Long Run Effects of Folic Acid Fortification

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Abstract

Recent concerns about the effectiveness of food and nutrition assistance programs, driven by the challenges of altering consumer behavior, have called attention to reformulation—an understudied intervention that enhances food and nutrition security with minimal dietary changes. This paper explores a specific instance of reformulation: the folic acid fortification of enriched grain products in the U.S. during the late 1990s. The fortification substantially improved folate access and reduced birth defects. This paper examines the impact of folic acid fortification by comparing cohorts exposed and not exposed to the fortification across regions with varying baseline folate deficiency levels. The findings show that maternal exposure to folic acid fortification increases the likelihood of young adults enrolling in college or graduate/professional schools by 0.63-0.78 percentage points. These results suggest that micronutrient supplementation provides broad, long-lasting benefits for children and underscores the potential of fortification as an enhancement to current food assistance policies.

1 Introduction

The U.S. federal government allocates hundreds of billions of dollars annually to enhance food safety and nutrition. 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. 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 deficiency in folate, a critical nutrient for neurodevelopment. Maternal folate deficiency, particularly concerning during pregnancy, can lead to severe birth defects and cognitive impairments in children (Roth et al., 2011; Irvine et al., 2022). To counter these risks, the U.S. Food and Drug Administration (FDA) mandated the fortification of 40µg/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 on children's human capital outcomes.

I leverage geographical variation in folate-deficiency-related birth defects 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 was effective, we should observe more 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 incidence of folate-deficiency-related birth defects, specifically congenital anomalies of the central nervous system (CNS anomalies), 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 on school enrollment.

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) CNS anomaly rates declined after fortification, driven primarily by regions with higher pre-existing rates; and (4) CNS anomaly rates are negatively correlated with key biomarkers for folate deficiency, supporting the use of CNS anomaly rates as a proxy for spatial variation in pre-existing folate deficiency. My main results indicate that maternal exposure to folic acid fortification is associated with higher tertiary school enrollment in young adulthood.

This paper contributes to three strands of literature. First, it adds to the limited 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 army classification test scores and increased income Feyrer, Politi and Weil (2017); Serena (2019); Adhvaryu et al. (2020); Huang, Liu and Zhou (2020); Deng and Lindeboom (2022*a*); Tafesse (2022), little is known about the human capital effects of folic acid fortification. Unlike iodine and iron deficiencies Niemesh (2015), which primarily affect thyroid function and blood oxygen transport, folate deficiency directly affects nervous system development, potentially leading to more severe health consequences. Folic acid fortification, therefore, may have a greater influence on cognitive development and subsequent economic outcomes, such as educational attainment and income. Additionally, since folic acid fortification is less widely implemented than salt iodization and iron supplementation, particularly in developing countries[1], causal evidence from the U.S. can inform and motivate broader adoption of this policy.

This paper also contributes to the fetal origins literature by exploring the long-term effects of early-life nutritional access. Existing research demonstrates that early-life nutritional conditions have lasting effects, with negative shocks like famine (Meng and Qian, 2006; Almond et al., 2007; Chen and Zhou, 2007; Meng and Qian, 2009; Lindeboom, Portrait and Van den Berg, 2010; Scholte, Van Den Berg and Lindeboom, 2015; Deng and Lindeboom, 2022*b*) and Ramadan fasting (Almond and Mazumder, 2011; Almond, Mazumder and Van Ewijk, 2015; Majid, 2015; Greve, Schultz-Nielsen and Tekin, 2017) leading to poorer adult health and labor outcomes, while positive interventions, such as breastfeeding (Fitzsimons and Vera-Hernández, 2022), iodine supplementation (Field, Robles and Torero, 2009; Araújo, Carrillo and Sampaio, 2021), and food assistance (Hoynes, Schanzenbach and Al-

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

mond, 2016; Chorniy, Currie and Sonchak, 2020; East, 2020), 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 describes the data, Section 4 outlines the research design and discusses identifying assumptions, Sections 5 and 6 present empirical results for short-term and long-term outcomes, respectively, Section 7 discusses effect size and cost-effectiveness, and Section 8 concludes.

2 Background

2.1 Folate deficiency disorder

Folate deficiency is a major cause of neural tube defects (NTDs), the most common congenital anomalies of the central nervous system (CNS anomalies) in newborns (Smithells et al., 1983). Severe NTDs, such as anencephaly[2] are typically fatal, with most affected infants dying before or shortly after birth. Mild NTDs, like spina bifida[3] allow survival into adulthood but carry a high risk of lifelong physical and mental disabilities (Yi et al., 2011). In the early 1990s, approximately 4,000 fetuses in the U.S. (about 1 in 1,000) were affected by NTDs annually, with one-third lost due to selective or spontaneous abortions (Cragan et al., 1995; Mersereau et al., 2004). Folate deficiency can also lead to other congenital CNS anomalies, such as hydrocephaly and microcephaly (Naz et al., 2016; Liu et al., 2018; Wertelecki et al., 2018; Kempińska et al., 2022). These anomalies 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 CNS anomalies become more detectable (Blumenfeld, Siegler and Bronshtein, 1993), and many

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

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

pregnant women in the U.S. do not receive adequate prenatal care.

2.2 Sources of folate

Folate can be naturally obtained in foods such as beef liver, dark green leafy vegetables, beans, peas, nuts, fruits, and fruit juices. The poor stability of food folate under typical cooking conditions can substantially reduce the eventual amount of folate digested, which makes food folate less attractive as a means to enhance the folate status of pregnant women (McNulty and Pentieva, 2004). Despite proper cooking methods, it is still difficult to achieve the recommended level of folate intake for pregnant women from regular diets (Czeizel, 2000). According to the third National Health and Nutrition Examination Survey (NHANES III), mean daily folate consumption is 233.68 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 guidance[4], folic acid supplementation should start at least one month prior to conception. However, approximately 50% of pregnancies are unintended in the U.S. (Finer and Zolna, 2016). From 1995 to 1998, only about 30% of women in the U.S. reported taking vitamin supplements containing folic acid every day and less than 10% of them knew folic acid should be taken before pregnancy (Petrini, Damus and Johnston, 1999). Moreover, low-income women may have more difficulties accessing and affording folate-rich foods and folic acid supplements (Czeizel, 2000). Therefore, policymakers need to come up with a more affordable, ideally passive means to ensure folic acid adequacy for pregnant women.

2.3 Folic acid fortification 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

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

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. Although folic acid fortification is only mandated for enriched grain products, its effects are widespread, as these products are ingredients in many other foods. For example, some chips contain folic acid because they include enriched wheat flour (Figure 1).

Figure 1: Chips with Enriched Wheat Flour as an Ingredient

3 Data

The data for this analysis come from multiple sources. The primary treatment variable is the pre-existing rate of central nervous system (CNS) anomalies at the state-of-birth level, which reflects varying exposure to folic acid fortification. This rate is calculated using restricted-access Vital Statistics Natality Data. I then link spatial variations in pre-existing CNS anomaly rates to outcome variables from Vital Statistics Natality Data and the American Community Survey, based on state and year of birth.

