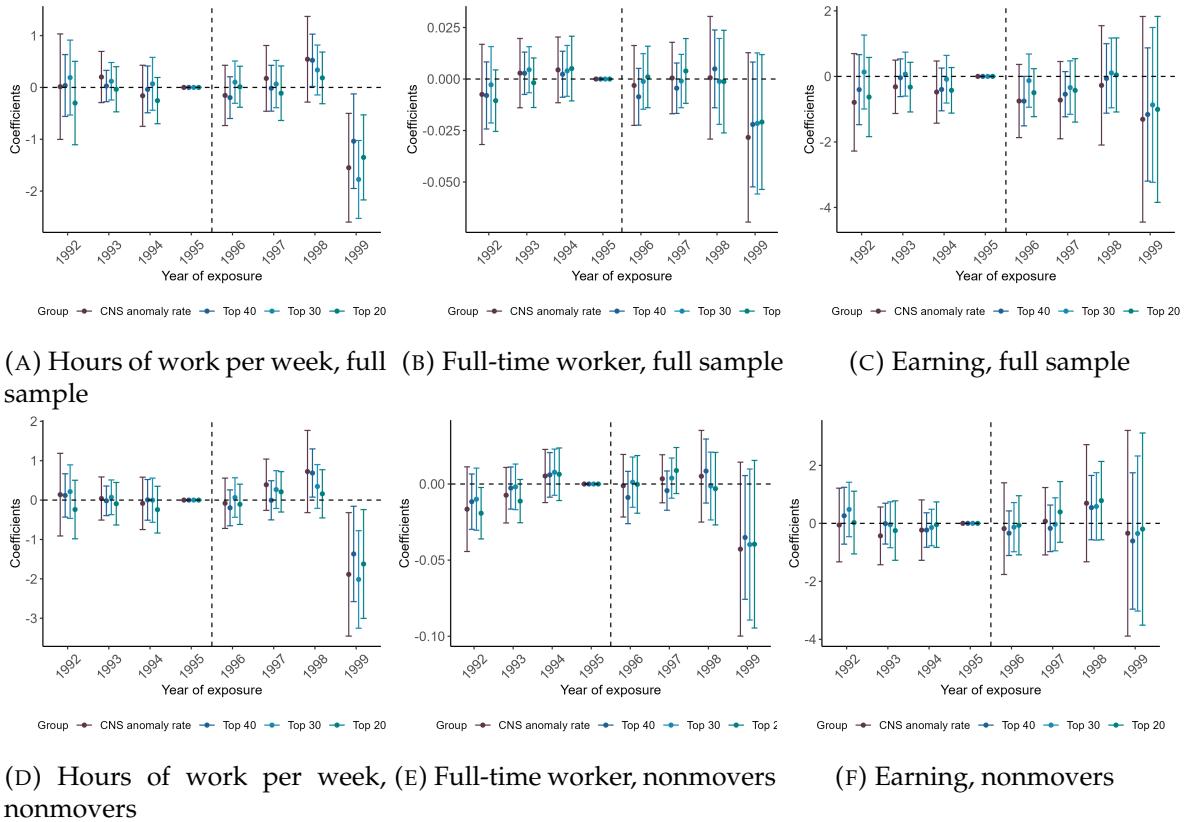


FIGURE B5: DYNAMIC EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON LABOR OUTCOMES OF YOUNG ADULTS OLDER THAN 22 YEARS OLD

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 B4: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON EDUCATIONAL OUTCOMES OF YOUNG ADULTS, BY GENDER

| | Full sample | | | | Nonmovers | | | |
|-------------------------|---------------------|----------------------|-----------------------|-----------------------|---------------------|-----------------------|-----------------------|----------------------|
| | HS or equivalences | All post-secondary | College | Grad. or Prof. | HS or equivalences | All post-secondary | College | Grad. or Prof. |
| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) |
| Panel A: female | | | | | | | | |
| CNS anomaly rate × Post | -0.0007 (0.0024) | 0.0084** (0.0039) | 0.0117** (0.0047) | 0.0021 (0.0018) | 0.0008 (0.0023) | 0.0135*** (0.0035) | 0.0195*** (0.0065) | 0.0025 (0.0020) |
| Observations | 697,675 | 697,675 | 391,263 | 306,412 | 495,378 | 495,378 | 281,626 | 213,752 |
| R ² | 0.0074 | 0.1460 | 0.0604 | 0.0090 | 0.0079 | 0.1408 | 0.0559 | 0.0099 |
| Dep. var. mean | 0.9438 | 0.4032 | 0.5442 | 0.0766 | 0.9401 | 0.4001 | 0.5335 | 0.0675 |
| Top 30 × Post | -0.0012 (0.0021) | 0.0093** (0.0039) | 0.0145*** (0.0048) | 0.0034 (0.0021) | 0.0009 (0.0023) | 0.0169*** (0.0041) | 0.0262*** (0.0069) | 0.0045 (0.0031) |
| Observations | 697,675 | 697,675 | 391,263 | 306,412 | 495,378 | 495,378 | 281,626 | 213,752 |
| R ² | 0.0074 | 0.1459 | 0.0604 | 0.0090 | 0.0079 | 0.1408 | 0.0559 | 0.0099 |
| Dep. var. mean | 0.9438 | 0.4032 | 0.5442 | 0.0766 | 0.9401 | 0.4001 | 0.5335 | 0.0675 |
| Panel B: male | | | | | | | | |
| CNS anomaly rate × Post | 0.0004 (0.0020) | 0.0055 (0.0048) | 0.0059 (0.0064) | 0.0056*** (0.0019) | -0.0030 (0.0020) | 0.0062 (0.0047) | 0.0061 (0.0069) | 0.0053* (0.0030) |
| Observations | 742,846 | 742,846 | 416,406 | 326,440 | 532,035 | 532,035 | 300,194 | 231,841 |
| R ² | 0.0127 | 0.1172 | 0.0482 | 0.0054 | 0.0137 | 0.11689 | 0.0475 | 0.00562 |
| Dep. var. mean | 0.9183 | 0.3241 | 0.4441 | 0.0456 | 0.9114 | 0.3180 | 0.4335 | 0.0382 |
| Top 30 × Post | 0.0034 (0.0021) | 0.0099* (0.0049) | 0.0152** (0.0075) | 0.0061*** (0.0022) | 0.0002 (0.0025) | 0.0118** (0.0051) | 0.0175** (0.0087) | 0.0062** (0.0028) |
| Observations | 742,846 | 742,846 | 416,406 | 326,440 | 532,035 | 532,035 | 300,194 | 231,841 |
| R ² | 0.0127 | 0.1172 | 0.0482 | 0.0054 | 0.0137 | 0.1169 | 0.0475 | 0.0056 |
| Dep. var. mean | 0.9183 | 0.3241 | 0.4441 | 0.0456 | 0.9114 | 0.3180 | 0.4335 | 0.0382 |

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: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON LABOR OUTCOMES OF YOUNG ADULTS AGED 19-22, BY GENDER

| | Full sample | | | Nonmovers | | |
|-------------------------|------------------------------|--------------------------|---------------------------------|------------------------------|--------------------------|---------------------------------|
| | Hours worked per week (1) | Working full-time (2) | Annual earning (\$1,000) (3) | Hours worked per week (4) | Working full-time (5) | Annual earning (\$1,000) (6) |
| Panel A: female | | | | | | |
| CNS anomaly rate × Post | -0.2379 (0.1599) | -0.0107** (0.0041) | 0.0338 (0.1411) | -0.3994* (0.2103) | -0.0175*** (0.0062) | -0.0988 (0.1737) |
| Observations | 391,263 | 301,752 | 391,263 | 281,626 | 215,074 | 281,626 |
| R ² | 0.0449 | 0.0378 | 0.0511 | 0.0505 | 0.0402 | 0.0529 |
| Dep. var. mean | 21.9409 | 0.3164 | 9.7462 | 21.7403 | 0.3141 | 9.6894 |
| Top 30 × Post | -0.4686*** (0.1519) | -0.0201*** (0.0057) | -0.2001 (0.1853) | -0.7103*** (0.2211) | -0.0261*** (0.0095) | -0.4206* (0.2167) |
| Observations | 391,263 | 301,752 | 391,263 | 281,626 | 215,074 | 281,626 |
| R ² | 0.0449 | 0.0378 | 0.0511 | 0.0506 | 0.0403 | 0.0529 |
| Dep. var. mean | 21.9409 | 0.3164 | 9.7462 | 21.7403 | 0.3141 | 9.6894 |
| Panel B: male | | | | | | |
| CNS anomaly rate × Post | 0.1094 (0.1572) | -0.0054 (0.0062) | -0.2252 (0.1512) | -0.0928 (0.2044) | -0.0078 (0.0074) | -0.5152*** (0.1814) |
| Observations | 416,406 | 315,513 | 416,406 | 300,194 | 223,958 | 300,194 |
| R ² | 0.0535 | 0.0365 | 0.0539 | 0.0639 | 0.0412 | 0.0582 |
| Dep. var. mean | 25.0536 | 0.4787 | 12.3416 | 24.4155 | 0.4697 | 12.1622 |
| Top 30 × Post | -0.1136 (0.1689) | -0.0107 (0.0067) | -0.2726 (0.1846) | -0.2706 (0.2311) | -0.0127 (0.0088) | -0.5661** (0.2222) |
| Observations | 416,406 | 315,513 | 416,406 | 300,194 | 223,958 | 300,194 |
| R ² | 0.0535 | 0.0365 | 0.0539 | 0.0639 | 0.0412 | 0.0582 |
| Dep. var. mean | 25.0536 | 0.4787 | 12.3416 | 24.4155 | 0.4697 | 12.1622 |