3.1 Restricted-use 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.

Natality data serves several purposes. First, I calculate the pre-existing CNS anomaly rate by dividing the number of CNS anomalies by the total number of births from January 1989 to June 1993. This period was selected because the 1989 revisions to U.S. birth certificates included congenital anomaly data, though five states (Louisiana, Nebraska, Oklahoma, New York, and New Mexico) began reporting these anomalies at different times. To maximize state coverage, I include data up to mid-1993 to construct pre-existing CNS anomaly rates. Cohorts born after this period for cross-cohort comparisons to allow for at least four pre-periods in event study analysis. The resulting rates show significant spatial variation (Figure 2).

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.

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—and examine its effects on birth outcomes such as birth weight, incidence of low birth weight, and

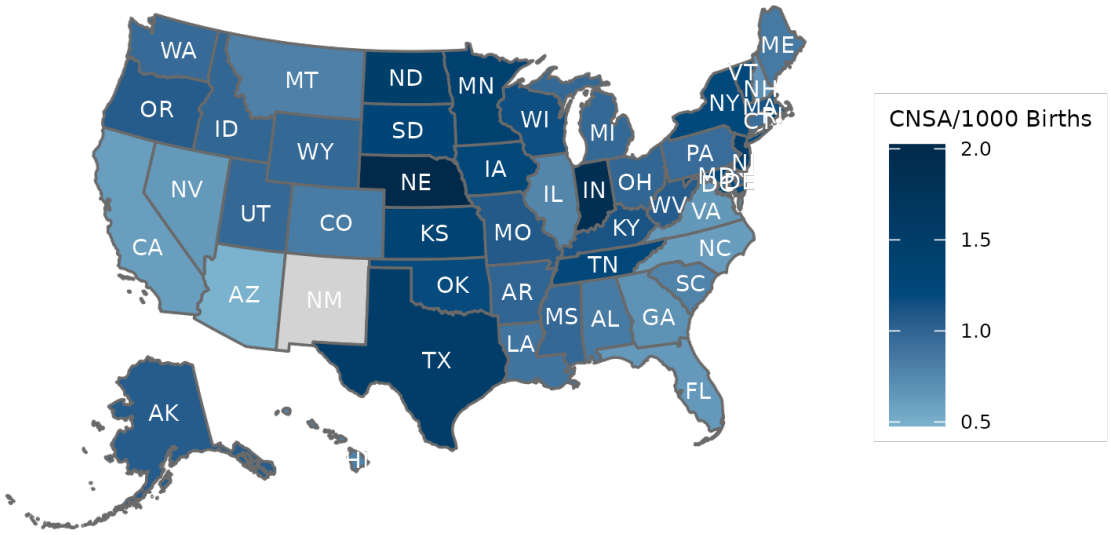


FIGURE 2: PRE-EXISTING CNS ANOMALY RATES BY STATE-OF-BIRTH

Notes: Congenital anomalies of the central nervous system, or CNS anomalies, include anencephaly, spinal bifida, hydrocephaly, microcephaly, and other CNS anomalies. 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. Quintiles are weighted by number of births at state level.

preterm births.

3.2 American Community Surveys

I link state-level pre-existing CNS anomaly rates to young adult outcomes from the American Community Survey Public-Use Microdata Sample (ACS PUMS) for the periods 2017–2019 and 2021–2022, excluding ACS 2020 due to its high nonresponse rate caused by the pandemic[5]. 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. Therefore, the most relevant human capital outcomes to examine are high school education and tertiary school enrollment. I focus on tertiary enrollment since high school graduation rates were already high, leaving limited

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

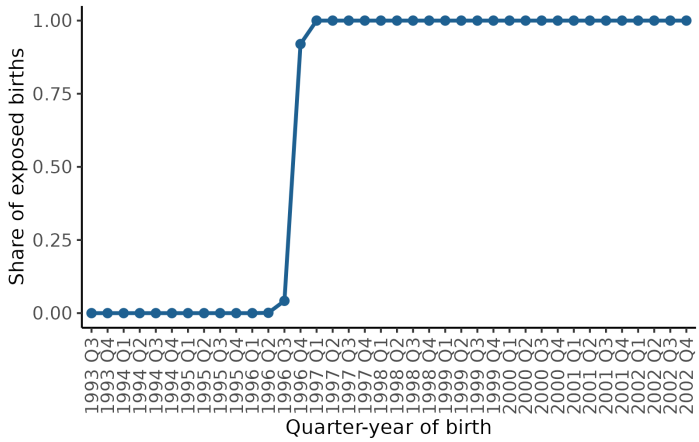


FIGURE 3: SHARE OF EXPOSED BIRTHS BY QUARTER-AND-YEAR-OF-BIRTH

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

room for further improvement.

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

4 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, Thuilliez and Valfort, 2021), measles rates for measles vaccination (Atwood, 2022), pneumonia rates for Sulfa antibiotic introduction (Lazuka, 2020), and goiter rates for salt iodization (Feyrer,

Politi and Weil, 2017; Adhvaryu et al., 2020).

4.1 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:

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

where Y_{ist} represents the outcome for individual i who born in state s and quarter-and-year t, CNS anomaly rate$_s$ is a measure of pre-existing CNS anomaly rates at state-of-birth level, the dummy variable $Post_{it}$ indicates whether the cohort is exposed, 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) state-of-residence-by-survey-year fixed effects, (ii) individual characteristics including gender, race dummies, and Hispanic origin, (iii) 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, (iv) a Bartik-style measure of county-level unemployment rate from Ganong and Liebman (2018) to control for local economic condition at birth, and (v) state-level baseline characteristics listed in Figure **??** interacted with linear time trend (quarter-and-year-of-birth) to control for possible differences in trends across counties.

4.2 Identifying assumptions

The validity of this research design hinges on several assumptions. First, pre-existing variation in CNS anomaly rates is 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 Figure 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 replace $Post_{st}$ with $\sum_{\gamma=1992,\gamma\neq1995}^{2002} \mathbf{1}\{t \in \gamma\}$ in Equation 1. 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.