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 ON EDUCATIONAL OUTCOMES OF YOUNG ADULTS, BY WHITE AND NON-WHITE

| | Full sample | | | | Nonmovers | | | |
|-------------------------|----------------------|----------------------|----------------------|----------------------|----------------------|-----------------------|-----------------------|----------------------|
| | HS or equivalences | All post-secondary | College | Grad. or Prof. | HS or equivalences | All post-secondary | College | Grad. or Prof. |
| | | (1) | (2) | (3) | (4) | (5) | (6) | (8) |
| Panel A: white | | | | | | | | |
| CNS anomaly rate × Post | 0.0014 (0.0014) | 0.0045 (0.0049) | 0.0076 (0.0054) | 0.0036* (0.0018) | 0.0005 (0.0016) | 0.0066 (0.0046) | 0.0089 (0.0062) | 0.0034 (0.0027) |
| Observations | 1,013,479 | 1,013,479 | 570,466 | 443,013 | 710,631 | 710,631 | 405,080 | 305,551 |
| R ² | 0.0097 | 0.1456 | 0.0575 | 0.0096 | 0.0103 | 0.1424 | 0.0528 | 0.0107 |
| Dep. var. mean | 0.9388 | 0.3739 | 0.5121 | 0.0647 | 0.9340 | 0.3699 | 0.5019 | 0.0559 |
| Top 30 × Post | 0.0026** (0.0013) | 0.0090* (0.0048) | 0.0142** (0.0064) | 0.0049** (0.0023) | 0.0020 (0.0018) | 0.0127** (0.0048) | 0.0181** (0.0081) | 0.0055 (0.0037) |
| Observations | 1,013,479 | 1,013,479 | 570,466 | 443,013 | 710,631 | 710,631 | 405,080 | 305,551 |
| R ² | 0.0097 | 0.1456 | 0.0575 | 0.0096 | 0.0103 | 0.1424 | 0.0528 | 0.0107 |
| Dep. var. mean | 0.9388 | 0.3739 | 0.5121 | 0.0647 | 0.9340 | 0.3699 | 0.5019 | 0.0559 |
| Panel B: non-white | | | | | | | | |
| CNS anomaly rate × Post | -0.0041 (0.0030) | 0.0113** (0.0046) | 0.0098 (0.0069) | 0.0055* (0.0030) | -0.0053* (0.0030) | 0.0153*** (0.0049) | 0.0203** (0.0084) | 0.0060** (0.0026) |
| Observations | 427,042 | 427,042 | 237,203 | 189,839 | 316,782 | 316,782 | 176,740 | 140,042 |
| R ² | 0.0138 | 0.1206 | 0.0682 | 0.0146 | 0.0161 | 0.1217 | 0.0707 | 0.0144 |
| Dep. var. mean | 0.9144 | 0.3399 | 0.4528 | 0.0531 | 0.9089 | 0.3352 | 0.4434 | 0.0461 |
| Top 30 × Post | -0.0018 (0.0027) | 0.0138** (0.0052) | 0.0144* (0.0083) | 0.0064** (0.0030) | -0.0022 (0.0030) | 0.0209*** (0.0062) | 0.0299*** (0.0105) | 0.0069** (0.0026) |
| Observations | 427,042 | 427,042 | 237,203 | 189,839 | 316,782 | 316,782 | 176,740 | 140,042 |
| R ² | 0.0138 | 0.1206 | 0.0682 | 0.0146 | 0.0161 | 0.1217 | 0.0707 | 0.0144 |
| Dep. var. mean | 0.9144 | 0.3399 | 0.4528 | 0.0531 | 0.9089 | 0.3352 | 0.4434 | 0.0461 |