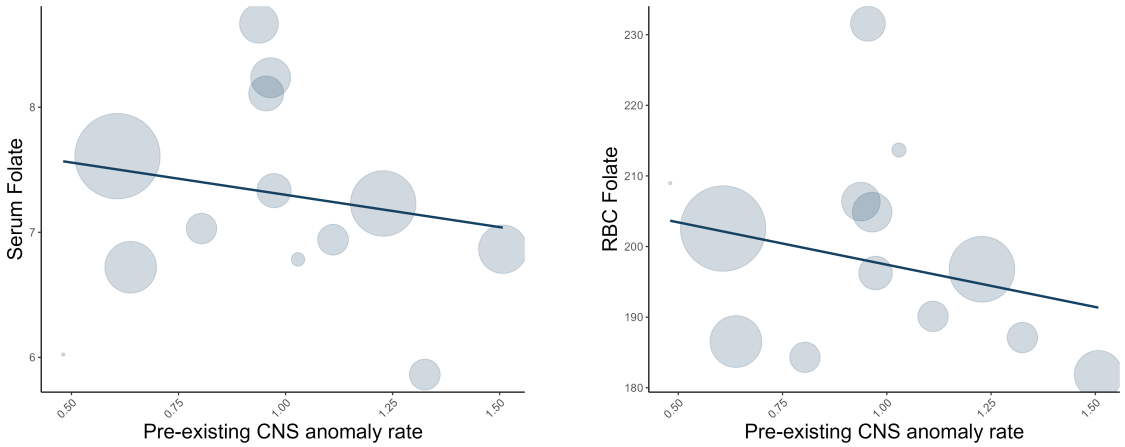
Table 1: Correlation between Pre-existing CNS Anomaly Rate and Baseline Characteristics

	Pre-existing CNS anomaly rates (per 1,000 births)			
	CZ-by-state mean (SD)	State level regression	CZ-by-state Level regression	
	(1)	(2)	(3)	(4)
Demographic features				
Share of black (%), 1988	7.42	-0.0214**	-0.0150***	-0.0098***
	(12.10)	(0.0095)	(0.0027)	(0.0036)
Share of female (%), 1988	50.95	0.1241	0.0327	-0.0538
	(1.52)	(0.1231)	(0.0362)	(0.0471)
Share of under 5 (%), 1988	7.47	0.2364*	0.1435***	0.0853
	(1.28)	(0.1326)	(0.0522)	(0.0918)
Share of over 65 (%), 1988	14.31	-0.0266	-0.0561**	0.0061
	(4.06)	(0.0568)	(0.0250)	(0.0293)
Birth rate (%), 1988	13.97	0.0015	-0.0147	0.0100
	(5.20)	(0.0259)	(0.0111)	(0.0126)
Death rate (%), 1988	9.93	0.1745	0.2168***	0.1281**
	(2.39)	(0.1826)	(0.0501)	(0.0551)
Log population, 1988	11.23	-0.0151	-0.1325***	-0.0784**
	(1.57)	(0.0709)	(0.0265)	(0.0316)
Economic condition				
Transfer income p.p. (1,000$), 1988	2.09	-0.3374	-0.2855**	-0.4117
	(0.38)	(0.3801)	(0.1397)	(0.2909)
Income p.p. (1,000$), 1985	8.69	0.1093	0.0822**	0.0438
	(1.83)	(0.0687)	(0.0336)	(0.0311)
Federal funds p.p. (1,000$), 1986	3.03	-0.1507	-0.0548**	-0.0213
	(1.43)	(0.0903)	(0.0251)	(0.0199)
Unemployment rate (%), 1986	8.53	0.0037	0.0207	0.0174
	(3.61)	(0.0388)	(0.0152)	(0.0157)
Agriculture				
Value of produces sold per farm (million $), 1987	0.07	-3.366*	-0.6388	0.1378
	(0.08)	(1.712)	(0.5037)	(0.3714)
Average farm size (1,000 acres), 1987	0.89	-0.0172	-0.0433***	-0.0010
	(1.96)	(0.0745)	(0.0165)	(0.0198)
State FE				✓
Observations		49	857	857
R^2		0.5505	0.1798	0.3567
Adjusted R^2		0.3836	0.1671	0.3074

Notes: In parentheses are heteroskasticity-robust standard errors. 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.

Second, the pre-existing CNS anomaly rate should reflect the level 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 9b), supporting the validity of this assumption.



(A) Serum Folate (B) RBC Folate

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

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

For CNS anomaly rate$_s$ in Equation 1, I report results using both continuous CNS anomaly rates and binary indicators for 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 may lose some variation but do not require the stricter parallel trend assumption. They also 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.

4.3 High impact group

This research design faces potential challenges with statistical power for several reasons. First, the causal chain between maternal exposure to folic acid fortification and young adult outcomes is long, suggesting a potentially small effect size. Second, the variation in pre-existing CNS anomaly rates is only available at the state-of-birth level, making the exposure measures less accurate for long-term outcomes. Finally, there may be substantial heterogeneity in effects. For example, individuals with better access to a nutrient-balanced diet or folic acid supplements might not benefit from fortification.

To address these concerns, I focus on nonmovers as a "high-impact" group—young adults who live in their state of birth at the time of the survey. Nonmovers are a more disadvantaged sub-sample: they are more likely to be people of color and Hispanic, more likely to come from the Midwest and South, less likely to pursue higher education, and less likely to have higher income or be employed. When employed, nonmovers also tend to earn lower wages (Table 2). Therefore, they may experience more pronounced effects of fortification. Nonmovers constitute approximately 70% of the full sample, ensuring sufficient economic significance. Additionally, Figure 5 shows that maternal exposure to folic acid fortification does not influence the likelihood of being a nonmover.

Table 2: 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***
Student in tertiary schools	0.2570	0.2654	-0.0084***
College student	0.2165	0.2003	0.0162***
Graduate/professional student	0.0405	0.0651	-0.0246***
Income	23.8943	29.2463	-5.3520***
In labor force	0.8033	0.8219	-0.0187***
Wage if in labor force	27.3314	32.7359	-5.3945***

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.

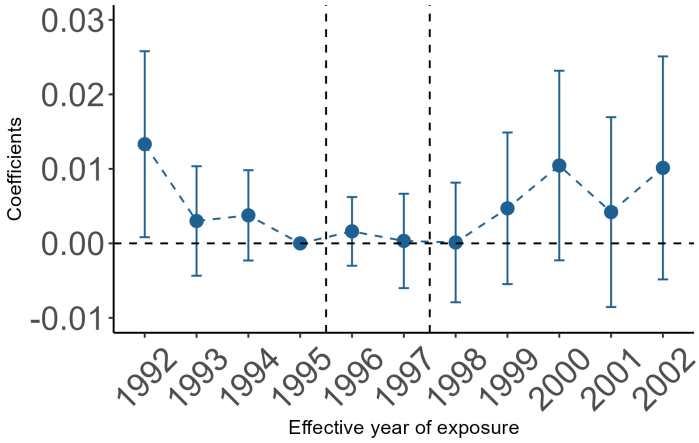


FIGURE 5: MATERNAL EXPOSURE TO FOLIC ACID FORTIFICATION DOES NOT AFFECT PROBABILITY OF BEING A NONMOVER

Notes: Dependent variable is whether individual is a nonmover. The corresponding DD estimate is -0.0010 (0.0032), both small and insignificant. Model specification is the same as Equation 1.

5 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.

5.1 Effect on folate content in foods

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 6, 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).