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 LABOR OUTCOMES OF YOUNG ADULTS, BY WHITE AND NON-WHITE

| | Full sample | | | Nonmovers | | |
|-------------------------|------------------------------|--------------------------|---------------------------------|------------------------------|--------------------------|---------------------------------|
| | Hours worked per week (1) | Working full-time (2) | Annual earning (\$1,000) (3) | Hours worked per week (4) | Working full-time (5) | Annual earning (\$1,000) (6) |
| Panel A: white | | | | | | |
| CNS anomaly rate × Post | -0.1388 (0.1323) | -0.0106** (0.0049) | -0.1379 (0.1076) | -0.3715** (0.1579) | -0.0171*** (0.0057) | -0.3196** (0.1392) |
| Observations | 570,466 | 452,605 | 570,466 | 405,080 | 319,023 | 405,080 |
| R ² | 0.0549 | 0.0674 | 0.0603 | 0.0606 | 0.0690 | 0.0639 |
| Dep. var. mean | 24.4178 | 0.3970 | 11.4403 | 24.0971 | 0.3916 | 11.4070 |
| Top 30 × Post | -0.3575** (0.1391) | -0.0182*** (0.0041) | -0.2184 (0.1394) | -0.5744*** (0.1748) | -0.0240*** (0.0049) | -0.4147** (0.1862) |
| Observations | 570,466 | 452,605 | 570,466 | 405,080 | 319,023 | 405,080 |
| R ² | 0.0549 | 0.0674 | 0.0603 | 0.0606 | 0.0691 | 0.0639 |
| Dep. var. mean | 24.4178 | 0.3970 | 11.4403 | 24.0971 | 0.3916 | 11.4070 |
| Panel B: non-white | | | | | | |
| CNS anomaly rate × Post | -0.0854 (0.2353) | -0.0026 (0.0064) | 0.0514 (0.1577) | -0.1400 (0.2971) | -5e-04 (0.0088) | -0.2363 (0.2108) |
| Observations | 237,203 | 164,660 | 237,203 | 176,740 | 120,009 | 176,740 |
| R ² | 0.0507 | 0.0536 | 0.0503 | 0.0560 | 0.0549 | 0.0510 |
| Dep. var. mean | 21.7321 | 0.4051 | 10.3383 | 21.1884 | 0.3977 | 10.0828 |
| Top 30 × Post | -0.2805 (0.2973) | -0.0079 (0.0076) | -0.3236 (0.2703) | -0.3819 (0.3860) | -0.0039 (0.0111) | -0.7287** (0.3057) |
| Observations | 237,203 | 164,660 | 237,203 | 176,740 | 120,009 | 176,740 |
| R ² | 0.0507 | 0.0536 | 0.0503 | 0.0560 | 0.0549 | 0.0511 |
| Dep. var. mean | 21.7321 | 0.4051 | 10.3383 | 21.1884 | 0.3977 | 10.0828 |

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, BY HISPANIC AND NON-HISPANIC

| | Full sample | | | | Nonmovers | | | |
|-------------------------|-----------------------|----------------------|-----------------------|-----------------------|------------------------|-----------------------|-----------------------|----------------------|
| | HS or equivalences | All post-secondary | College | Grad. or Prof. | HS or equivalences | All post-secondary | College | Grad. or Prof. |
| | | (1) | (2) | (3) | (4) | (5) | (6) | (8) |
| Panel A: Hispanic | | | | | | | | |
| CNS anomaly rate × Post | -0.0065** (0.0029) | 0.0041 (0.0040) | 0.0021 (0.0059) | 0.0081*** (0.0029) | -0.0097*** (0.0036) | 0.0055 (0.0051) | -1e-04 (0.0061) | 0.0064 (0.0045) |
| Observations | 250,217 | 250,217 | 141,842 | 108,375 | 195,317 | 195,317 | 111,960 | 83,357 |
| R ² | 0.0097 | 0.0987 | 0.0466 | 0.0074 | 0.0109 | 0.1024 | 0.0485 | 0.0081 |
| Dep. var. mean | 0.9059 | 0.3398 | 0.4405 | 0.0415 | 0.9048 | 0.3477 | 0.4474 | 0.0389 |
| Top 30 × Post | -0.0063* (0.0037) | 0.0088 (0.0057) | 0.0078 (0.0077) | 0.0093** (0.0036) | -0.0103** (0.0051) | 0.0100 (0.0068) | 0.0062 (0.0088) | 0.0077 (0.0054) |
| Observations | 250,217 | 250,217 | 141,842 | 108,375 | 195,317 | 195,317 | 111,960 | 83,357 |
| R ² | 0.0097 | 0.0987 | 0.0466 | 0.0074 | 0.0109 | 0.1024 | 0.0485 | 0.0081 |
| Dep. var. mean | 0.9059 | 0.3398 | 0.4405 | 0.0415 | 0.9048 | 0.3477 | 0.4474 | 0.0389 |
| Panel B: non-Hispanic | | | | | | | | |
| CNS anomaly rate × Post | 8e-04 (0.0016) | 0.0086 (0.0056) | 0.0109* (0.0059) | 0.0038** (0.0015) | 3e-04 (0.0015) | 0.0115 ** (0.0046) | 0.0159** (0.0062) | 0.0043** (0.0020) |
| Observations | 1,190,304 | 1,190,304 | 665,827 | 524,477 | 832,096 | 832,096 | 469,860 | 362,236 |
| R ² | 0.0112 | 0.1466 | 0.0652 | 0.0110 | 0.0131 | 0.1445 | 0.0636 | 0.0124 |
| Dep. var. mean | 0.9366 | 0.3681 | 0.5056 | 0.0651 | 0.9307 | 0.3607 | 0.4916 | 0.0558 |
| Top 30 × Post | 0.0016 (0.0014) | 0.0120** (0.0049) | 0.0171*** (0.0056) | 0.0051*** (0.0019) | 0.0015 (0.0017) | 0.0168*** (0.0043) | 0.0251*** (0.0064) | 0.0061** (0.0025) |
| Observations | 1,190,304 | 1,190,304 | 665,827 | 524,477 | 832,096 | 832,096 | 469,860 | 362,236 |
| R ² | 0.0112 | 0.1466 | 0.0652 | 0.0110 | 0.0131 | 0.1445 | 0.0637 | 0.0124 |
| Dep. var. mean | 0.9366 | 0.3681 | 0.5056 | 0.0651 | 0.9307 | 0.3607 | 0.4916 | 0.0558 |