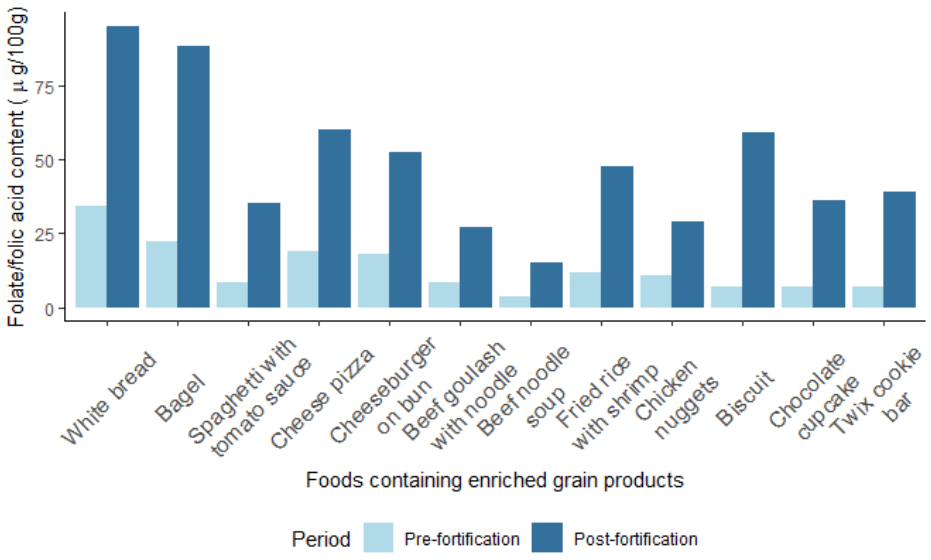


FIGURE 6: CHANGES IN FOLATE CONTENTS IN SELECTED FOODS DUE TO FORTIFICATION

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.

5.2 Effect on dietary folate intake

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 μg in the post-fortification period (Figure 7). Notably, these intake figures exclude folic acid obtained from nutritional supplements and medications (Ahluwalia et al., 2016).

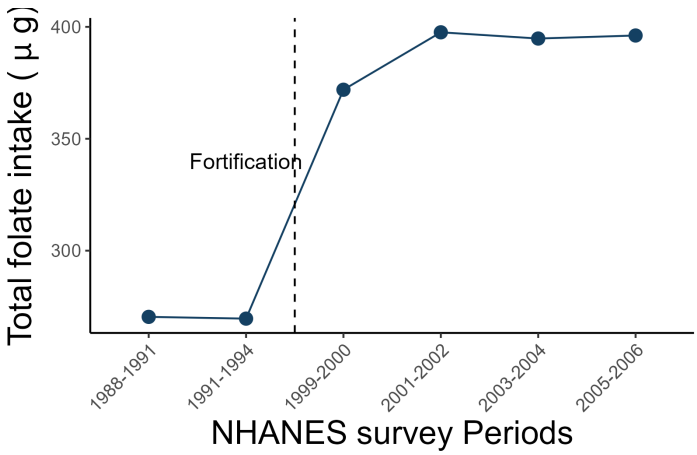


Figure 7: Dietary Folate Concentrations Before and After Fortificatio

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

5.3 Effect on blood folate

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 8 illustrates trends in serum and red blood cell (RBC) folate concentrations—both key biomarkers of folate deficiency. The results show that serum folate levels more than doubled, while RBC folate levels increased by nearly 50%, indicating a sustained improvement in folate absorption.

5.4 Effect on congenital anomalies

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 9a. Moreover, Figure

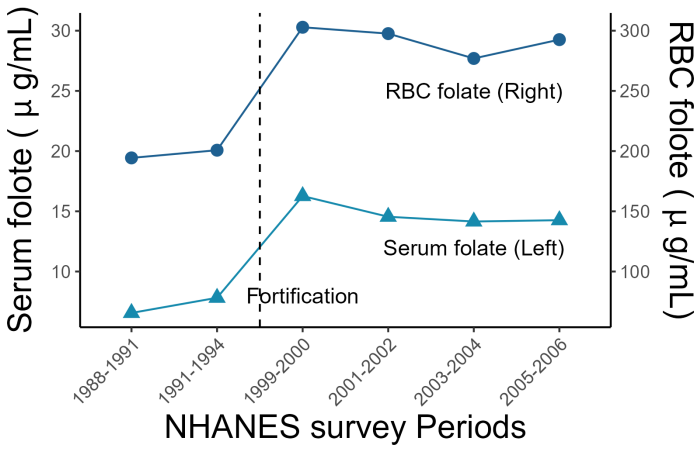
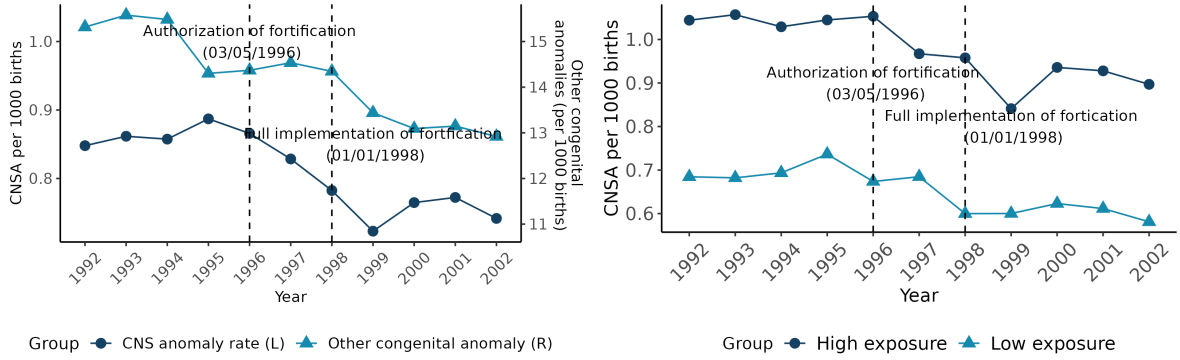


FIGURE 8: BLOOD FOLATE CONCENTRATIONS BEFORE AND AFTER FORTIFICATIONN

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.

9b shows that CNS anomaly rates declined in both high- and low-exposure regions, with a more pronounced decline in the high-exposure regions.



(A) CNS and non-CNS anomaly rates

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

FIGURE 9: TRENDS IN CONGENITAL ANOMALY RATES

Notes: The unit of CNS Anomaly rate is cases per 1,000 births. High exposure is defined if state of birth has higher-than-median pre-existing CNS anomaly rate. The threshold is state level number-of-birth-weighted median.

5.5 Effect 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 3). Figure 10 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 A1 and Figure A1).