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 LABOR OUTCOMES OF YOUNG ADULTS, BY HISPANIC AND NON-HISPANIC

| | Full sample | | | Nonmovers | | |
|-------------------------|------------------------------|--------------------------|---------------------------------|------------------------------|--------------------------|---------------------------------|
| | Hours worked per week (1) | Working full-time (2) | Annual earning (\$1,000) (3) | Hours worked per week (4) | Working full-time (5) | Annual earning (\$1,000) (6) |
| Panel A: Hispanic | | | | | | |
| CNS anomaly rate × Post | -0.1185 (0.3120) | -0.0022 (0.0088) | 0.1186 (0.2669) | -0.2162 (0.3299) | -0.0047 (0.0117) | 0.0055 (0.3388) |
| Observations | 141,842 | 104,174 | 141,842 | 111,960 | 80,452 | 111,960 |
| R ² | 0.0572 | 0.0617 | 0.0542 | 0.0641 | 0.0636 | 0.0583 |
| Dep. var. mean | 23.3623 | 0.4353 | 11.5870 | 22.6612 | 0.4252 | 11.2689 |
| Top 30 × Post | -0.4574 (0.3959) | -0.0081 (0.0107) | -0.0702 (0.3589) | -0.4586 (0.4600) | -0.0112 (0.0143) | -0.2316 (0.4458) |
| Observations | 141,842 | 104,174 | 141,842 | 111,960 | 80,452 | 111,960 |
| R ² | 0.0572 | 0.0617 | 0.0542 | 0.0641 | 0.0636 | 0.0583 |
| Dep. var. mean | 23.3623 | 0.4353 | 11.5870 | 22.6612 | 0.4252 | 11.2689 |
| Panel B: non-Hispanic | | | | | | |
| CNS anomaly rate × Post | -0.1431 (0.1322) | -0.0095* (0.0050) | -0.1911 (0.1194) | -0.3520** (0.1580) | -0.0150** (0.0058) | -0.4517*** (0.1264) |
| Observations | 665,827 | 513,091 | 665,827 | 469,860 | 358,580 | 469,860 |
| R ² | 0.0571 | 0.0615 | 0.0581 | 0.0633 | 0.0629 | 0.0606 |
| Dep. var. mean | 23.5856 | 0.3911 | 10.9555 | 23.2383 | 0.3853 | 10.8753 |
| Top 30 × Post | -0.3464** (0.1335) | -0.0175*** (0.0048) | -0.3159** (0.1566) | -0.5728*** (0.1750) | -0.0223*** (0.0064) | -0.6032*** (0.1772) |
| Observations | 665,827 | 513,091 | 665,827 | 469,860 | 358,580 | 469,860 |
| R ² | 0.0571 | 0.0615 | 0.0581 | 0.0634 | 0.0630 | 0.0606 |
| Dep. var. mean | 23.5856 | 0.3911 | 10.9555 | 23.2383 | 0.3853 | 10.8753 |

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 B10: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION, CONTROLLING FOR CENSUS DIVISION SPECIFIC TREND

| | School enrollment | | | Labor supply | | |
|-------------------------|-----------------------|------------------------|---------------------------------|--------------------------------------|----------------------------------|-------------------------------|
| | All post-secondary | College, 19 ≤ age ≤ 22 | Grad. or prof. school, age > 22 | Hours worked per week, 19 ≤ age ≤ 22 | Working full-time, 19 ≤ age ≤ 22 | Annual earning, 19 ≤ age ≤ 22 |
| | (1) | (2) | (3) | (4) | (5) | (6) |
| Panel A: full sample | | | | | | |
| CNS anomaly rate × Post | 0.0067 (0.0041) | 0.0051 (0.0039) | 0.0030** (0.0013) | -0.0857 (0.1260) | -0.0068 (0.0041) | -0.1269 (0.1062) |
| Observations | 1,314,116 | 736,549 | 577,567 | 736,549 | 564,360 | 736,549 |
| R ² | 0.1333 | 0.0605 | 0.0102 | 0.0531 | 0.0614 | 0.0566 |
| Dep. var. mean | 0.3598 | 0.4882 | 0.0592 | 23.7197 | 0.4030 | 11.1461 |
| Top 30 × Post | 0.0111** (0.0046) | 0.0089* (0.0049) | 0.0045** (0.0019) | -0.3676** (0.1379) | -0.0130*** (0.0043) | -0.3494** (0.1634) |
| Observations | 1,314,116 | 736,549 | 577,567 | 736,549 | 564,360 | 736,549 |
| R ² | 0.1333 | 0.0605 | 0.0102 | 0.0531 | 0.0614 | 0.0566 |
| Dep. var. mean | 0.3598 | 0.4882 | 0.0592 | 23.7197 | 0.4030 | 11.1461 |
| Panel B: nonmovers | | | | | | |
| CNS anomaly rate × Post | 0.0079** (0.0033) | 0.0071* (0.0037) | 0.0019 (0.0018) | -0.2848* (0.1694) | -0.0122** (0.0050) | -0.3139** (0.1462) |
| Observations | 938,092 | 531,061 | 407,031 | 531,061 | 401,793 | 531,061 |
| R ² | 0.1315 | 0.0585 | 0.0104 | 0.0582 | 0.0625 | 0.0582 |
| Dep. var. mean | 0.3551 | 0.4779 | 0.0507 | 23.2968 | 0.3974 | 11.0254 |
| Top 30 × Post | 0.0124*** (0.0045) | 0.0121** (0.0054) | 0.0028 (0.0022) | -0.6294*** (0.2058) | -0.0181*** (0.0056) | -0.5982*** (0.2171) |
| Observations | 938,092 | 531,061 | 407,031 | 531,061 | 401,793 | 531,061 |
| R ² | 0.1315 | 0.0585 | 0.0104 | 0.0582 | 0.0626 | 0.0583 |
| Dep. var. mean | 0.3551 | 0.4779 | 0.0507 | 23.2968 | 0.3974 | 11.0254 |

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 state-level baseline characteristics interacted with time trends. I account for census division specific trend by controlling for dummies for census division of birth interacted with quarter-and-year of birth.

TABLE B11: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON EDUCATIONAL OUTCOMES OF YOUNG ADULTS, CONTROLLING FOR STATE OF RESIDENCE FIXED EFFECTS

| | School enrollment | | | Labor supply | | |
|-------------------------|-----------------------|------------------------|---------------------------------|--------------------------------------|----------------------------------|-------------------------------|
| | All post-secondary | College, 19 ≤ age ≤ 22 | Grad. or prof. school, age > 22 | Hours worked per week, 19 ≤ age ≤ 22 | Working full-time, 19 ≤ age ≤ 22 | Annual earning, 19 ≤ age ≤ 22 |
| | (1) | (2) | (3) | (4) | (5) | (6) |
| CNS anomaly rate × Post | 0.0070* (0.0040) | 0.0089* (0.0045) | 0.0039*** (0.0013) | -0.0809 (0.1240) | -0.0082* (0.0041) | -0.1000 (0.0949) |
| Observations | 1,440,521 | 807,669 | 632,852 | 807,669 | 617,265 | 807,669 |
| R ² | 0.1385 | 0.0665 | 0.0126 | 0.0597 | 0.0645 | 0.0586 |
| Dep. var. mean | 0.3627 | 0.4927 | 0.0608 | 23.5413 | 0.3995 | 11.0806 |
| Top 30 × Post | 0.0096** (0.0038) | 0.0148*** (0.0048) | 0.0049*** (0.0016) | -0.3101** (0.1238) | -0.0155*** (0.0040) | -0.2401* (0.1411) |
| Observations | 1,440,521 | 807,669 | 632,852 | 807,669 | 617,265 | 807,669 |
| R ² | 0.1385 | 0.0665 | 0.0126 | 0.0597 | 0.0645 | 0.0586 |
| Dep. var. mean | 0.3627 | 0.4927 | 0.0608 | 23.5413 | 0.3995 | 11.0806 |
| CNS anomaly rate × Post | 0.0097*** (0.0035) | 0.0127** (0.0048) | 0.0040** (0.0019) | -0.2560 (0.1640) | -0.0125** (0.0049) | -0.3221** (0.1322) |
| Observations | 1,027,413 | 581,820 | 445,593 | 581,820 | 439,032 | 581,820 |
| R ² | 0.1341 | 0.0598 | 0.0116 | 0.0610 | 0.0628 | 0.0591 |
| Dep. var. mean | 0.3580 | 0.4821 | 0.0525 | 23.1146 | 0.3935 | 10.9597 |
| Top 30 × Post | 0.0143*** (0.0038) | 0.0217*** (0.0055) | 0.0055** (0.0024) | -0.5064*** (0.1882) | -0.0195*** (0.0057) | -0.5112*** (0.1804) |
| Observations | 1,027,413 | 581,820 | 445,593 | 581,820 | 439,032 | 581,820 |
| R ² | 0.1341 | 0.0598 | 0.0116 | 0.0610 | 0.0628 | 0.0591 |
| Dep. var. mean | 0.3580 | 0.4821 | 0.0525 | 23.1146 | 0.3935 | 10.9597 |

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, state-level baseline characteristics interacted with time trends, and, additionally, state-of-residence fixed effects.