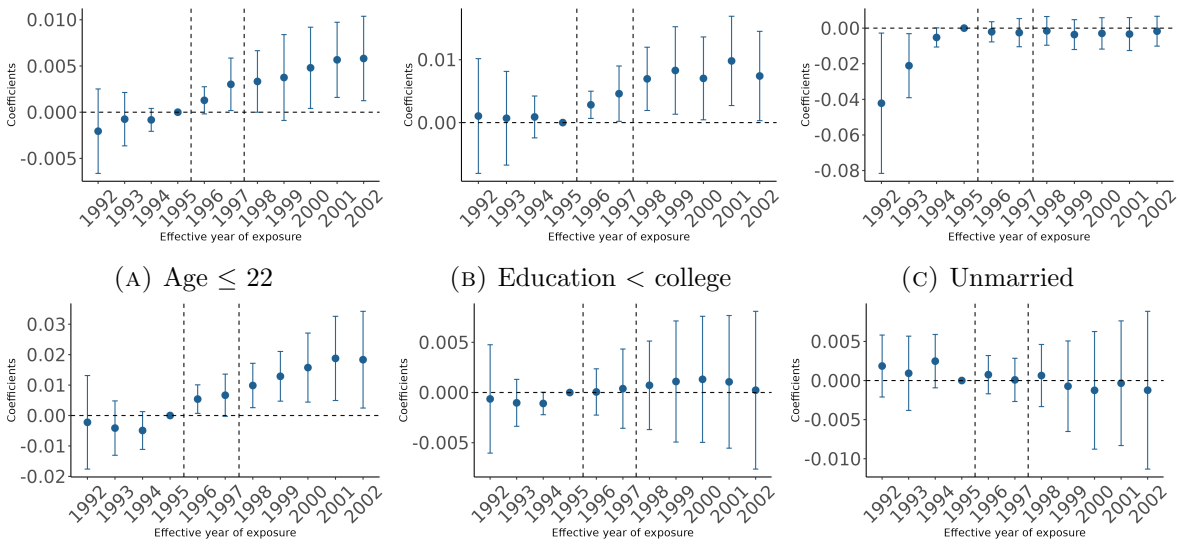
Table 3: Effects of Folic Acid Fortification on Maternal Characteristics, Continuous Exposure

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



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

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

FIGURE 10: 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.

5.6 Effect on test scores (in progress)

Sixth, I plan to investigate the effects of maternal exposure to folic acid fortification on children's or young adults' test scores. This analysis will require access to restricted-use data, and I am currently in the process of applying for the necessary data access.

5.7 Effect on school enrollment of young adults

Finally, I find that maternal exposure to folic acid fortification increase school enrollment at young adulthood, especially for relatively disadvantaged nonmovers. Table 4 presents effects of maternal exposure to folic acid fortification on total tertiary school enrollment of young adults for the full sample and non-movers. Overall, the findings indicate that maternal exposure to folic acid fortification increases the likelihood of young adults enrolling in college or graduate/professional schools. The effects are more pronounced among non-movers, consistent with the hypothesis that fortification has a greater impact on more disadvantaged populations.

I find that maternal exposure to folic acid fortification significantly increases the probability of young adults enrolling in college or graduate/professional schools, particularly in

regions with higher pre-existing CNS anomaly rates. In regions classified as high-exposure, defined by the top 30% or 40% of pre-existing CNS anomaly rates, maternal exposure to fortification leads to an increase in the probability of college or graduate/professional school enrollment by 0.63 or 0.78 percentage points, respectively, compared to regions with lower exposure. The estimates are even more pronounced for nonmovers. For this group, in regions classified as high-exposure—whether defined by the 75th percentile of pre-existing CNS anomaly rates for continuous exposure or by thresholds such as above the mean, top 50%, top 40%, or top 30%—maternal exposure to fortification leads to an increase in college or graduate/professional school enrollment by 0.99 to 1.46 percentage points, compared to regions with lower exposure. Importantly, Figures 11 and 12 show that the trends in coefficients during the pre-fortification periods remain fairly flat, irrespective of the exposure measures used. This consistency suggests that the significant results reported in Table 4 are not influenced by pre-fortification differential trends in outcomes.

Table 4: Effects of Maternal Exposure to Folic Acid Fortification on Tertiary School Enrollment of Young Adults, by Exposure Measure

	Continuous exposure	Binary exposure			
		Above mean	Above median	Top 40%	Top 30%
	(1)	(2)	(3)	(4)	(5)
			Panel A: full sample		
CNS anomaly measure × Post	0.0062	0.0063	0.0073	0.0063*	0.0078*
	(0.0041)	(0.0037)	(0.0044)	(0.0037)	(0.0039)
Observations	1,440,521	1,440,521	1,440,521	1,440,521	1,440,521
R^2	0.1397	0.1397	0.1397	0.1397	0.1397
Dependent variable mean	0.3627	0.3627	0.3627	0.3627	0.3627
			Panel B: Nonmovers		
CNS anomaly measure × Post	0.0099**	0.0102***	0.0146***	0.0105***	0.0126***
	(0.0037)	(0.0036)	(0.0044)	(0.0035)	(0.0037)
Observations	1,027,413	1,027,413	1,027,413	1,027,413	1,027,413
R^2	0.1352	0.1317	0.1334	0.1341	0.1342
Dependent variable mean	0.3580	0.3580	0.3580	0.3580	0.3580

Notes: Nonmovers are those who live in the state they were born at the time of survey. In parentheses are standard errors 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 (0.57). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, state-of-residence-by-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.

The results remain consistent when school enrollment is broken down by age. Tables

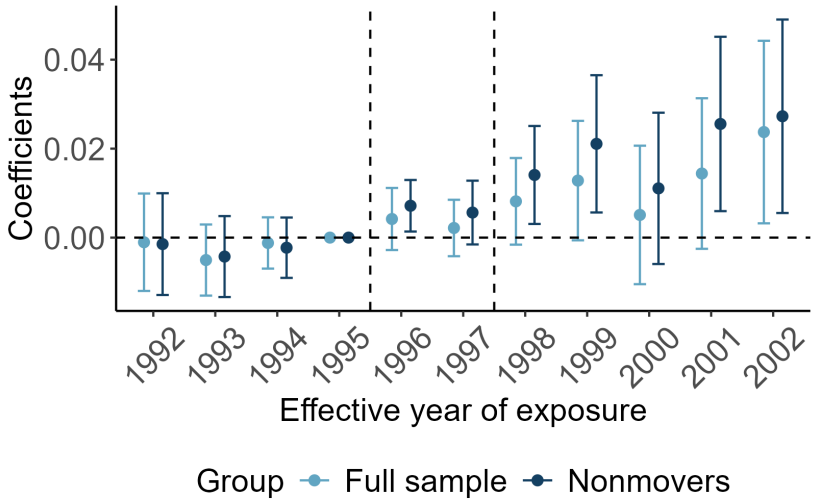


Figure 11: Dynamic Effects of Maternal Exposure to Folic Acid Fortification on Tertiary School Enrollment of Young Adults, Continuous Exposure