TABLE B12: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON EDUCATIONAL OUTCOMES OF YOUNG ADULTS, ACCOUNTING FOR MEAN REVERSION

| | School enrollment | | | Labor supply | | |
|-------------------------|-----------------------|------------------------|---------------------------------|--------------------------------------|----------------------------------|-------------------------------|
| | All post-secondary | College, 19 ≤ age ≤ 22 | Grad. or prof. school, age > 22 | Hours worked per week, 19 ≤ age ≤ 22 | Working full-time, 19 ≤ age ≤ 22 | Annual earning, 19 ≤ age ≤ 22 |
| | (1) | (2) | (3) | (4) | (5) | (6) |
| | Panel A: full sample | | | | | |
| CNS anomaly rate × Post | 0.0070* (0.0038) | 0.0078* (0.0043) | 0.0039*** (0.0013) | -0.1514 (0.1162) | -0.0070 (0.0044) | -0.0803 (0.0895) |
| Observations | 1,440,521 | 807,669 | 632,852 | 807,669 | 617,265 | 807,669 |
| R ² | 0.1385 | 0.0665 | 0.0126 | 0.0597 | 0.0645 | 0.0586 |
| Dep. var. mean | 0.3627 | 0.4927 | 0.0608 | 23.5413 | 0.3995 | 11.0806 |
| Top 30 × Post | 0.0094** (0.0038) | 0.0139*** (0.0046) | 0.0049*** (0.0017) | -0.3237** (0.1246) | -0.0143*** (0.0042) | -0.2263 (0.1350) |
| Observations | 1,440,521 | 807,669 | 632,852 | 807,669 | 617,265 | 807,669 |
| R ² | 0.1385 | 0.0665 | 0.0126 | 0.0597 | 0.0645 | 0.0586 |
| Dep. var. mean | 0.3627 | 0.4927 | 0.0608 | 23.5413 | 0.3995 | 11.0806 |
| | Panel B: nonmovers | | | | | |
| CNS anomaly rate × Post | 0.0104*** (0.0034) | 0.0124** (0.0047) | 0.0039** (0.0019) | -0.4192** (0.1687) | -0.0103* (0.0054) | -0.3272** (0.1299) |
| Observations | 1,027,413 | 581,820 | 445,593 | 581,820 | 439,032 | 581,820 |
| R ² | 0.1341 | 0.0598 | 0.0116 | 0.0611 | 0.0628 | 0.0591 |
| Dep. var. mean | 0.3580 | 0.4821 | 0.0525 | 23.1146 | 0.3935 | 10.9597 |
| Top 30 × Post | 0.0148*** (0.0037) | 0.0216*** (0.0054) | 0.0055** (0.0024) | -0.5479** (0.2077) | -0.0171** (0.0064) | -0.5116*** (0.1818) |
| Observations | 1,027,413 | 581,820 | 445,593 | 581,820 | 439,032 | 581,820 |
| R ² | 0.1341 | 0.0599 | 0.0116 | 0.0611 | 0.0628 | 0.0591 |
| Dep. var. mean | 0.3580 | 0.4821 | 0.0525 | 23.1146 | 0.3935 | 10.9597 |

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, state-level baseline characteristics interacted with time trends. I account for mean reversion by controlling for dependent variable means of young adults of 19-29 years old who were born from 1989 to 1992 interacted with the post-fortification dummy.

TABLE B13: EFFECTS OF IN-UTERO EXPOSURE TO FOLIC ACID FORTIFICATION ON EDUCATIONAL OUTCOMES OF YOUNG ADULTS, PLACEBO TEST USING COHORTS BORN IN 1983-1992

| | School enrollment | | | Labor supply | | |
|-------------------------|---------------------|------------------------|---------------------------------|--------------------------------------|----------------------------------|-------------------------------|
| | All post-secondary | College, 19 ≤ age ≤ 22 | Grad. or prof. school, age > 22 | Hours worked per week, 19 ≤ age ≤ 22 | Working full-time, 19 ≤ age ≤ 22 | Annual earning, 19 ≤ age ≤ 22 |
| | (1) | (2) | (3) | (4) | (5) | (6) |
| Panel A: full sample | | | | | | |
| CNS anomaly rate × Post | -0.0025 (0.0037) | – – | 0.0020 (0.0021) | – – | – – | – – |
| Observations | 1,553,624 | 357,804 | 1,195,820 | 357,804 | 267,338 | 357,804 |
| R ² | 0.1013 | 0.0543 | 0.0086 | 0.0543 | 0.0547 | 0.0523 |
| Dep. var. mean | 0.2559 | 0.4759 | 0.0540 | 22.6329 | 0.3719 | 8.5552 |
| Top 30 × Post | -0.0042 (0.0043) | – – | 0.0019 (0.0013) | – – | – – | – – |
| Observations | 1,553,624 | 357,804 | 1,195,820 | 357,804 | 267,338 | 357,804 |
| R ² | 0.1013 | 0.0543 | 0.0086 | 0.0543 | 0.0547 | 0.0523 |
| Dep. var. mean | 0.2559 | 0.4759 | 0.0540 | 22.6329 | 0.3719 | 8.5552 |
| Panel B: nonmovers | | | | | | |
| CNS anomaly rate × Post | -0.0017 (0.0040) | – – | 0.0015 (0.0013) | – – | – – | – – |
| Observations | 1,096,269 | 260,644 | 835,625 | 260,644 | 191,687 | 260,644 |
| R ² | 0.1067 | 0.0535 | 0.0090 | 0.0615 | 0.0561 | 0.0548 |
| Dep. var. mean | 0.2508 | 0.4665 | 0.0449 | 22.0863 | 0.3610 | 8.3581 |
| Top 30 × Post | -0.0039 (0.0050) | – – | 0.0024 (0.0014) | – – | – – | – – |
| Observations | 1,096,269 | 260,644 | 835,625 | 260,644 | 191,687 | 260,644 |
| R ² | 0.1067 | 0.0535 | 0.0090 | 0.0615 | 0.0561 | 0.0548 |
| Dep. var. mean | 0.2508 | 0.4665 | 0.0449 | 22.0863 | 0.3610 | 8.3581 |