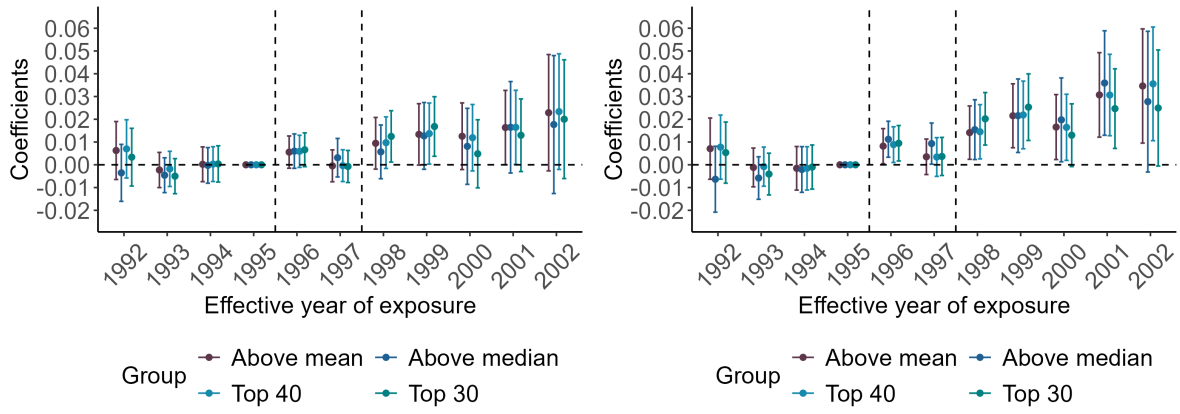
Notes: Nonmovers are those who live in the state they were born at the time of survey. Standard errors are clustered on state of birth. All regressions and dependent variable means are weighted by ACS sample weight. Both coefficients and standard errors are rescaled by the difference between 25th percentile and 75th percentile CNS anomaly rates (0.57). 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.

A2 and A3 show the effects of maternal exposure to folic acid fortification on college enrollment among young adults aged 19-22 and on graduate/professional school enrollment among those over 22 years old. In regions classified as high-exposure, maternal exposure to fortification increases the probability of college enrollment by 0.78-0.89 percentage points for young adults aged 19-22 and the probability of graduate/professional school enrollment by 0.48-0.73 percentage points, compared to regions with lower exposure. For nonmovers, the effects are even more pronounced, with college enrollment increasing by 1.19-1.8 percentage points and graduate/professional school enrollment by 0.45-0.98 percentage points. Notably, there are no significant pre-fortification trends in differential outcomes for either set of results, regardless of the exposure measures used (see Figures A2, A3, A4, and A5).

6 Discussion (in progress)

6.1 Effect size

In this session I compare effective size of long run effects of folic acid fortification to other nutrition enhancement programs, including salt iodization, iron fortification of bread, and



(A) Full sample (B) Nonmovers

FIGURE 12: DYNAMIC EFFECTS OF MATERNAL EXPOSURE TO FOLIC ACID FORTIFICATION ON TERTIARY SCHOOL ENROLLMENT OF YOUNG ADULTS, BINARY EXPOSURE

Notes: Nonmovers are those who live in the state they were born at the time of survey. 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 (0.53). 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.

food assistance programs.

6.2 Cost-effectiveness

Cost-benefit analysis for U.S. Expected cost-benefit ratio for populous developing countries such as China, India, and Brazil.

7 Conclusion (in progress)

In this paper, I compare birth outcomes and young adults' educational and labor outcomes of cohorts exposed and unexposed to folic acid fortification in their first trimester across regions with high and low pre-existing CNS anomaly rates to identify effects of folic acid fortification in the United States. I document short and long run effects on human capital. In the short run, folic acid fortification increases shares of births given by disadvantaged mothers, likely through improving fetus survival rate. In the long run, maternal exposure to folic acid fortification increases probability of enrolling in tertiary schools for young adults who live in their state of birth. These results are robust to different exposure measures, sub-samples, and model specifications.

I compare effect sizes of fortification and food assistance programs (not done yet) and

find fortification is more cost-effective. This indicates fortification, and reformulation intervention in general, can play a bigger role in existing food and nutrition assistance policy portfolio.

This paper has several limitations. First, existing data do not allow me to distinguish marginal survivors who would otherwise not have been born if there were no fortification from others, or always survivor. Since folic acid fortification changes decomposition of births who survive to young adulthood, it is unclear whether imprecise results on birth and long run outcomes are driven by heterogeneity of effect or worse outcomes of marginal survivors. One possible mechanism of imprecise estimates on long run effects is that, if fortification both increased outcomes of always survivors and adds marginal survivors to exposed cohorts, mean estimates would be noisier since improved outcomes of always survivors in exposed cohorts would be diluted by marginal survivors with outcomes worse than always survivors in unexposed cohorts. Second, I assume folic acid fortification works during the first trimester of gestation, supported by scientific literature. If fortification also affected children in other life stages, which is less clear according to medical research, its effects would be more difficult to pin down as we do not know migration dynamics of individuals hence their total exposure time.

Consistent with the board fetal origin literature, positive long run effects of folic acid fortification indicate that early-life intervention can yield broad, long-lasting benefits for children. Additionally, with evidence from the U.S., it is reasonable to believe that Folic acid fortification could work even better in developing world where accessibility to folate-rich food or folic acid supplements is much lower. Low unit cost of fortification and without requiring change in consumers' behaviors make folic acid fortification a particularly appealing public health intervention for developing countries.

For future research it would be interesting to extend the scope of this paper to different life stages when better data is available. At this point oldest exposed cohorts are still in their 20s. It would be interesting to revisit effects of folic acid fortification in a few years when exposed cohorts become old enough to study how fortification affects a wider range of human capital outcomes such as years of education, income of full-time workers, and family formation. Another promising direction future research can go is to examine effects of folic acid fortification in developing country setting. Effects of folic acid fortification is expected to be more prominent in developing countries as folate deficiency is much more severe there.

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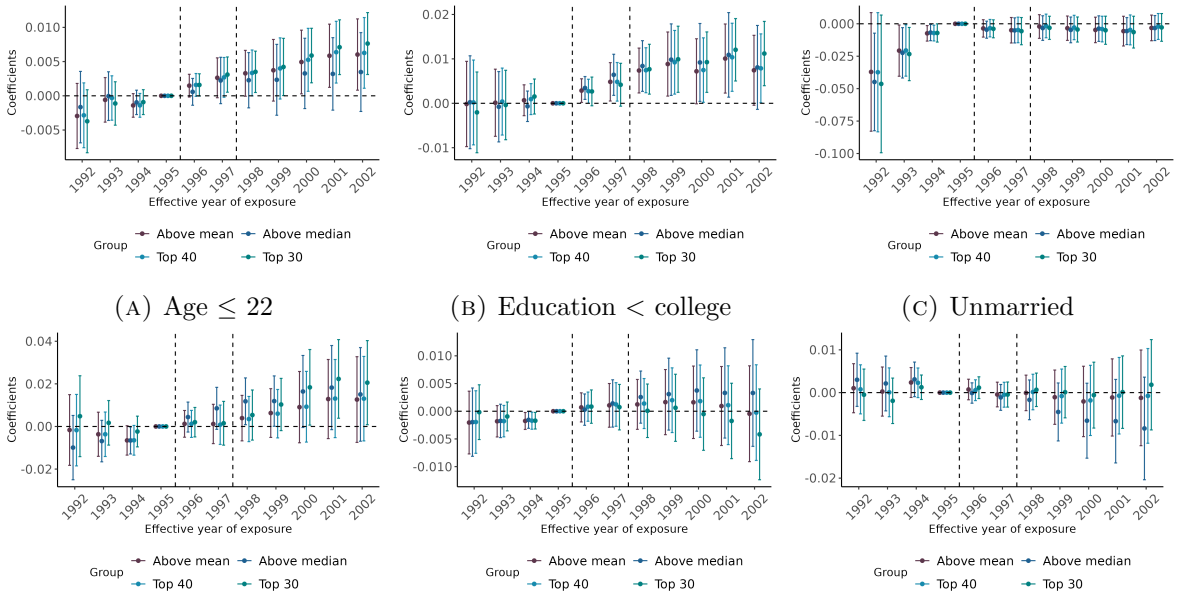
Appendices

A Tables and figures

Table A1: Effects of Folic Acid Fortification on Maternal Characteristics, Binary Exposure

	Share of mothers with following characteristics					
	Age $\leq$ 22	Education $<$ college	Unmarried	Inadequate prenatal care	Non-white	Hispanic
	(1)	(2)	(3)	(4)	(5)	(6)
Above mean × Post	0.0044***	0.0065**	0.0074**	0.0088	0.0024	-0.0015
	(0.0014)	(0.0031)	(0.0032)	(0.0066)	(0.0026)	(0.0035)
Observations	111,683	111,678	111,683	111,683	111,683	111,678
R^2	0.9095	0.9394	0.9090	0.7826	0.9847	0.9906
Dependent Variable Mean	0.2697	0.5462	0.3198	0.2341	0.1976	0.1801
Above median × Post	0.0027	0.0081**	0.0078***	0.0161**	0.0034	-0.0049
	(0.0018)	(0.0034)	(0.0029)	(0.0066)	(0.0027)	(0.0038)
Observations	111,683	111,678	111,683	111,683	111,683	111,678
R^2	0.9094	0.9394	0.9090	0.7835	0.9847	0.9906
Dependent Variable Mean	0.2697	0.5462	0.3198	0.2341	0.1976	0.1801
Top 40 × Post	0.0046***	0.0064**	0.0078**	0.0087	0.0025	-0.0012
	(0.0014)	(0.0031)	(0.0032)	(0.0066)	(0.0026)	(0.0035)
Observations	111,683	111,678	111,683	111,683	111,683	111,678
R^2	0.9095	0.9394	0.9090	0.7826	0.9847	0.9906
Dependent Variable Mean	0.2697	0.5462	0.3198	0.2341	0.1976	0.1801
Top 30 × Post	0.0052***	0.0074**	0.0085**	0.0098	0.0007	0.0005
	(0.0014)	(0.0033)	(0.0032)	(0.0077)	(0.0026)	(0.0031)
Observations	111,683	111,678	111,683	111,683	111,683	111,678
R^2	0.9095	0.9394	0.9091	0.7826	0.9847	0.9906
Dependent Variable Mean	0.2697	0.5462	0.3198	0.2341	0.1976	0.1801

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



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

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

FIGURE A1: 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.

Table A2: Effects of Maternal Exposure to Folic Acid Fortification on College Enrollment of the 19-22-Year-Old

	Continuous exposure	Binary exposure			
		Above mean	Above median	Top 40%	Top 30%
	(1)	(2)	(3)	(4)	(5)
			Panel A: full sample		
CNS anomaly measure × Post	0.0056	0.0085*	0.0061	0.0089*	0.0078*
	(0.0042)	(0.0049)	((0.0058)	(0.0049)	(0.0046)
Observations	807,669	807,669	807,669	807,669	807,669
R^2	0.0675	0.0675	0.0675	0.0675	0.0675
Dependent variable mean	0.4927	0.4927	0.4927	0.4927	0.4927
			Panel B: Nonmovers		
CNS anomaly measure × Post	0.0119**	0.0138**	0.0127**	0.0146**	0.0180***
	(0.0047)	(0.0058)	(0.0061)	(0.0057)	(0.0055)
Observations	581,820	581,820	581,820	581,820	581,820
R^2	0.0614	0.0614	0.0614	0.0613	0.0614
Dependent variable mean	0.4821	0.4821	0.4821	0.4821	0.4821

Notes: Nonmovers are those who live in the state they were born at the time of survey. In parentheses are standard errors 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 (0.57). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, state-of-residence-by-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.

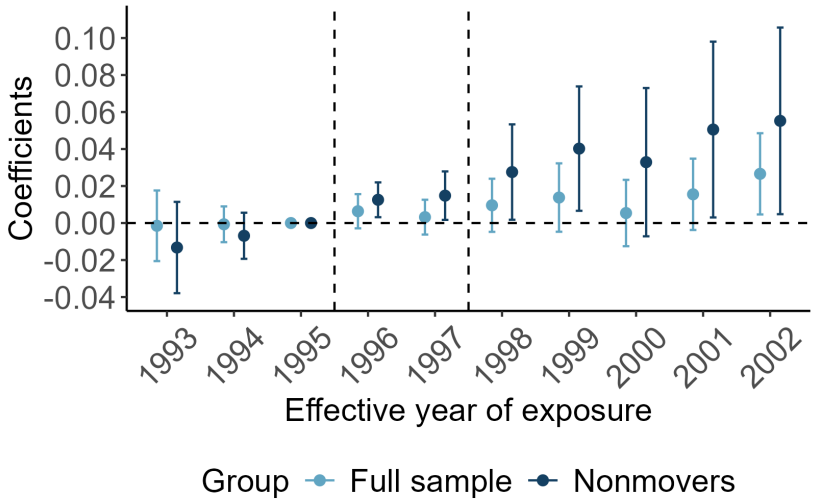
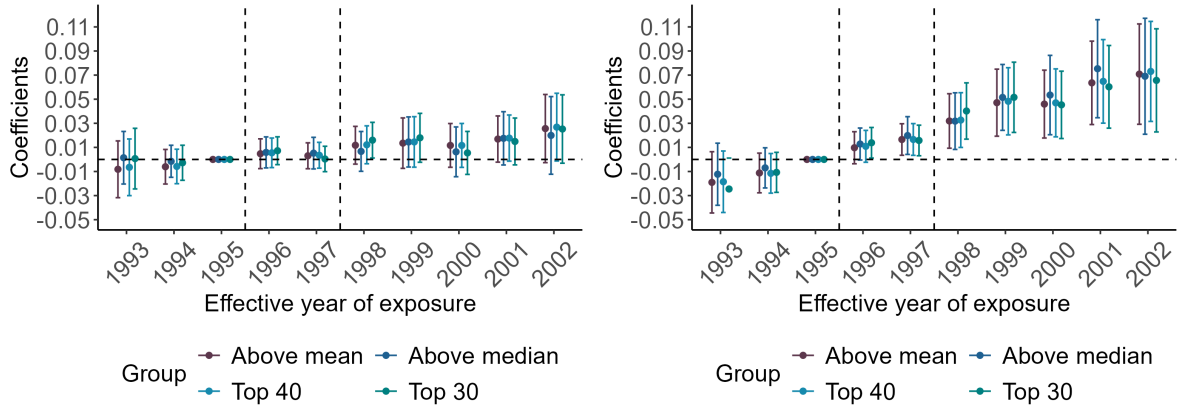