Notes: --: removed due to collinearity. The sample is young adults aged 19-29 from ACSs 2011-2016. The timing of pseudo-treatment is defined to be 1986. 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, state-level baseline characteristics interacted with time trends.

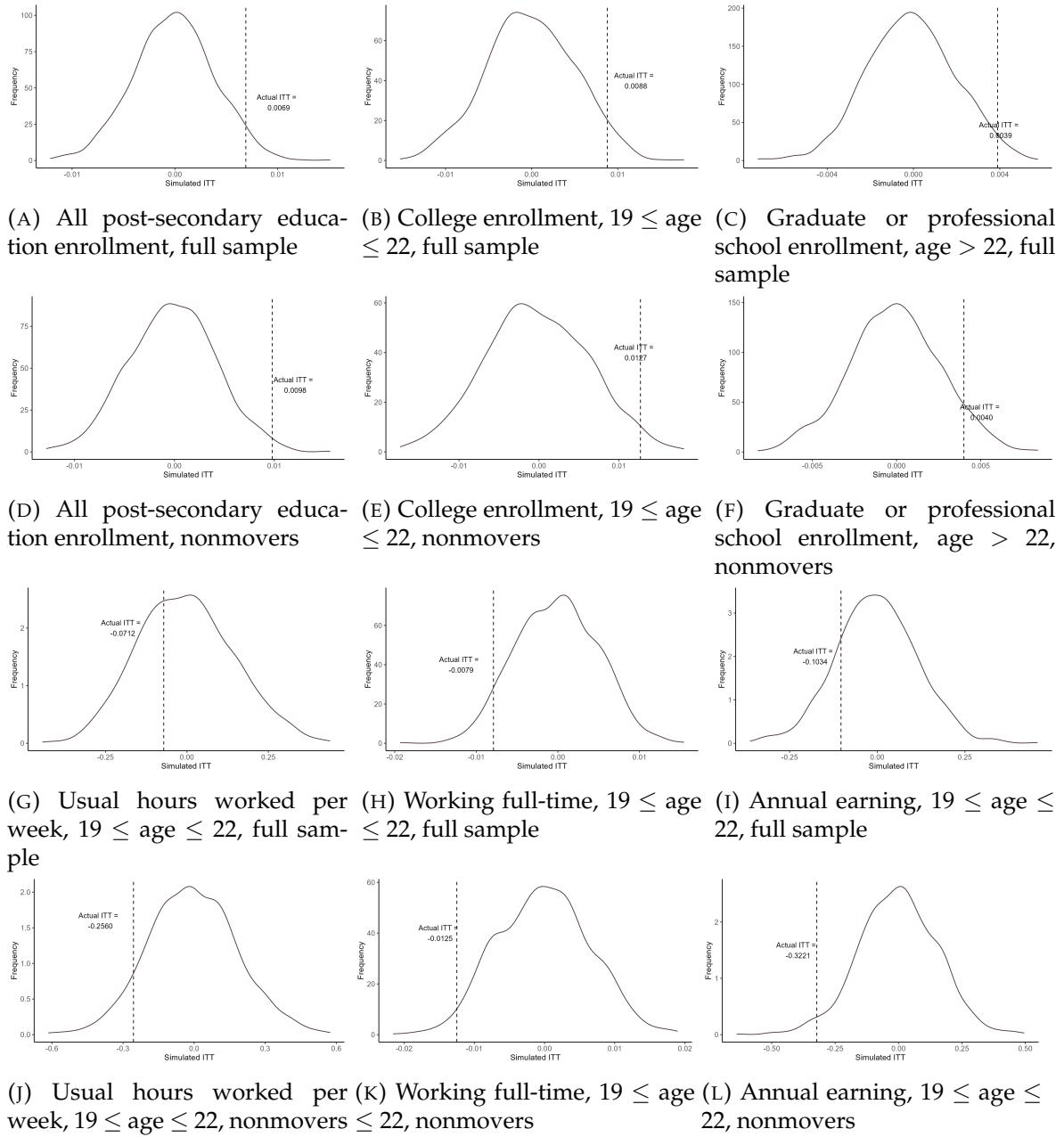


FIGURE B6: RANDOMIZATION TEST

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.