FIGURE A2: DYNAMIC EFFECTS OF MATERNAL EXPOSURE TO FOLIC ACID FORTIFICATION ON COLLEGE ENROLLMENT OF THE 19-22-YEAR-OLD, CONTINUOUS EXPOSURE

Notes: Nonmovers are those who live in the state they were born at the time of survey. 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 (0.57). 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.



(A) Full sample

(B) Nonmovers

FIGURE A3: DYNAMIC EFFECTS OF MATERNAL EXPOSURE TO FOLIC ACID FORTIFICATION ON ON COLLEGE ENROLLMENT OF THE 19-22-YEAR-OLD, BINARY EXPOSURE

Notes: Nonmovers are those who live in the state they were born at the time of survey. 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 (0.57). 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 A3: Effects of Maternal Exposure to Folic Acid Fortification on Graduate/Professional School Enrollment of the Over-22-Year-Old

	Continuous exposure	Binary exposure			
		Above mean	Above median	Top 40%	Top 30%
	(1)	(2)	(3)	(4)	(5)
			Panel A: full sample		
CNS anomaly measure × Post	0.0048***	0.0067***	0.0053**	0.0070***	0.0073***
	(0.0013)	(0.0017))	(0.0023)	(0.0017)	(0.0015)
Observations	632,852	632,852	632,852	632,852	632,852
R^2	0.0134	0.0134	0.0134	0.0134	0.0134
Dependent variable mean	0.0608	0.0608	0.0608	0.0608	0.0608
			Panel B: Nonmovers		
CNS anomaly measure × Post	0.0045**	0.0083***	0.0051*	0.0086***	0.0098***
	(0.0019)	(0.0025)	(0.0029)	(0.0025)	(0.0023)
Observations	445,593	445,593	445,593	445,593	445,593
R^2	0.0126	0.0127	0.0123	0.0127	0.0127
Dependent variable mean	0.0525	0.0525	0.0525	0.0525	0.0525

Notes: Nonmovers are those who live in the state they were born at the time of survey. In parentheses are standard errors 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 (0.57). Percentiles are weighted by number of births. I control for state-of-birth fixed effects, quarter-and-year-of-birth fixed effects, state-of-residence-by-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.

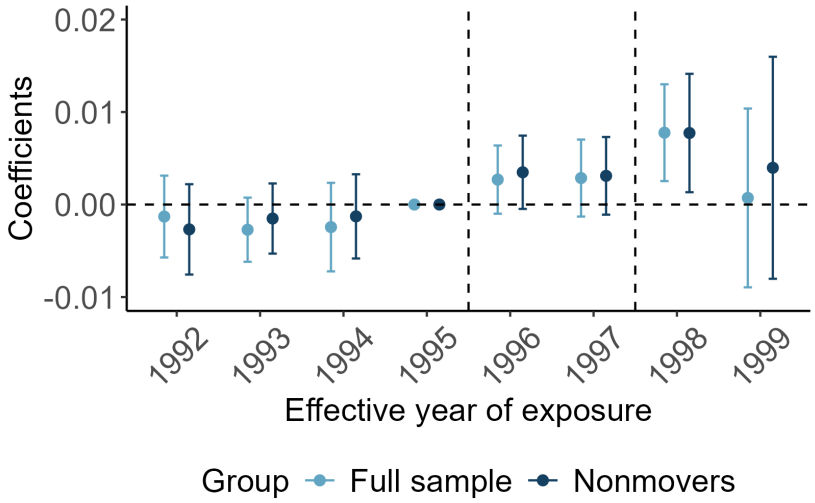
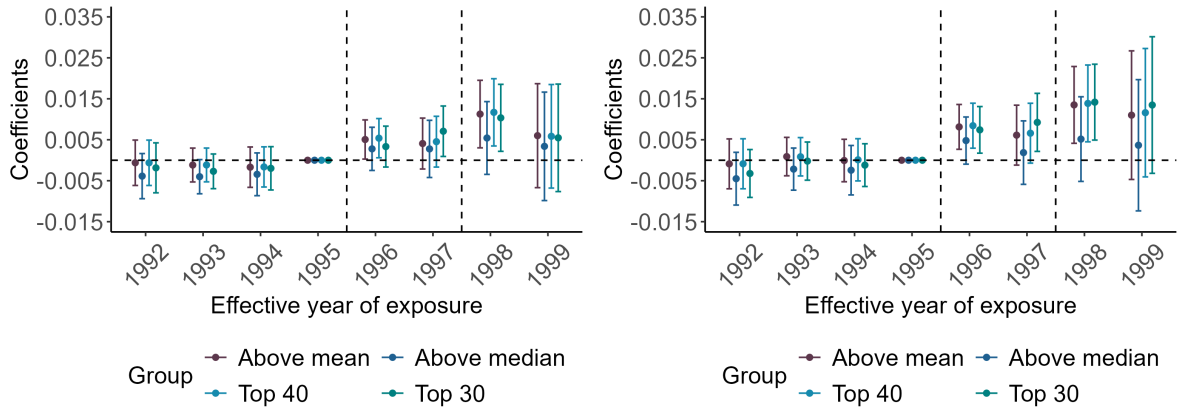


Figure A4: Dynamic Effects of Maternal Exposure to Folic Acid Fortification on Graduate/Professional School Enrollment of the Over-22-Year-Old, Continuous Exposure

Notes: Nonmovers are those who live in the state they were born at the time of survey. 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 (0.57). 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.



(A) Grad./prof. school enrollment conditional on over 22 years old. full sample

(B) Grad./prof. school enrollment conditional on over 22 years old. nonmovers

FIGURE A5: DYNAMIC EFFECTS OF MATERNAL EXPOSURE TO FOLIC ACID FORTIFICATION ON GRADUATE/PROFESSIONAL SCHOOL ENROLLMENT OF THE OVER-22-YEAR-OLD, BINARY EXPOSURE

Notes: Nonmovers are those who live in the state they were born at the time of survey. 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 (0.57). 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 state-level pre-intervention characteristics interacted with linear time trend.